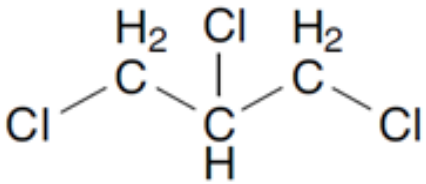
Report to the Hawaii Department of Health, Safe Drinking Water Branch, Regarding the Human Health Risks of 1,2,3-Trichloropropane in Tap Water



Prepared by

Eric P. Hooker, MS, DABT
Keri G. Fulcher, MS
Herman J. Gibb, PhD, MPH
Tetra Tech Sciences
Arlington, VA

Original April 24, 2012
Revised June 18, 2012

TETRA TECH

Table of Contents

Executive Summary

1,2,3-Trichloropropane (TCP) is a persistent environmental contaminant found in groundwater and treated tap water in Hawaii. The Hawaii Department of Health (HDOH) requested Tetra Tech to review the available data on the potential human health effects of TCP as a drinking water contaminant and comment on the adequacy of Hawaii's MCL for TCP. This review includes discussions of previous assessments of TCP, evaluation of available data, and the development of an alternative dose-response assessment of the human cancer risk associated with TCP in tap water.

There are no studies available on the health effects of TCP in humans. The primary studies that describe the health effects of long-term exposure to TCP are chronic bioassays conducted in rats and mice by the National Toxicology Program (NTP). In those studies, the incidence of tumors in multiple tissues was increased in a dose-dependent manner in all animals tested. However, the method of administration (gavage) and dose levels used in these studies poorly represent the exposure that humans would be expected to experience drinking TCP-contaminated water.

In previous reviews of TCP, dose-response assessments of carcinogenic potency have been based on either tumor incidence in a single tissue type (pancreas) or a combination of tumor types in the most sensitive animal tested (female mice). An alternative approach includes the overall combined carcinogenic risk of all tumor types in both sexes of both species tested. The result of these different approaches has produced cancer potency values that range from 0.0588 to 30 $(mg/kg\text{-}day)^{-1}$. The most conservative of these values [30 $(mg/kg\text{-}day)^{-1}$] was derived from multiple tumors in female mice including the forestomach, a tumor site which may not be relevant to humans.

There are no measured data of total human exposure to TCP in tap water (includes ingestion and bathing). Therefore, mathematical exposure models were employed to estimate the amount of TCP that humans are likely to absorb through three routes: ingestion, inhalation, and transdermal absorption. The models are conservative and likely overestimate actual human exposure.

The cancer risk levels associated with a range of TCP concentrations in tap water were estimated using cancer slope value, exposure estimates based on models, and application of age-dependent adjustment factors (ADAF). Regulation of contaminants in water is generally established with cancer risk values within the range of 10^{-6} to 10^{-4}. In a population the size of Hawaii's, it would be impossible to detect an excess cancer risk of 10^{-4}.

To date, the State of Hawaii is the only jurisdiction to set an enforceable maximum contaminant level (MCL) for TCP. Based on existing dose-response assessments, the

current MCL of 0.6 µg/L is associated with an estimated human cancer risk that ranges from 2.8×10^{-6} to 1.4×10^{-3}. Between 2000 and 2011, the concentration of TCP in treated tap water in Hawaii has only exceeded the MCL a few times, and the average concentration of TCP in tap water is close to the detection limit of 0.04 µg/L.

In addition to the analysis of potential human health effects, the establishment of a MCL for a suspected carcinogen is based on factors such as technological limitations and economic cost-benefit considerations. An analysis of technological and economic limitations of removing TCP from public water works is beyond the scope of the current project. Estimates of the cancer risk for a range of TCP concentrations in tap water have been calculated to provide HDOH with information on which to base regulatory decisions regarding TCP.

1. Introduction

Tetra Tech, Inc., was requested by the State of Hawaii Department of Health (HDOH) to examine the potential human health concerns associated with 1,2,3-Trichloropropane (TCP) and comment on the adequacy of the MCL. This document presents that analysis and includes:

- a brief history of uses of TCP in Hawaii and regulations for TCP in drinking water in Hawaii
- descriptions of existing studies and reviews on the health hazards of TCP
- a comparison of risk assessments of TCP prepared for HDOH with the current toxicological review by the United States Environmental Protection Agency (U.S. EPA)
- an evaluation of the adequacy of the current HDOH MCL for TCP
- TCP drinking water regulations/guidance set by various states, countries, and international organizations.
- a review and analysis of TCP concentrations in source well water and treated tap water in Hawaii

2. History of TCP Sources and Water Regulation in Hawaii

Sources of TCP in Hawaii

TCP is an impurity associated with a fumigant known as D-D, a mixture of 1,2-dichloropropane, 1,3-dichloropropene, and 2,3-dichloropropene (HDOH 2009a). D-D was formerly used as a soil fumigant to control nematodes on pineapple farms in Hawaii (HDOH 2009a). Fumigant formulations containing 1,2,3-trichloropropane were banned in the late 1970s to early 1980s after about 20 to 30 years of use. However, consistent with other chlorinated hydrocarbons, TCP is persistent and can still be found in groundwater decades after its application to crops was discontinued. See Section 8 for a summary of the most recent data on TCP concentrations at water treatment facilities in Hawaii.

TCP Water Regulations in Hawaii

The United States Environmental Protection Agency (U.S. EPA) has set Maximum Contaminant Level Goals (MCLG) and Maximum Contaminant Level (MCL) on a number of drinking water contaminants. An MCLG is the level of a contaminant in drinking water below which there is no known or expected risk to health. MCLGs allow for a margin of safety and are non-enforceable public health goals. A MCL is the highest level of a contaminant that is allowed in drinking water. MCLs are set as close to MCLGs

as feasible using the best available treatment technology and taking cost into consideration. MCLs are enforceable standards.

The U.S. EPA has not established a MCL or MCLG for TCP. States may set water regulations that are more protective than the standards and regulations set by the U.S. EPA. The State of Hawaii is the only state to have established an enforceable MCL for TCP. In the mid-1980's, the State of Hawaii established a MCL of 0.8 µg/L based in part on non-carcinogenic effects and limited toxicity information that was available at the time (Environ 1985). Subsequent studies by the National Toxicology Program (NTP 1993) suggested that TCP had carcinogenic potential.

In the early 1990's, HDOH commissioned Toxicologic Consultative Services to review the health effects of TCP in consideration of the recent NTP studies (Tardiff 1992). This review proposed a long term action level of 3.0 µg/L and a long term goal of 0.3 µg/L for TCP. No change to the state MCL was made at that time.

In 2001, HDOH commissioned Dr. Robert Tardiff, then with The Sapphire Group, Inc., to conduct an updated review of the available data on TCP. Based on his review of the data, including data on mode of action and toxicokinetics, Dr. Tardiff (2001) proposed a long term action level of 6.0 µg/L and a long term goal of 0.6 µg/L for TCP. As of 2005, the current MCL for TCP is 0.6 µg/L (HDOH 2005).

One of the goals of the current review is to evaluate the available data in consideration of current guidelines and methodologies and to make recommendations to the State of Hawaii regarding the adequacy of the State's MCL.

3. Review of Studies on the Carcinogenicity of TCP

There are no studies of the carcinogenic potential of TCP in humans available. All that is known of the carcinogenicity of TCP comes from laboratory studies in animals and *in vitro* testing.

All of the current cancer hazard and dose response assessments of TCP are based on the results of chronic two-year oral gavage studies of TCP in rats and mice that were conducted by the NTP (1993; Irwin et al 1995). In the chronic rat study, groups of 60 male and 60 female F344/N rats received 0, 3, 10, or 30 mg/kg-day TCP in corn oil by oral gavage 5 days/week for up to 104 weeks. After 15 months, 10 rats per group were sacrificed to allow an interim evaluation of gross and histopathological toxic effects. Due to high mortality in rats receiving 30 mg/kg at the interim evaluation, the survivors in the high-dose group were sacrificed at week 65 (females) and week 76 (males).

TCP administered to male rats caused dose-dependent increases in the incidence of squamous cell papillomas and carcinomas of the oral mucosa and forestomach, adenomas of the pancreas and kidney, adenomas or carcinomas of the preputial gland, and carcinomas of the Zymbal's gland. TCP administered to female rats caused dose-dependent increases in incidences of squamous cell papillomas and carcinomas of the oral mucosa and forestomach, adenomas or carcinomas of the clitoral gland, adenocarcinomas of the mammary gland, and carcinomas of the Zymbal's gland. The incidence of these lesions is presented in Table 1 (page 6).

In the NTP (1993) chronic mouse study, groups of 60 mice/sex received 0, 6, 20, or 60 mg/kg-day TCP in corn oil by gavage 5 days/week for up to 104 weeks. High rates of mortality led to the early termination of mice in the 60 mg/kg-day group at 73 weeks (females) and 79 weeks (males) and in the 20 mg/kg-day group at 88 weeks (both sexes). Dose-dependent increases in incidence of squamous cell papillomas and carcinomas of the forestomach, hepatocellular adenomas or carcinomas of the liver, and Harderian gland adenomas occurred in male mice. TCP administered to female mice caused dose-dependent increases in incidence of squamous cell carcinomas of the oral mucosa, squamous cell papillomas and carcinomas of the forestomach, hepatocellular adenomas or carcinomas of the liver, Harderian gland adenomas, and uterine adenomas, adenocarcinomas, and stromal polyps. The incidence of these lesions is presented in Table 2 (page 7).

Genetic Toxicity

The NTP (1993) concluded that there is *clear evidence of carcinogenic activity* for TCP in rats and mice of both sexes. Supporting these conclusions is the fact that TCP was positive for genotoxic effects in the Ames bacterial mutagenicity assay and the L5178Y mouse lymphoma mutagenicity assay. TCP also induced chromosomal aberrations and sister chromatid exchanges in Chinese hamster ovary cells. These positive *in vitro* genotoxic effects occurred only in the presence of metabolic activation.

Table 1. Incidence of Non-Neoplastic Lesions and Neoplasms in Rats (Irwin et al 1995)[a]

Sex	Tissue and Lesion Type	Control	3 mg/kg-day	10 mg/kg-day	30 mg/kg-day
Males	Oral mucosa[b]	60	60	59	60
	Squamous cell papilloma	0	4	10[c]	22[c]
	Squamous cell carcinoma	0	0	11[c]	25[c]
	Forestomach	60	60	59	60
	Hyperplasia, basal cell	0	7[c]	12[c]	9[c]
	Hyperplasia, squamous	3	28[c]	13[c]	6
	Squamous cell papilloma	0	31[c]	36[c]	46[c]
	Squamous cell carcinoma	0	9[c]	28[c]	14[c]
	Pancreas	60	60	59	60
	Hyperplasia	28	48[c]	53[c]	56[c]
	Adenoma	5	21[c]	37[c]	31[c]
	Adenocarcinoma	0	0	2	1
	Kidney	60	60	59	60
	Hyperplasia	0	1	23[c]	35[c]
	Adenoma	0	2	20[c]	26[c]
	Preputial gland	59	57	59	58
	Adenoma	5	3	6	11[c]
	Carcinoma	0	3	3	6
	Adenoma or carcinoma	5	6	9	17[c]
Females	Oral mucosa	60	59	60	60
	Squamous cell papilloma	1	5	10[c]	21[c]
	Squamous cell carcinoma	0	1	21[c]	23[c]
	Forestomach	60	59	59	60
	Hyperplasia, basal cell	0	10[c]	5[c]	9[c]
	Hyperplasia, squamous	1	26[c]	15[c]	16[c]
	Squamous cell papilloma	0	14[c]	37[c]	24[c]
	Squamous cell carcinoma	0	3	9[c]	6[c]
	Pancreas	60	59	60	60
	Hyperplasia	5	15[c]	24[c]	11[c]
	Adenoma	0	0	2	0
	Kidney	60	57	60	59
	Hyperplasia	0	2	3	12[c]
	Adenocarcinoma	0	0	0	1
	Clitoral gland	56	56	58	59
	Adenoma	5	11	14[c]	12[c]
	Carcinoma	0	0	4	6
	Adenoma or carcinoma	5	11	18[c]	17[c]
	Mammary Gland	60	59	60	60
	Fibroadenoma or adenoma	16	23	22[c]	2
	Adenocarcinoma	1	6	12[c]	22[c]

a. Transcribed from Tables 1 and 2 of Irwin et al. (1995).
b. Number of rats for which each tissue type was examined.
c. Significantly different from controls ($p < 0.05$ or < 0.01).

Table 2. Incidence of Non-Neoplastic Lesions and Neoplasms in Mice (Irwin et al 1995)[a]

Sex	Tissue and Lesion Type	Control	3 mg/kg-day	10 mg/kg-day	30 mg/kg-day
Males	Oral mucosa[b]	60	59	60	60
	Squamous cell papilloma	0	0	0	2
	Forestomach	60	59	60	60
	Hyperplasia, squamous	8	37[c]	32[c]	38[c]
	Squamous cell papilloma	3	35[c]	25[c]	35[c]
	Squamous cell carcinoma	0	41[c]	54[c]	55[c]
	Liver	60	59	60	60
	Hepatocellular adenoma	12	18	21[c]	31[c]
	Hepatocellular carcinoma	4	11[c]	6	3
	Harderian gland	60	59	60	60
	Adenoma	1	2	10[c]	11[c]
Females	Oral mucosa	60	60	60	60
	Squamous cell papilloma	1	0	1	0
	Squamous cell carcinoma	0	0	1	5[c]
	Forestomach	60	60	60	60
	Hyperplasia, squamous	11	25[c]	23[c]	36[c]
	Squamous cell papilloma	0	28[c]	27[c]	33[c]
	Squamous cell carcinoma	0	47[c]	55[c]	51[c]
	Liver	60	60	60	60
	Hepatocellular adenoma	7	9	9	36[c]
	Hepatocellular carcinoma	1	3	0	2
	Harderian gland	60	60	60	60
	Adenoma	3	6	7	10[c]
	Uterus	60	60	60	59
	Stromal polyp	0	2	2	7[c]
	Adenoma	0	1	0	4
	Carcinoma	0	4[c]	3[c]	8[c]
	Adenoma or carcinoma	0	5[c]	3[c]	12[c]

a. Transcribed from Tables 3 and 4 of Irwin et al. (1995).
b. Number of rats for which each tissue type was examined.
c. Significantly different from controls ($p < 0.05$ or < 0.01).

Human Cancer Risk

The following organizations have reviewed the carcinogenicity of TCP and made conclusions or recommendations regarding the cancer risk of TCP to humans.

IARC

The International Agency for Research on Cancer (IARC) last reviewed the carcinogenicity of TCP in 1995. The IARC Working Group concluded that there is inadequate evidence of carcinogenicity in humans but sufficient evidence in experimental animals and that the overall evaluation is "probably carcinogenic to humans" (Group 2A) (IARC 1995). In making the overall evaluation, the IARC Working Group noted that TCP caused tumors at multiple sites and produced a high incidence of tumors in two species of rodents (the NTP 1993 studies in mice and rats). They also noted that the

metabolism of TCP is qualitatively similar in human and rodent microsomes and that TCP is mutagenic to bacteria and mammalian cells *in vitro* and binds to DNA of animals treated *in vivo*. The IARC working group also noted the lack of any studies of the long-term health effects of TCP in humans.

NTP

In its 12th Report on Carcinogens (NTP 2011), TCP is classified as reasonably anticipated to be a human carcinogen, based on sufficient evidence of carcinogenicity from studies in experimental animals.

ECHA

The European Chemicals Agency (ECHA) determined that TCP meets the criteria for classification as a carcinogen and reproductive toxin in accordance with Article 57 (a) and Article 57 (c) of REACH (ECHA 2011). This classification appears to be primarily based on the IARC (1995) assessment of TCP.

European Union

TCP was assigned the risk classification code and labeling requirement, "R45: May Cause Cancer" (EINECS 2012)

TCP is not currently listed in the European Union's Pesticide Database (EU 2011).

Update of Literature Search

The IRIS (2009) review of TCP was the latest comprehensive assessment of this chemical by an authoritative body. An extensive search of the scientific literature was conducted to identify any new information about the toxicity or other health effects of TCP that may have become available since the 2009 IRIS review.

Keyword searches for "trichloropropane" "TCP" and TCP's CAS number "96-18-4" were entered into the databases of published and unpublished sources listed below.

- Agency for Toxic Substances and Disease Registry (ATSDR)
- Carcinogenic Potency Database (CPDB)
- European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC) - e.g., joint assessments of commodity chemicals reports (JACC Reports)
- European Chemicals Agency (ECHA), formerly European Chemicals Bureau (ECB)
    - European chemical Substances information System (ESIS)
    - International Uniform Chemical Information Database (IUCLID)
- Health Canada
- International Agency for Research on Cancer (IARC)
- International Programme on Chemical Safety (IPCS)
    - Concise International Chemical Assessment Document (CICADS)
    - Environmental Health Criteria (EHC)
    - Joint Expert Committee on Food Additives (JECFA)
    - Joint Meeting on Pesticide Residues (JMPR)
- National Industrial Chemicals Notification and Assessment Scheme (NICNAS)

- National Toxicology Program (NTP)
- Organization for Economic Cooperation and Development, Screening Information Data Sets (SIDS) and SIDS Initial Assessment Report (SIDS; SIARS)
- US Environmental Protection Agency (U.S. EPA)
    - IRIS
    - Toxic Substance Control Act Test Submissions (TSCATS)
- World Health Organization (WHO)
    - Air Quality Guidelines
    - Drinking Water Guidelines
- TOXNET
    - Chemical Carcinogenesis Research Information System (CCRIS)
    - ChemID
    - Genetic Toxicology Data Bank (GENETOX)
    - Hazardous Substances Data Bank (HSDB)
    - TOXLINE

When the option was available, limitations were set to narrow the results to information released between December 2008 and January 2012. Any published or unpublished information identified in the searches was retrieved and reviewed.

Searches in the open literature identified four references that address TCP since the 2009 IRIS review:

Han (2010)

Han (2010) published a case report of a 45 year-old male farmer in China who intentionally ingested 10 to 15 mL of a liquid that was later determined to be TCP. The patient developed nausea, vomiting, loss of consciousness, disturbance, petechiae and fever. He was admitted to a hospital and diagnosed with acute liver failure. Treatments included supplements of glutathione, vitamin K, and pantoprazole and transfusion of blood plasma, platelets and red blood cells. The patient was also found to have hepatitis C and was a habitual alcohol drinker. The patient showed no sign of improvement in the 20 hours following admission, and his family took him home. There was no follow-up of the patient's outcome. Because the exposure was of a single exposure to an unknown concentration of TCP, and the patient had hepatitis C, this case report is not helpful in the assessment of long-term, low concentration exposures to TCP in public drinking water.

Tardiff and Carson (2010)

Tardiff and Carson (2010) published a review of TCP in which they proposed an alternative approach to a cancer hazard assessment of TCP. They stated that the high premature mortality rate and excessive weight loss in rodents indicate that the dose levels that were used exceeded the maximum tolerated dose. They also noted that the doses used were several orders of magnitude higher than likely human exposure to TCP. The authors also suggest that, due to toxicokinetic factors and possible non-genotoxic modes of action, extrapolation of cancer hazards may conform to nonlinear dose-response at dose levels lower than the point of departure. They calculated 50th percentile and lower

90% confidence limit BMD_{10} values of 1.4 and 0.99 mg/kg-day, respectively, for combined tumor sites including rat kidney, oral cavity, mammary gland, preputial gland, and clitoral gland and mouse liver, oral cavity, and uterus. Because the authors suggest a non-linear dose-response for TCP, a cancer value (CV, comparable to a reference dose) of 0.0099 (~0.01) mg/kg-day was calculated based on the 90% confidence limit BMD_{10} (0.99 mg/kg-day) divided by an uncertainty factor of 100, which includes 10x for interspecies variability and 10x for intraspecies (human) variability. The CV was then multiplied by 80 kg to represent adult body weight to produce a lifetime average daily intake (LADI) of 0.79 mg/day. The LADI was then adjusted by a relative source contribution (RSC[a]) value of 50%, and the exposure level was divided by 2 L/day to represent daily tap water consumption. The derived drinking water equivalent level (DWEL) for TCP was 0.20 mg/L (200 µg/L). The DWEL represents the concentration of TCP in tap water below which there is not believed to be an excess cancer risk even when humans are exposed to that concentration for a lifetime.

Current *Guidelines for Carcinogen Risk Assessment* (U.S. EPA 2005a) allow for a non-linear extrapolation of cancer endpoints if there are sufficient data to establish the mode of action and to conclude that it is not linear at low doses. Although the approach proposed by Tardiff and Carson (2010) is innovative, there are insufficient data to support the authors' assumption that cancer incidence related to TCP would be non-linear at low doses. The authors speculate about a background level of endogenous mutations, and that TCP-induced genetic mutations should be considered only at response rates above that background level. They also cite several carcinogens with mutagenic modes of action that have non-linear dose-response curves for mutagenesis, but the compounds cited as examples are not structurally or chemically related to TCP. The authors provide some evidence of possible non-genotoxic modes of action of TCP particularly involving irritation of the forestomach at high dose levels. They admit, however, that whether one mode of action might be dominant or whether different modes of action are activated at different dose levels is unclear. In summary, the current data are not sufficient to support a non-linear approach for regulatory purposes.

As a matter of comparison, Tardiff and Carson (2010) also calculated the carcinogenic potency of TCP by using a linear default assumption for low dose extrapolation (per U.S. EPA, 2005a). Using the same selection of endpoints as in the non-linear approach (rat kidney, oral cavity, mammary gland, preputial gland, and clitoral gland and mouse liver, oral cavity, and uterus), the resulting cancer slope factor was 0.101 $(mg/kg\text{-}day)^{-1}$. The authors believe this value overestimates the risk of cancer to humans. The concentration

[a] Relative source contribution (RSC) is the fraction of exposure to the contaminant that comes from sources other than tap water.

of TCP associated with 10^{-5} (or 1 in 100,000) cancer risk and a cancer slope factor of 0.101 $(mg/kg\text{-}day)^{-1}$ is 3.96 µg/L.

ATSDR (2011)

In 2011, the Agency for Toxic Substances and Disease Registry (ATSDR) published an addendum to its previous Toxicological Profile on TCP (ATSDR 1992). The addendum is intended to be used in conjunction with the 1992 profile and not to replace it. The addendum updated the review of studies related to the health effects of TCP.

Gelhaus et al. (2011)

Gelhaus et al. (2011) published a review of approaches to cancer assessment in EPA's Integrated Risk Information System (IRIS) program. TCP was used as an example of a chemical with a mutagenic mode of carcinogenic action in which age-dependent adjustment factors (ADAF) for early-life exposure would be appropriately applied for determination of lifetime carcinogenic risk. The ADAF method is used in the current risk assessment of TCP (See Section 5).

4. Evaluate Previous Assessments of TCP

The Hawaii Department of Health (HDOH) asked Tetra Tech to contrast and discuss the differences between three existing assessments of the human health risks of TCP in water in Hawaii. The first assessment was conducted in 1992 by Toxicologic Consulting Services under contract to HDOH, with Dr. Robert G. Tardiff as the principal author (Tardiff 1992). Approximately a decade later, Dr. Tardiff re-evaluated the risk of TCP in drinking water to address new data, particularly developments in understanding the mode of action and toxicokinetics of TCP (Tardiff 2001). Subsequent to the 2001 assessment by Dr. Tardiff, Hawaii reduced the MCL of TCP in drinking water from 0.8 µg/L to 0.6 µg/L.

In 2009, the U.S. Environmental Protection Agency's (U.S. EPA) Integrated Risk Information System (IRIS) published a hazard and dose response assessment of TCP (IRIS 2009). In the absence of any clinical or epidemiological data on the effects of TCP in humans, all three assessments (Tardiff 1992, 2001; IRIS 2009) relied on the NTP (1993) chronic oral gavage studies conducted in rats and mice as the critical study on which to base a hazard and dose response assessment. The NTP bioassays were and remain the only carcinogenicity data available on TCP. The differences in the cancer hazard and dose response assessments by Tardiff (1992, 2001) and IRIS (2009) result from differences in how the NTP data are interpreted and applied.

Tardiff 1992

In his 1992 review of TCP, Dr. Tardiff used a linearized multistage (Global 86) model to calculate cancer potency factors for five types of cancer observed in rats and mice in the NTP (1993) study[b]. These cancer types include those observed in the pancreas and kidney in male rats, the mammary gland in female rats, the uterus in female mice, and the liver in male and female mice. Dr. Tardiff did not calculate cancer potency factors for tumors observed in the oral cavity, forestomach, preputial gland, clitoral gland, Zymbal's gland, or Harderian gland. He dismissed the tumors reported in the oral cavity and in the forestomach because these tissues were the "point of application," meaning that when the gavage doses were administered, the oral cavity and forestomach came in contact with the TCP in corn oil before the test material was absorbed and diluted into systemic circulation. The argument is that the oral cavity and forestomach were exposed to higher concentrations of TCP than other tissues, and therefore a different mode of action may have caused the tumors observed in these tissues, compared to presumed genotoxic modes of action in tissues that are distant from the site of application (e.g., liver, kidneys, etc). Dr. Tardiff did not address why he excluded the carcinogenicity incidences in preputial gland, clitoral gland, Zymbal's gland, or Harderian gland in his 1992 analysis; it is presumed that his reasoning was that humans do not have these glands.

The cancer potency factors that Dr. Tardiff reported included maximum likelihood estimate (MLE) values with 95% upper confidence limits (UCLs). Rodent doses were adjusted for human equivalent dose using the (body weight ratio)$^{3/4}$ method (per U.S. EPA 1992). The MLE and 95% UCL values reported by Dr. Tardiff are presented in Table 3 (below). Of the five tumor types analysed by Dr. Tardiff, the response in the pancreas had the greatest cancer potency, with a MLE of 8.65×10^{-2} (mg/kg-day)$^{-1}$.

Table 3. Cancer potency factors for TCP administered orally to rats and mice derived from the linearized multistage model (from Table 2 of Tardiff 1992)

Species	Organ	Tumor Type	MLE (mg/kg-day)$^{-1}$	95 % UCL (mg/kg-day)$^{-1}$
Rat	Pancreas	Adenoma	8.65×10^{-2}	1.20×10^{-1}
Rat	Kidney	Adenoma	5.09×10^{-2}	6.49×10^{-2}
Rat	Mammary Gland	Adenoma	4.07×10^{-2}	5.43×10^{-2}
Mouse	Uterus	Adenoma & Adenoarcinoma	5.98×10^{-3}	1.11×10^{-2}
Mouse	Liver	Cellular Adenoma & Carcinoma	1.56×10^{-2}	3.18×10^{-2}

[b]Dr. Tardiff cited an unpublished 1991 version of the NTP study report.

Tardiff (1992) estimated the exposure levels of TCP to humans on the island of Oahu by reporting measured concentrations of TCP in well water from different water treatment facilities for the years 1989 to 1991. Dr. Tardiff reported that during this time period the average concentration of TCP in ground water was approximately 0.16 µg/L[c]. The samples appear to have all been from source well water. He noted that no TCP had been detected in tap water on Oahu. At that time, the limit of detection for TCP was 0.30 µg/L. The daily TCP exposure estimates of Oahu residents from drinking water was estimated by multiplying the mean concentration of ground water (0.16 µg/L) by the water consumed per day, divided by body weight. Dr. Tardiff made this calculation for two populations: young children, weighing 10 kg, who consume 1 L of water/day and adults, weighting 70 kg, who drink 2 L of water/day. Thus the oral exposure to TCP was 0.016 µg/kg-day (0.16 µg/L x 1 L/day ÷ 10 kg) for children and 0.005 µg/kg-day (0.16 µg/L x 2 L/day ÷ 70 kg) for adults. Because TCP is a volatile organic compound, there is an additional potential for exposure from inhalation and dermal absorption while showering or bathing. Studies of the exposure to another volatile organic compound, chloroform, during showering indicated that inhalation and dermal absorption contributed an internal dose equivalent of 30% of the daily ingested chloroform (Jo et al. 1990a, 1990b). Dr. Tardiff added an assumed 30% to the oral daily drinking water dose of TCP to estimate a total daily exposure of 0.022 µg/kg-day (0.016 µg/kg-day x 1.3) for children and 0.007 µg/kg-day (0.005 µg/kg-day x 1.3) for adults. Chloroform is more volatile[d] than TCP, so the actual atmospheric release of TCP is assumed to be much lower than that of chloroform. Thus, the actual exposure from these parenteral routes is probably much lower than the 30% used in the modeled dose.

By combining the human exposure levels to the estimated cancer potency of pancreatic tumors in rats, Dr. Tardiff calculated the lifetime risk of humans on Oahu developing cancer to be 6 to 7 in 10 million (assuming 70-year exposure to 0.16 µg/L in tap water). Tardiff (1992) proposed a short-term action level of 30 to 300 µg/L, a long term action level of 3.0 µg/L, and a long-term goal of <0.30 µg/L.

Tardiff 2001

In his 2001 review, Dr. Tardiff updated the literature search on the potential human health effects of TCP. New toxicokinetic and mode of action studies available in the preceding decade supported earlier conclusions that TCP is a carcinogen that probably works though a mutagenic DNA-interactive mode of action. Dr. Tardiff noted that the

[c] Note that the estimated concentration of TCP in groundwater reported by Tardiff is below the limit of detection. In his calculations, Tardiff used a value of 0.15 µg/L (half of the limit of detection) to represent samples in which TCP was not detected.

[d] The vapor pressure of TCP is 3.69 mmHg at 25˚C; the vapor pressure of chloroform is 197 mmHg at 25˚C (ChemID Plus database at http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?CHEM, accessed 17 April 2012).

database for TCP still lacked epidemiology studies, and the only available study for estimating carcinogenic risk was still the NTP (1993) study. Again, Dr. Tardiff dismissed tumors in the oral cavity, forestomach, preputial gland, clitoral gland, Zymbal's gland, and Harderian gland reported in the NTP study. As in his 1992 evaluation, Dr. Tardiff based his cancer assessment exclusively on the incidence of pancreatic tumors in TCP-exposed male rats. He used a linearized multistage benchmark dose model to estimate a 10% effective dose (ED_{10}) value of 1.7 mg/kg-day (lower bound on dose = 1.5; upper bound on risk = 0.1). Dr. Tardiff recommended an MCL value of 0.6 µg/L based on the unadjusted ED_{10} value of 1.7 mg/kg-day and a cancer risk of 1 in 1,000,000. This recommended MCL was subsequently adopted by the State of Hawaii.

Dr. Tardiff also estimated the human exposure to TCP in ground water on the islands of Oahu, Maui, and Kauai, based on data from water samples collected from 1993 to 2000. On Oahu, the mean TCP concentrations in ground water samples were 0.09 to 0.27 µg/L. On Maui, the mean concentration was 0.01 µg/L, and on Kauai it was 0.1 µg/L. He noted that the detection limit of TCP in water samples was reduced to 0.04 µg/L since the previous assessment. None of these TCP concentrations would have been included in the 1992 assessment, when the detection limit was 0.3 µg/L (Tardiff 1992). Dr. Tardiff used the mean concentrations of TCP in water to estimate daily human exposure levels. To estimate the oral ingested daily dose, he assumed that an adult weighs 70 kg and consumes 2 L of water per day and a child weighs 10 kg and consumes 1 L of water per day. However, rather than adding 30% of the oral dose to account for inhalation and dermal absorption during showering or bathing that he used in the 1992 review, he estimated the parenteral (non-oral) contribution to be 18 to 20% of the drinking water dose. His reasoning for the 18 to 20% is that TCP is less volatile than chloroform, the compound on which the 30% measured figure was based (Jo et al. 1990a, a990b). Dr. Tardiff estimated the total average doses of TCP in water via all exposure routes to be 0.002 to 0.007 µg/kg-day for Oahu, 0.0003 µg/kg-day for Maui, and 0.003 µg/kg-day for Kauai. Based on these exposures and the cancer risk potency, he calculated the risk of cancer from TCP in tap water to be two to four in 10 million on Oahu, two in 100 million on Maui, and two in 10 million on Kauai. As in the 1992 assessment, the estimated exposure and risk values were based primarily on TCP concentrations in pre-treated source or well water. Treatment of source water with granular activated carbon (GAC) prior to distribution to homes reduces the TCP concentrations in effluent tap water.

IRIS 2009

The U.S. EPA's IRIS program published a Toxicological Review of TCP in 2009 (IRIS 2009). The document reviewed all available published and unpublished data regarding the cancer and non-cancer human health hazards of TCP. As with the Tardiff (1992, 2001) reviews, the IRIS investigators identified no data on effects of TCP in humans and

thus relied on animal and *in vitro* data to represent the hazards of TCP to humans. For non-cancer health effects, IRIS calculated a reference dose of 0.004 mg/kg-day based on increased absolute liver weights in male rats as reported in the chronic toxicity studies of TCP by NTP (1993).

The IRIS (2009) carcinogenicity assessment of TCP was based primarily on the NTP (1993) studies in rats and mice. The quantification of cancer risk (i.e., calculation of a cancer slope factor) was based on multistage-Weibull models of the incidence of tumors of the alimentary system (oral cavity and forestomach), pancreas, kidney, preputial gland, clitoral gland, mammary gland, and Zymbal's gland in F344/N rats and the alimentary system (oral cavity and forestomach), liver, Harderian gland, and uterus in B6C3F1 mice. Tumor risk was calculated for the combined tumor types for each species-sex group, and of these, female mice were found to be the most sensitive species-sex group with an overall cancer slope factor of 30 $(mg/kg\text{-}day)^{-1}$. This oral cancer slope factor is based on incidence of tumors in the alimentary system (including oral cavity and forestomach tumors), liver, Harderian gland and uterus of female mice. IRIS noted that this slope factor should not be used with exposures greater than 0.6 mg/kg-day, because the observed dose-response relationships do not continue linearly above this level.

Among the peer review and public comments on the IRIS report were suggestions that forestomach tumors should not be included in the calculation of an oral cancer slope factor. The arguments for excluding forestomach tumors from the analysis include the following: 1) humans do not have a forestomach or an analogous organ; 2) the bolus dose of a high concentration of TCP in corn oil does not represent any realistic human exposure; and 3) the slowed emptying of the forestomach into the glandular stomach results in a prolonged contact with forestomach epithelial tissues. The IRIS reviewers considered these comments but ultimately decided to retain the forestomach tumor data for their assessment, based on the following considerations: 1) while humans do not have a forestomach, squamous epithelial tissues in the oral cavity and the upper two-thirds of the esophagus in humans are comparable to the rodent forestomach; 2) most genotoxic forestomach carcinogens appear to act through a mutagenic mode of action; and 3) TCP is a multi-site carcinogen that is also genotoxic, meeting recommendations by IARC (2003) for determination that the forestomach tumors are likely relevant to human carcinogenesis. As a matter of comparison, IRIS calculated and reported the oral cancer slope factor values for the incidences of all tumor types except forestomach tumors. By excluding forestomach tumors, the overall cancer slope factors were 1.3 $(mg/kg\text{-}day)^{-1}$ for male rats, 0.9 $(mg/kg\text{-}day)^{-1}$ for female rats, 0.9 $(mg/kg\text{-}day)^{-1}$ for male mice, and 1.3 $(mg/kg\text{-}day)^{-1}$ for female mice. Thus, without inclusion of the forestomach tumors, the male rats and female mice are equally sensitive to overall carcinogenicity. Comparisons of the cancer slope factors calculated by IRIS with and without forestomach tumors are

presented in Table 4 (below). (Note: whether forestomach tumors should be included in the calculation of cancer potency is discussed further in the Forestomach Tumors section of this report, page 18.)

The IRIS review did not address specific exposure concentrations in water or make any recommendation for any water concentration limits (e.g., MCL or action levels).

In accordance with *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b), the IRIS review explains how to apply age-dependent adjustment factors (ADAF) to calculate lifetime cancer risk from a given daily exposure to TCP. ADAF uses weighted factors to emphasize the higher risk of exposures during the earlier stages of life of infancy and childhood. A more complete discussion of the application of ADAF in lifetime risk calculations is provided in the ADAF section on page 27.

Table 4. Comparison of IRIS-calculated overall cancer slope factors per sex per species with and without forestomach tumors (IRIS 2009)

Species	Sex	Cancer slope factor for tumors in all tissues $(mg/kg\text{-}day)^{-1}$	Cancer slope factor for tumors in all tissues except forestomach $(mg/kg\text{-}day)^{-1}$
Rat	Male	4.1	1.3
	Female	1.5	0.9
Mouse	Male	6.8	0.9
	Female	28	1.3

Comparisons of Tardiff and IRIS assessments

The dose response assessments of TCP conducted by Tardiff (1992, 2001) and IRIS (2009) reviewed the same source data (NTP 1993) but used different approaches. The differences are outlined below.

Carcinogenicity Endpoints

Both dose response assessments by Dr. Tardiff (1992, 2001) focused on five specific tumor types: pancreas and kidney in male rats, the mammary gland in female rats, the uterus in female mice, and the liver in mice of both sexes. He selected these specific tumor types because they were located at sites distant from the dose application site (i.e., not part of the GI tract) and the affected tissues in rodents have anatomical analogs in humans. Of the five tumor types considered by Dr. Tardiff, the pancreatic tumor in male rats was the most sensitive endpoint to TCP-induced carcinogenicity. In both reviews of TCP by Tardiff (1992, 2001), incidence of pancreatic tumors in male rats was the endpoint used in quantification of cancer risk.

In contrast to the approach used by Tardiff, IRIS (2009) considered all of the tumor types observed in rats and mice to be relevant to the cancer risk of TCP in humans. The IRIS investigators reported cancer slope factors for each tumor type per species-sex group (i.e., male rats, female rats, male mice, and female mice) and the overall combined cancer slope factor for each species-sex group. Using this approach, female mice were the most sensitive species-sex group. In response to peer reviewer and public comments that it was not appropriate to include the forestomach tumor data in cancer potency calculation, IRIS also calculated cancer slope factors based on all tumor types except the forestomach. However, the final oral cancer slope factor for TCP endorsed by IRIS is based on multiple tumor types in female mice.

Dose Adjustment Factors

In each of the reviews (Tardiff 1992, 2001; IRIS 2009), the doses of TCP administered to rodents was adjusted to human equivalent dose by the (body weight ratio)$^{3/4}$ method described in the *Draft Report: A Cross-Species Scaling Factor for Carcinogen Risk Assessment Based on Equivalence of mg/kg$^{3/4}$/Day* in Volume 57, Number 109, of the Federal Register (U.S. EPA 1992).

The second Tardiff (2001) review of TCP considered three ED_{10} values. The first ED_{10} was based on male rat dose levels with no adjustments. The second ED_{10} value was based on body weight scaling (per U.S. EPA 1992) of male rat dose levels. The third proposed ED_{10} was based on ¾ body weight scaling and a 10x factor for the slower metabolic rate in human liver tissue. The adjustment for metabolic rate was based on the study by Weber and Sipes (1992) in which TCP was reportedly bioactivated to a reactive metabolite (1,3-dichloroacetone, DCA) at a rate ten-times faster in rat liver microsomes than in human liver microsomes *in vitro*. Tardiff (2001) reasoned that this metabolic difference made humans less susceptible to TCP carcinogenicity than rats, whereas most dose adjustments presume that humans are more susceptible. His ultimate recommendation for an MCL appears to be based on the first ED_{10}, (male rat dose levels with no adjustments).

In addition to the body weight scaling adjustment, the IRIS (2009) review of TCP also adjusted dose levels to reflect daily exposure to TCP (dose levels multiplied by 5/7). This was done because in the NTP (1993) studies, rats and mice were dosed five days per week, but humans are likely to be exposed seven days per week. Neither of the Tardiff reviews made this adjustment.

Cancer Potency

In his 2001 report, Dr. Tardiff reported cancer potency in terms of ED_{10} values with units of mg/kg-day. In his 1992 report, he reported cancer potency in terms of a MLE with 95 % UCL in units of (mg/kg-day)$^{-1}$, which are the units of a cancer slope factor used by the

U.S. EPA (IRIS 2009). It is not clear why Dr. Tardiff (2001) did not report cancer potency in terms of a cancer slope factor in units of $(mg/kg\text{-}day)^{-1}$. Table 5 (below) summarizes the cancer potency values proposed by Tardiff (1992, 2001) and IRIS (2009).

Table 5. Summary of Cancer Potency Values Proposed by Tardiff and IRIS

	Tardiff 1992	Tardiff 2001	IRIS 2009
Critical Study	NTP 1992; Irwin et al. 1995	NTP 1992; Irwin et al. 1995	NTP 1992; Irwin et al. 1995
Critical Endpoint	Pancreatic tumors in male rats	Pancreatic tumors in male rats	Combined tumors in female mice
Dose Adjustment Factors	(body weight ratio)$^{3/4}$	none	(body weight ratio)$^{3/4}$ and 5/7 for dose frequency
Proposed Cancer Slope Factor	CSF = 0.12 $(mg/kg\text{-}day)^{-1}$	CSF = 0.0588 $(mg/kg\text{-}day)^{-1}$[a]	CSF = 30 $(mg/kg\text{-}day)^{-1}$

CSF = Cancer slope factor; ED_{10} = 10% effective dose.
a. The ED_{10} value of 1.7 mg/kg-day was converted to a cancer slope factor, assuming a linear relationship from the ED_{10} to the zero:zero origin (per U.S. EPA 2005a): 0.1 ÷ 1.7 mg/kg-day = 0.0588 $(mg/kg\text{-}day)^{-1}$.

5. Quantitative Risk Assessment of TCP in Water

The quantitative assessment of carcinogenic risk has two primary components: a dose response assessment and an exposure assessment. This section presents discussions of the approaches used in the current dose response and exposure assessments.

Dose Response Assessment

As stated previously, there are no human or epidemiological studies on the health effects of TCP. There are non-cancer health effects associated with TCP in animals. Since TCP has been determined to be a carcinogen in animals and is presumed to be a possible carcinogen in humans, cancer is the primary health effect of interest on which to base regulatory decisions. The NTP (1993) chronic bioassay of TCP in rats and mice provides the only available information on carcinogenicity; some subchronic and *in vitro* studies provide additional details for consideration in the toxicological evaluation.

Forestomach Tumors

The inclusion of forestomach tumors in the cancer dose response assessment is a controversial issue. The following are considerations regarding the inclusion of the forestomach tumors observed in the NTP (1993) chronic bioassays of TCP in rats and mice.

Anatomical Differences

Humans do not have a forestomach (IARC, 2003; Proctor et al., 2007; Chandra et al. 2010). Humans have histologically analogous tissue types in the esophagus, but in rodents the forestomach is a storage compartment where materials have prolonged contact with the lumen epithelial tissues (IARC, 2003; Proctor et al., 2007). The histologically analogous human esophagus is a passageway and not a storage compartment, thus ingested materials have only brief contact with the lumen epithelial tissues. Therefore, while the tissue types of the rodent forestomach and the human esophagus may be similar, the differences in the functions of these organs make the rodent forestomach anatomically not applicable to the human health hazard assessment of TCP.

Route of Administration

Ideally, the risk assessment of TCP in drinking water would be based on human data. Barring the availability of human data, the risk assessment is best derived from chronic exposure studies using drinking water as a route of administration. Irwin et al. (1995) stated that the limited aqueous solubility of TCP precluded administration in drinking water. Because a chronic drinking water study is not available, the chronic oral gavage studies of TCP in rats and mice (NTP 1993) serve as a substitute. However, the route of administration of TCP in rats and mice used by NTP may have influenced effects in rat and mouse forestomachs that are not relevant to human exposure to TCP in tap water.

The NTP (1993) studies used corn oil as a vehicle because TCP is not easily soluble in water. IARC (2003) reported that oral gavage with corn oil as the vehicle results in longer retention of the administered dose in the forestomach than when the agent is given in water (i.e., emptying of the forestomach into the glandular stomach is a slower process when the forestomach is full of corn oil). The IRIS (2009) report mentions this effect of corn oil on the forestomach but states that the effect of this increased retention time in the forestomach is unknown. Citing examples of ethylene dibromide, dibromochloropropane, and chloroform (each of which has structural similarities to TCP), La et al. (1996) stated that "gavage administration of chemicals produces greater incidence of forestomach tumors compared to other routes of administration."

In a toxicokinetics study by Mahmood et al. (1991), 30 mg/kg [2-^{14}C]-TCP was administered to rats and mice by corn oil gavage. The presence of detectable TCP in different tissues was measured 6, 24, and 60 hours post-administration. Most of the radiolabeled TCP was found in the forestomach and glandular stomach with smaller quantities in the intestines, adipose tissue, liver, and kidneys. At 24 hours, the concentrations of radiolabelled TCP and/or metabolites in the forestomach, intestines, liver, and kidney were similar. At 60 hours after administration, the majority of the radiolabel was excreted in the urine or feces. The persistence of TCP in the rodent

forestomach for at least 24 hours after administration in corn oil is not representative of the brief exposure likely to be experienced by the human esophagus while drinking water containing TCP at much lower concentrations.

Villeneuve et al. (1985) administered drinking water with concentrations of 0, 1, 10, 100, or 1,000 mg/L TCP to Sprague-Dawley rats for 90 days. The dose levels were approximately equal to 0, 0.13, 1.3, 11.3, and 113 mg/kg-day. There were signs of toxicity in the liver and kidney, but no effects on the forestomach were reported.

TCP in corn oil was administered by oral gavage 5 days/week to F344/N rats and B6C3F1 mice for 120 days at doses of 0, 8, 16, 32, 63, 125, or 250 mg/kg-day (NTP 1993). In rats, there were signs of toxicity in the liver, kidney, and nasal epithelium, but no effects on the forestomach were reported. Acanthosis (hyperplasia) and hyperkeratosis of the forestomach were observed in mice at the 125 and 250 mg/kg-day dose levels.

Merrick et al. (1991) administered 0, 1.5, 7.4, 15, or 60 mg/kg-day TCP in corn oil to Sprague-Dawley rats by oral gavage for 90 days. The report focused on cardiotoxicity and did not report any effects in the forestomach.

Comparison of the drinking water study by Villeneuve et al. (1985) and the gavage studies by NTP (1993) and Merrick et al. (1991) show no signs of irritation effects in rats by either route. Hyperplasia and hyperkeratosis of the forestomach were reported in mice given TCP by corn oil gavage (NTP 1993). These effects occurred at dose levels higher than the highest dose in the Villeneuve et al. (1985) drinking water study, so a comparison regarding the effect of dose route of TCP exposure on forestomach health cannot be made.

In a study by La et al. (1996), male B6C3F1 mice were given equivalent doses of 6 mg/kg-day ^{14}C-TCP for 5 days via either corn oil gavage or drinking water. DNA adducts and cellular proliferation rates were measured in forestomach tissues. Mice that received TCP by oral gavage in corn oil had a higher mean number of DNA adducts in the forestomach tissue than mice receiving TCP by drinking water, although this difference was not statistically significant. The mice that received TCP by oral gavage in corn oil had a statistically significantly higher cellular proliferation of forestomach tissue than mice given the equivalent dose in drinking water. The dose of 6 mg/kg-day in this five-day study (La et al. 1996) is the same as the lowest dose used in the 2-year mouse carcinogenicity study in which forestomach tumors were induced at this dose level. Additionally, mice given TCP by corn oil gavage had between 1.4 and 2.4 times more of TCP's major metabolite (a reactive glutathione conjugate) than mice receiving TCP in drinking water. The concentration of TCP in the drinking water was not reported but can

be estimated to be 1.5 mg/L[e], which is nearly 2000 times the current MCL for TCP in Hawaii (0.0006 mg/L). The findings in the La et al. (1996) study indicate that the corn oil gavage administration of TCP may exaggerate the toxicological potency of TCP in human exposures via tap water.

Dose Selection

The current *Guidelines for Carcinogen Risk Assessment* (U.S. EPA 2005a) recommend that studies in which excessively high doses are used should be interpreted with caution when extrapolating the results to humans. Tardiff and Carson (2010) noted that the high number of premature deaths and excessive body weight loss in the chronic rat NTP (1993) study suggest that the doses exceeded the maximum tolerated dose (MTD) for TCP. The implication of this to an analysis of a dose response is that there is great uncertainty about extrapolation of carcinogenic or toxic responses at doses below the lowest dose level. Proctor et al. (2007) stated that forestomach tumors that occur at doses that exceed the MTD should not be considered relevant for human risk assessment.

Internal Exposure

The concentration of TCP at the lowest dose in the chronic rat and mouse studies (NTP 1993) was 0.6 mg/mL[f] (600,000 µg/L), whereas the maximum and average concentration of TCP found in treated tap water in Hawaii between 2000 and 2011 were 0.81 µg/L and 0.037 µg/L, respectively (HDOH 2011). Thus, at the lowest doses tested, the forestomachs of rats and mice in the NTP studies were exposed to concentrations of TCP that were between 740,000 and 1,620,000 times what the human esophagus or stomach would be exposed to via drinking tap water in Hawaii. Also, the gavage doses were administered as a forced bolus once a day, whereas the human exposure would be in about 2 liters of water consumed throughout the day. The extremely high concentration of a chemical in corn oil deposited into the forestomach with a prolonged direct contact with epithelial lining is not representative of the momentary passing of an extremely low concentration of the same chemical in water through the human esophagus.

Mode of Action

IARC (2003) and Proctor et al. (2007) state that mode of action is an essential factor in determining whether forestomach carcinogenicity is relevant to human cancer risk. TCP was found to be positive in *in vitro* assays of genetic toxicity (NTP 2003). In general,

[e] According to the U.S. EPA's Biological Reference Values (U.S. EPA 1988), the average water consumption rate for male B6C3F1 mice is 0.25 L/kg-day. 6 mg/kg-day ÷ 0.25 L/kg-day = 1.5 mg/L

[f] The target concentration for the lowest dose levels in the rat and mouse studies was 0.65 mg TCP/g corn oil (NTP 1993). The density of corn oil is 0.9 g/mL (Lewis 1997). Thus, 0.65 mg/g x 0.9 g/mL = 0.6 mg/mL.

forestomach tumors caused by a chemical that is known to be a mutagen or interact with DNA while causing tumors at multiple tissue sites would be deemed to be relevant to humans, however dosimetric factors must also be considered. In the case of TCP, multiple dosimetric factors such as the irritating nature of oral gavage, the extremely high concentrations of TCP administered, use of doses that exceeded the MTD, and the prolonged exposure to forestomach tissues suggest that the forestomach tumors may be due to a non-genotoxic mode of action. Proctor et al. (2007) explained that repeated gavage doses of high concentrations of a chemical can irritate the epithelial lining of the forestomach. Tardiff and Carson (2010) suggested that this is likely to be the case for TCP, and that forestomach hyperplasia and inflammation leads to tumorigenic expression through a tumor promotion mechanism such as reparative hyperplasia. Hyperplasia and hyperkeratosis, which are signs of chronic irritation, in the forestomach were observed in mice that were given TCP by corn oil gavage daily for 8 or 17 weeks (NTP 1993).

There is evidence that TCP is irritating to epithelial tissues in other parts of the body. TCP was found to be an "intense skin irritant" in rabbits (McOmie and Barnes 1949). Silverman et al. (1946) reported that humans (12/sex) exposed to air containing 1000 ppm TCP for 15 minutes experienced irritation of the eyes and throat. A single 4-hour exposure to 126 ppm TCP vapor caused eye irritation in rats and mice (Gushow and Quast 1984). Repeated intermittent exposure to vapor concentrations as low as 15 ppm for 13 weeks (Johannsen et al. 1988) caused irritation of the eyes and respiratory tract in rats. It is reasonable to assume that a chemical that is irritating to the skin, eyes, and respiratory tract would also be irritating to the cells lining the forestomach. Chandra et al. (2010) stated that chronic inflammation or local irritation of forestomach mucosa associated with high concentrations of a chemical, may play a role in continuous induction of cell proliferation and the ultimate development of carcinomas.

The major DNA adduct that formed in the forestomach after oral administration of TCP to mice was identified as S-[1-(hydroxymethyl)-2-(N7-guanyl)ethyl]glutathione (La et al. 1996). The authors state that covalent binding to the N7 position of guanine generally does not lead to miscoding of genes. Boysen et al. (2009) indicates that N7-guanine adducts are formed frequently endogenously, increase with age, do not persist, and are unlikely to be mutagenic. As stated by Tardiff and Carson (2010), “these types of adducts are considered to have minimal biological relevance in relationship to exposure to mutagenic carcinogens.”

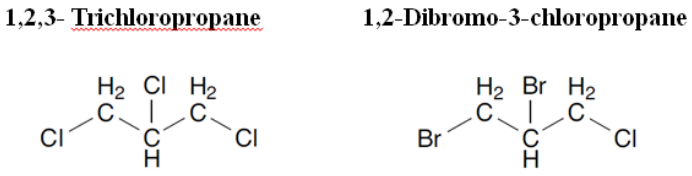
IRIS (2009) proposed that the mode of action for TCP tumorigenicity involves mutagenicity via reactive metabolites, but the IRIS reviewers also noted that the mode of action of TCP-induced forestomach tumors may include promotion. The use of corn oil as a vehicle for the administration of carcinogenic chemicals has been shown to increase

the incidence and severity of epithelial cell proliferation of the forestomach in rats (Ghanayem et al., 1986, as cited by IRIS 2009).

Analogous Compound

1,2-dibromo-3-chloropropane (DBCP) is structurally analogous to TCP (see Figure 1 below) and has also been used agriculturally as a nematodicide (NTP 2011). Like TCP, IARC classified DBCP as possibly carcinogenic to humans (Group 2B) (IARC 1999). DBCP is classified as reasonably anticipated to be a human carcinogen in the NTP's *Report on Carcinogens* (NTP 2011). The U.S. EPA's IRIS program has not evaluated the carcinogenicity of DBCP.

Figure 1. Comparison of Chemical Structures of TCP and DBCP (NTP 2011)



The National Cancer Institute (NCI 1978) performed chronic bioassays of DBCP in which Osborne-Mendel rats and B6C3F1 mice were administered DBCP in corn oil by oral gavage for up to 78 weeks. A high incidence (93 to 100%) of forestomach tumors occurred in all DBCP treatment groups in both sexes of both species. The incidence of squamous cell carcinomas of the forestomach was statistically higher with DBCP treatment in rats and mice of both sexes. These responses are qualitatively similar to those produced by TCP. The NCI (1978) report stated that the method of administration might have played a role in inducing these neoplasms, as DBCP is a skin and mucous membrane irritant and proliferative lesions of the forestomach have been noted in other bioassays where chemical irritants have been administered by gavage.

No chronic drinking water studies of DBCP were identified, but a chronic dietary carcinogenicity bioassay of DBCP in Charles River rats was conducted by Hazleton Laboratories America, Inc. (1977, cited in WHO 1996). Male and female rats had significantly increased incidences of carcinoma of the renal tubules and squamous cell carcinoma of the glandular stomach. Male rats also showed an increase in liver tumors. Forestomach tumors did not occur in the chronic feeding study, which is dosimetrically more similar to drinking water than oral gavage is.

The World Health Organization (WHO 1996) published a background document for its *Guidelines for Drinking Water Quality* which included a dose response analysis of carcinogenicity studies for DBCP. WHO based its quantitative carcinogenicity

assessment of DBCP on the incidence of stomach, kidney, and liver tumors in the dietary study. WHO (1996) did not consider the forestomach tumors from the corn oil gavage studies in its estimations of cancer risk from human consumption of DBCP-contaminated water. An analogous chronic dietary study is not available for TCP, but the similarities between the two compounds in terms of chemical structure and carcinogenic responses in corn oil gavage to rodents suggest that they may act similarly in a non-bolus route of administration. Therefore, the example of WHO's approach to DBCP supports the exclusion of forestomach tumors in the development of a MCL for TCP.

Conclusions Regarding Forestomach Tumors

IRIS (2009) concluded that the forestomach tumors in rats and mice chronically exposed to TCP were relevant to human cancer risk. However, there are challenges to this conclusion that are worth consideration.

While IRIS and IARC affirm that the rodent forestomach is histologically analogous to the human esophagus, the two organs have functional differences (storage containment versus passageway) that make them less analogous for the purposes of cancer risk assessment. The gavage route of administration of TCP in a corn oil vehicle poorly represents consumption of TCP in drinking water and appears to overpredict the risk of cancer in the forestomach. In subchronic studies of TCP, the gavage route produced toxicities that were not seen in drinking water exposures, and the LOELs in the gavage studies were lower than in the drinking water studies.

The IRIS (2009) cancer slope factor of 30 $(mg/kg\text{-}day)^{-1}$, based on forestomach tumors in female mice, would appear to overpredict the carcinogenic risk of TCP. However, in the absence of a more appropriate chronic exposure study, caution should be taken in excluding the forestomach tumor data completely.

Alternative Cancer Slope Factor for TCP

In the absence of a chronic bioassay of TCP in drinking water at appropriate dose levels (e.g., dose levels that do not exceed the MTD), the relevance of forestomach tumors in the rodent gavage model to human exposures is debatable. A compromise approach would be to include all tumor types in the risk assessment, including the forestomach tumors, and base the final cancer slope factor on the geometric mean of the cancer slope factors for the four species-sex categories (male and female rats and mice). The geometric mean of the overall cancer slope factors reported in Table 5-5 of the IRIS (2009) review of TCP is 5.8 $(mg/kg\text{-}day)^{-1}$. See Table 6 (next page).

The U.S. EPA used this geometric mean approach in its carcinogenicity dose response assessment of the pesticide dichlorvos, for which the IRIS reviewers considered all of the tumor findings (forestomach in mice, pancreatic and leukemia in rats) to be of equal

relevance, rather than selecting a slope factor considered the most relevant or selecting the highest slope factor (IRIS 1994; Proctor et al. 2007).

Table 6. Geometric Mean of Cancer Slope Factors of TCP

Species	Sex	Overall cancer slope factor for tumors in all tissues $(mg/kg\text{-}day)^{-1}$
Rat	Male	4.1
	Female	1.5
Mouse	Male	6.8
	Female	28
Geometric Mean		5.84973

Human Exposure Models of TCP in Tap Water

Exposure to TCP in tap water[g] is presumed to occur through three routes: ingestion, inhalation, and dermal absorption. Ingestion would occur primarily through drinking water. There is also potential for ingestion of TCP through foods that were prepared with contaminated tap water, but food was not included in the ingestion model. Inhalation and transdermal exposures are presumed to occur during showering and bathing. The mathematical models used to estimate the exposure to TCP in tap water via the oral, inhalation, and transdermal routes are described in detail in Appendix A. An attempt has been made to select values that describe "typical" conditions (e.g. the *Exposure Factors Handbook*, U.S. EPA 2011a).

Because age-dependent adjustment factors apply in the risk assessment, the exposure from each route of administration must be calculated separately for infants of less than 2 years of age, children of ages 2 to 16 years, and adults 16 years and older.

Table 7 (next page) presents the total daily exposures for each age group for a range of TCP concentrations in water. The range of 0.01 to 1.0 µg/L was selected to encompass the measured TCP concentrations in tap water reported by HDOH (2011a; see Table 13 on page 38). Total daily exposure is approximately proportional to concentration of TCP in tap water.

[g] "Tap water" refers to water that is delivered to a household plumbing system, including water that is filtered at a water treatment facility or obtained from a private well.

Table 7. Total Daily Exposures to TCP per Age Group for 0.01 to 1.0 µg/L TCP in Tap Water

TCP Conc in Water (µg/L)	Total Daily Exposure to TCP (mg/kg-day)		
	Adult, >16 yrs	Child, 2-16 yrs	Infant, < 2 yrs
0.01	0.00000037	0.00000039	0.000001
0.04 (detection limit)	0.0000015	0.0000015	0.000004
0.10	0.0000037	0.0000039	0.00001
0.20	0.0000074	0.0000077	0.00002
0.30	0.000011	0.000016	0.00003
0.40	0.000015	0.000015	0.00004
0.50	0.000019	0.000019	0.00005
0.60 (current MCL)	0.000022	0.000023	0.00006
0.70	0.000026	0.000027	0.000071
0.80	0.000029	0.000031	0.000081
0.90	0.000033	0.000035	0.000091
1.0	0.000037	0.000039	0.00010

Comparison to Tardiff (2001) Exposure Estimate

In his risk assessment of TCP in Hawaii groundwater, Tardiff (2001) calculated exposure to TCP by using the generic equation for lifetime average daily dose (LADD), which is LADD = (C x IF x AF) ÷ BW, where C is concentration of the TCP in water (µg/L); IF is intake factor (assumed 2 L/day for adults, 1 L/day for children); AF is absorption factor (unitless, assumed 90% absorption of ingested TCP); and BW is body weight (assumed 70 kg for adults, 10 kg for young children). This is essentially the same formula used for estimating oral ingestion exposure used in the current exposure models (see Appendix A). One difference is that in the current assessment, the adult BW value of 80 kg[h] was used rather than the standard default of 70 kg.

While the current exposure assessment uses exposure models for inhalation and transdermal absorption, Tardiff (2001) estimated that 18% to 20% of the daily ingested dose can be added to account for the amount of TCP that is absorbed by the inhalation and transdermal routes during showering and bathing. Based on the conservative exposure models calculated here, the combined amount of TCP exposure from inhalation and transdermal absorption during showering/bathing is equal to approximately 3.5%, 2.5%, and 9.1% of the orally ingested TCP for adults, children, and infants, respectively. Despite the differences in approach, default values, and assumptions, the overall TCP exposure estimated in the current assessment is similar to exposures estimated by Tardiff

[h] Based on data in the current *Exposure Factors Handbook* (US EPA 2011a).

(2001). Table 8 (below) illustrates the estimated exposure levels for an example concentration of 0.1 µg/L TCP in water.

Table 8. Comparison of Average Daily Dose of TCP in Water (mg/kg-day) by Sum of Ingestion, Inhalation, and Transdermal Absorption, Assumes 0.1 µg/L TCP in Water.

	Adult, >16 yrs	Child, 2-16 yrs	Infant, < 2 yrs
Tardiff (2001)[i]	0.0000031	Not calculated[j]	0.00001
Current Assessment	0.0000037	0.0000039	0.00001

Age Dependent Adjustment Factors

Regardless of which value one uses as a cancer slope factor, the estimation of carcinogenic potency of TCP is based on data from adult exposures. Because the database on TCP suggests a mutagenic mode of action for carcinogenicity and there are no chemical-specific data to evaluate differences in susceptibility during early-life, the current U.S. EPA guidelines recommend that age-dependent adjustment factors (ADAFs) be applied in accordance with the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b). This guidance establishes ADAFs for three specific age groups, specifically a 10x factor for young children less than 2 years of age, a 3x factor from children aged 2 to <16 years, and a 1x factor for adults 16 years and above. The 10-fold and 3-fold adjustments in slope factor are combined with age specific exposure estimates when estimating cancer risks from early life (<16 years age) exposure to TCP.

To demonstrate the use of the ADAFs, sample calculations are presented in Table 9 (next page) for three exposure duration scenarios, including full lifetime, assuming a constant TCP exposure to 0.001 mg/L TCP in tap water. This example uses the IRIS-derived cancer slope factor (unit risk) of 30 $(mg/kg\text{-}day)^{-1}$ and the estimated exposure levels per age group at 1 µg/L for TCP, calculated as reported in Table 7.

[i] Tardiff (2001) reported adult and child oral doses of 0.0026 and 0.009 µg/kg-day, respectively, with 0.1µg/L; 20% was added to these values to estimate total exposure from all three routes of exposure.

[j] The *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b) that defined these age ranges was published after the Tardiff (2001) report.

Table 9. Application of ADAFs for a 70-year exposure to 0.001 mg/L TCP from ages 0 to 70

Age Group	ADAF	Unit Risk $(mg/kg\text{-}day)^{-1}$	Exposure concentration (mg/kg-day)	Duration Adjustment	Partial Risk
0 - <2 yrs	10	30	0.00010	2 yrs/70 yrs	0.000864248
2 - <16yrs	3	30	0.000039	14 yrs/70 yrs	0.000693144
≥ 16 yrs	1	30	0.000037	54 yrs/70 yrs	0.00085581
				Total Risk	0.0024132

The partial risk for each age group is the product of the values in the columns of Table 9 (e.g., 10 × 30 × 0.001 × 2/70 = 0.000864248 for young children less than 2 years old), and the Total Risk is the sum of the partial risks. Thus for a lifetime exposure to 1 µg/L TCP in tap water from birth to age 70 and using the IRIS-recommended cancer slope factor of 30 $(mg/kg\text{-}day)^{-1}$, the estimated risk of developing cancer is 0.0024, or 1 in 416.

The ADAF value for each life stage is fixed, but the exposure concentration, exposure duration, and duration adjustment can be varied for different exposure scenarios. Unit risk should be fixed once a cancer slope factor is established, based on an appropriate quantitative hazard assessment. The ADAF methodology was used in determination of cancer risk in the derivation of the proposed MCL in Section 6 of this report.

6. MCL for TCP in Hawaii

Because TCP is considered a probable human carcinogen with a presumed genotoxic mechanism, the U.S. EPA's Office of Water would likely require the Maximum Contaminant Level Goal (MCLG) to be set at zero mg/L. However, because of limitations in technology available to remove and detect extremely small concentrations of a material, the MCL cannot realistically be zero. The analysis of technological and economic limitations of removing TCP from public water works is beyond the scope of the current project. Alternatively, this report provides estimates of the cancer risk of different concentrations of TCP in tap water and some perspective regarding these risks.

The cancer slope factor of 30 $(mg/kg\text{-}day)^{-1}$ proposed by IRIS (2009) is based on forestomach tumors in the female mouse and likely over-predicts the true cancer potency of TCP in humans. If the forestomach tumors are excluded from consideration, the cancer slope factor is 1.3 $(mg/kg\text{-}day)^{-1}$. In the absence of the data necessary to settle the forestomach tumor question, a compromise approach is proposed. This approach combines the dose response for all tumor sites (including forestomach tumors) for both mice and rats; the resulting cancer slope factor is 5.8 $(mg/kg\text{-}day)^{-1}$. As indicated previously, this approach has precedence in EPA's approach to the cancer slope value for dichlorovos.

Calculation of Cancer Risk

Table 10 (next page) compares the lifetime cancer risk associated with different cancer slope factors for the range of TCP concentrations that have been measured in public water in Hawaii. The various cancer slope factors range from 0.0588 $(mg/kg\text{-}day)^{-1}$ (Tardiff 2001) to 30 $(mg/kg\text{-}day)^{-1}$ (IRIS 2009), a range that covers almost 3 orders of magnitude.

Considerations for an MCL for TCP in Hawaii

With a few notable exceptions, regulation of contaminants in water are generally established with theoretical cancer risk estimates within the range of 10^{-6} to 10^{-4}. Table 11 (page 31) presents the TCP concentration in water that are associated with the cancer risk levels of 10^{-4}, 10^{-5}, and 10^{-6} for cancer slope factors summarized in Table 10. At an estimated cancer risk level of 10^{-5}, the TCP concentration is below limit of detection for the two most conservative cancer slope factors of 30 and 5.8 $(mg/kg\text{-}day)^{-1}$. The TCP concentrations at cancer risk level of 10^{-4}, are within the detectable range in water and range from 0.042 to 1.0 µg/L.

Section 8 of this report discusses the TCP concentrations of water samples collected at 16 water treatment facilities in Hawaii between 2000 and 2011 (HDOH 2011a). Over the 11-year period, the average TCP concentrations in treated tap water at water treatment facilities were 0.034 µg/L on Maui, 0.043 µg/L on Oahu, 0.037 µg/L on Kauai, and 0.037 µg/L for the three islands combined (HDOH 2011a). These average TCP concentrations are close to the lower detection limit of 0.04 µg/L and probably overestimate the actual TCP concentrations, because non-detected samples were assigned a value one-half of the lower detection limit. Of the 998 tap water samples reported, 296 samples (30%) had detectable TCP.

Table 10. Estimated Lifetime Cancer Risk of TCP in Water Based on Five Possible Cancer Slope Factor Values.

TCP Concentration in Water (µg/L)	Total Lifetime Cancer Risk, based on ADAF				
	CSF=30 per mg/kg-day (all tumor types in female mice; IRIS 2009)	CSF=5.8 per mg/kg-day[a] (all tumor types in rats and mice of both sexes; current review)	CSF=1.3 per mg/kg-day (all tumor types in female mice and male rats, excludes forestomach tumors; IRIS 2009)	CSF = 0.12 per mg/kg-day (pancreatic tumors in male rats; Tardiff 1992)	CSF = 0.0588 per mg/kg-day (pancreatic tumors in male rats; Tardiff 2001)
0.01	2.4×10^{-5}	4.7×10^{-6}	1.0×10^{-6}	9.7×10^{-8}	4.7×10^{-8}
0.04 (detection limit)	9.7×10^{-5}	1.9×10^{-5}	4.2×10^{-6}	3.9×10^{-7}	1.9×10^{-7}
0.10	2.4×10^{-4}	4.7×10^{-5}	1.0×10^{-5}	9.7×10^{-7}	4.7×10^{-7}
0.20	4.8×10^{-4}	9.3×10^{-5}	2.1×10^{-5}	1.9×10^{-6}	9.5×10^{-7}
0.30	7.2×10^{-4}	1.4×10^{-4}	3.1×10^{-5}	2.9×10^{-6}	1.4×10^{-6}
0.40	9.7×10^{-4}	1.9×10^{-4}	4.2×10^{-5}	3.9×10^{-6}	1.9×10^{-6}
0.50	1.2×10^{-3}	2.3×10^{-4}	5.2×10^{-5}	4.8×10^{-6}	2.4×10^{-6}
0.60 (current MCL)	1.4×10^{-3}	2.8×10^{-4}	6.3×10^{-5}	5.8×10^{-6}	2.8×10^{-6}
0.70	1.6×10^{-3}	3.2×10^{-4}	7.3×10^{-5}	6.8×10^{-6}	3.3×10^{-6}
0.80	1.9×10^{-3}	3.7×10^{-4}	8.4×10^{-5}	7.7×10^{-6}	3.8×10^{-6}
0.90	2.2×10^{-3}	4.2×10^{-4}	9.4×10^{-5}	8.6×10^{-6}	4.3×10^{-6}
1.0	2.4×10^{-3}	4.7×10^{-4}	1.0×10^{-4}	9.7×10^{-6}	4.7×10^{-6}

Table 11. TCP Concentrations in Tap Water Associated with Cancer Risk

Cancer Risk Level	TCP Concentration in Tap Water (µg/L)				
	CSF=30 per mg/kg-day (IRIS 2009)	CSF=5.8 per mg/kg-day (current rview)	CSF=1.3 per mg/kg-day (IRIS 2009)	CSF = 0.12 per mg/kg-day (Tardiff 1992)	CSF = 0.0588 per mg/kg-day (Tardiff 2001)
10^{-4} (1 in 10,000)	0.042	0.22	1.0	11	22
10^{-5} (1 in 100,000)	0.0042	0.022	0.1	1.1	2.2
10^{-6} (1 in 1,000,000)	0.00042	0.0022	0.01	0.11	0.22
Shaded cells indicate TCP concentrations that are below the lower limit of detection.					

It is important for the reader to have a perspective on the meaning of a 10^{-4} theoretical excess lifetime cancer risk. Theoretical excess lifetime risks are estimated from a high dose exposure and assume a lifetime of exposure. The theoretical relative risk can be expressed as the (excess lifetime risk + background risk) ÷ background risk. The lifetime (background) risk of all cancer in the U.S. population (through age 85) is 0.3667 (NCI 2012). If the theoretical excess lifetime risk is 10^{-4}, then the relative risk is approximately 1.00027 (=0.3667 + 1 x 10^{-4}/0.3667). The population needed to detect such a risk would be 743,812,362[k]. The population of Hawaii is about 1.3 million (U.S. Census 2010). Thus it would take several hundred times the population of Hawaii to even detect a theoretical excess lifetime cancer risk of 10^{-4}.

The regulation of water contaminants at cancer risk levels greater than 10^{-4} is not unprecedented. For example, the MCL for inorganic arsenic, a known human carcinogen, is 10 µg/L (HDOH 2009b), which is associated with an estimated cancer risk of 5 x 10^{-4}[l].

Table 12 (next page) provides the current MCL values for several halogenated alkanes that are structurally similar to TCP. Because the determination of a MCL for a particular chemical is based on an analysis of health effects, and technological and economic

[k] Calculated with WinPepi, Compare2, Sample Size, comparison of proportions (S1) to calculate sample size (Abramson 2011).

[l] The drinking water unit risk for cancer is 0.00005 (µg/L)$^{-1}$ for inorganic arsenic (U.S. EPA 1998); 10 µg/L x 0.00005 (µg/L)$^{-1}$ = 0.0005.

factors, there is not a simple relationship between the dose response assessment (cancer slope factor) and the MCL value.

Table 12. Current MCL Values of Halogenated Alkanes Similar to TCP

Compound	U.S. EPA Cancer Slope Factor[a] $(mg/kg\text{-}day)^{-1}$	U.S. EPA MCLG[b,c] (µg/L)	U.S. EPA MCL[c] (µg/L)	State of Hawaii MCL[d] (µg/L)	U.S. EPA Health Advisory[c,e] (µg/L at 10^{-4} cancer risk)
Dibromochloropropane	-	0	0.2	0.04	3
1,2-Dichloropropane	-	0	5	5	60
1,3-Dichloropropene	0.05	-	-	-	40
1,2- Dichloroethane	-	0	5	5	40
1,2- Dibromoethane	2	0	0.05	0.04	2
1,1,1-Trichloroethane	-	200	200	200	-
1,1,2-Trichloroethane	0.057	3	5	5	60
Trichloroethylene	0.046	0	5	5	300
1,1,1,2-Tetrachloroethane	0.026	-	-	-	100
1,1,2,2-Tetrachloroethane	0.2	-	-	-	40
Tetrachloroethylene	0.0021	0	5	5	-
1,1-Dichloroethylene	-	7	7	7	6
cis-1,2-Dichloroethylene	-	70	70	70	-
trans-1,2-Dichloroethylene	-	100	100	100	-
Carbon tetrachloride	0.7	0	5	5	50
Chloroform	-	70	80	-	-
Dichloromethane	0.002	0	5	5	500

a. Source: U.S. EPA IRIS database
b. Maximum contaminant level goal, set at zero (0) as a default for known or suspected carcinogens.
c. Source: U.S. EPA 2011c
d. Source: SDWB 2009
e. A Health Advisory is an unenforceable guidance value provided as information to regulators.

Adequacy of the Current MCL for TCP

The current MCL for TCP in Hawaii is 0.6 µg/L; this MCL is based in part on a review of TCP by Tardiff (2001). The Tardiff review was based on an ED_{10} value of 1.7 mg/kg-day and a 1 in 1,000,000 risk of cancer. The ED_{10} value was based on the incidence of pancreatic tumors in male rats in the NTP (1993) chronic bioassays, and the value was not adjusted to account for dose extrapolation from rats to humans. At the time of the Tardiff (2001) report, the use of age-dependent adjustment factors was not in common practice, so the cancer risk estimate was based on exposure during adulthood.

Applying the exposure models and age-dependent adjustment factors, the MCL of 0.6 µg/L TCP in water produces an estimated cancer risk of 2.8×10^{-6} to 1.4×10^{-3} for the range of cancer slope factors proposed in existing reviews (see Table 10).

Between 2000 and 2011, the concentration of TCP in tap water exceeded the current MCL of 0.6 µg/L in 16 samples collected on Oahu. These include 5 of 189 samples at Mililani and 11 of 65 samples at Kipapa Acres. The most recent samples to exceed the current MCL at these two sites were collected in 2008 at Mililani and in 2005 at Kipapa Acres. Thus, several years have passed since the MCL for TCP was exceeded. TCP concentration in tap water did not exceed the current MCL on Maui or Kauai during the 11-year period examined (see Section 8 and Appendix C for details).

7. Other Drinking Water Guidance and Regulations Regarding TCP

The State of Hawaii established a maximum contaminant level (MCL) of 0.0006 mg/L (0.6 µg/L) for TCP in tap water (HDOH 2009b). A search for MCLs and other regulatory limits or guidelines for TCP in other states was performed. A search was also conducted for guidance and regulations on TCP in federal agencies, national governments, and international organizations.

States

Searches of drinking water quality laws, guidelines, and regulations from each of the 50 states were conducted to determine whether they regulate TCP in drinking water and/or tap water. The search was conducted by following the link for each individual state provided by the U.S. EPA on its Local Drinking Water database[m]. This database served as a useful guide for identifying the state-level agencies that regulated water quality, but there is no consistency between states' administrative structures. When the information was not found at the links provided by the U.S. EPA's Local Drinking Water database, additional searches were performed within each state's websites to identify the water contaminant regulatory information.

A summary of the search of all 50 states is presented in Appendix B. Hawaii is the only state that has issued a MCL for TCP in drinking water. Some other states have different kinds of limits or recommendations for TCP in water.

- Alaska has a Groundwater Cleanup level of 0.6 µg/L, based on an oral cancer slope factor of 7 $(mg/kg\text{-}day)^{-1}$ (Alaska Department of Environmental

[m] Source: http://water.epa.gov/drink/local/index.cfm (accessed December 2011 and January 2012)

Conservation 2008). The source of the cancer slope factor value was not identified.

- The State of California has a Public Health Goal (PHG) of 0.0007 µg/L (0.7 ng/L) and a notification level[n] of 0.005 µg/L (5 ng/L) for TCP (CA-OEHHA 2009). The public health goal was calculated based on a cancer slope factor of 25 $(mg/kg\text{-}day)^{-1}$ for carcinomas of the forestomach in female mice in the NTP (1993) study.

  Because the EPA Analytical Method No. 504.1, with its lower detection limit of 0.02 µg/L, is not deemed sensitive enough to protect public health, the state developed two gas chromatography-mass spectrometry (GC/MS) methods: Purge and Trap GC/MS and Liquid-Liquid Extraction GC/MS (California Department of Health Services 2002a,b) that are capable of quantification of TCP in water at 0.005 µg/L (CA-DPH 2007). The notification level was set at the detection limit of 0.005 µg/L (5 ng/L), thus any detectable TCP in California drinking water requires notification.

  Response levels are concentrations in drinking water at which California DPH recommends removal of a source from service. The response level for TCP is 100 times the notification level, or 0.5 µg/L (CA-DPH 2010).

- Florida has a lifetime Health Advisory Level (HAL)[o] of 40 µg/L (FDOH 2011). There was no information on what health effects this HAL was based.

- Minnesota published a cancer health based value of 0.003 µg/L and a chronic noncancer health based value of 20 µg/L (MDH 2010). These figures were derived from the IRIS (2009) cancer slope factor of 30 $(mg/kg\text{-}day)^{-1}$.

- In 1999, the New Jersey Department of Environmental Protection Division of Science, Research and Technology (DSRT) developed a health-based drinking water guidance value of 0.005 µg/L for TCP based upon a 10^{-6} risk level and a slope factor of 7 $(mg/kg/day)^{-1}$ (NJDEP 2008).

  The New Jersey Drinking Water Quality Institute recommended a Practical Quantitation Limit of 0.03 µg/L for TCP (NJDWQI 2009). The NJDWQI report

[n] A notification level is a health-based advisory levels established by CA-DPH for chemicals in drinking water that lack maximum contaminant levels (MCLs). When chemicals are found at concentrations greater than their notification levels, certain requirements and recommendations apply.

[o] The HAL concentration of a chemical in drinking water is a value that, based on the available data, is virtually certain not to cause adverse human health effects if consumed over a lifetime.

stated that a health-based MCL for TCP "is in the process of development and is anticipated to be established somewhere in the range of 0.0015µg/L to 0.009 µg/L," and that TCP was anticipated to be included in the New Jersey Safe Drinking Water Regulations update expected in 2009 (NJDWQI 2009). However a thorough search in 2012 of New Jersey's state government websites and all water-related state laws and regulations identified no existing MCL for TCP, so it appears the anticipated MCL has not yet been adopted by the New Jersey state regulators.

- The states of Alabama, Arizona, Missouri, Nebraska, New York, and Washington included TCP in lists of organic compounds that are required to be measured in drinking water but for which there are no regulatory limits.
- The remainder of state-level governments showed no intention to regulate TCP in drinking water beyond federal-level requirements.

U.S. EPA

There is no current federal MCL or other regulatory limit of TCP in water. However, there have been recent developments which may lead to efforts to regulate TCP in water at the federal level.

The Safe Drinking Water Act includes a process that the U.S. EPA must follow to identify and list unregulated contaminants which may require a national drinking water regulation in the future. EPA must periodically publish this list of contaminants (called the Contaminant Candidate List or CCL) and decide whether to regulate at least five or more contaminants on the list. The U.S. EPA uses this list of unregulated contaminants to prioritize research and data collection efforts to help it determine whether it should regulate a specific contaminant. In 2009, TCP was included in the U.S. EPA's Third Candidate Contaminant List (CCL 3). The CCL is a list of contaminants that are currently not subject to any proposed or promulgated national primary drinking water regulations but are known or anticipated to occur in public water systems and may require regulation under the Safe Drinking Water Act (U.S. EPA 2009). Inclusion in the CCL 3 is not a guarantee, however, that an MCL on TCP will be promulgated[p].

In March 2010, the U.S. EPA announced plans for a new Drinking Water Strategy for assessing the health effects of certain drinking water contaminants that pose health

[p] One can visit the U.S. EPA's CCL and Regulatory Determinations Home page at http://water.epa.gov/scitech/drinkingwater/dws/ccl/index.cfm for more information and updates in the review of CCL 3 chemicals.

concerns (U.S. EPA 2010). TCP is among the first 16 volatile organic compounds (VOCs) that are scheduled to be evaluated under the new Drinking Water Strategy. The strategy will address contaminants as groups rather than one at a time so that enhancement of drinking water protection can be achieved cost-effectively. The first group of compounds to be evaluated in the new strategy includes eight currently regulated compounds (benzene; carbon tetrachloride; 1,2-dichloroethane; 1,2-dichloropropane; dichloromethane; tetrachloroethylene; trichloroethylene; vinyl chloride) and eight unregulated compounds (aniline; benzyl chloride; 1,3-butadiene; 1,1-dichloroethane; nitrobenzene; oxirane methyl; TCP; and urethane) (U.S. EPA 2011b). All of the compounds selected are known or suspected by the U.S. EPA to cause cancer. The evaluation of the group of VOCs was expected to begin by the end of March 2011. The Agency noted that typically, it takes about 2 to 2.5 years to develop a proposed rule and about 2 years to promulgate a final rule (U.S. EPA 2011b).

International

TCP is not included in the World Health Organization (WHO)'s current Drinking Water Guidelines, Fourth Edition (WHO 2011). The WHO (2003) reviewed TCP in a Concise International Chemical Assessment Document, but did not set an acceptable daily intake level for TCP.

TCP is not included in the current lists of water contaminants regulated in the European Union (1998), United Kingdom (UK 2010a,b), Canada (Health Canada 2010), or Australia (Australian Government 2011).

In Osaka, Japan, TCP was found in 18 samples of surface water from urban rivers and their estuaries, at concentrations ranging from the lower detection limit (0.18 µg/L) to 100 µg/L (NTP 2011), but a search of Japan's water quality regulations identified no mention of TCP in water.

8. TCP Contamination in Water in Hawaii

The Hawaii Department of Health provided Tetra Tech with an Excel spreadsheet containing the analytical results of TCP measured in water samples collected at water treatment facilities on the islands of Oahu, Maui, and Kauai from 2000 to 2011 (HDOH 2011a). The spreadsheet included TCP concentration data from samples collected at water source wells (WL) and from treated tap water (TP).

The WL and TP sample data were organized per water treatment plant identified by region or community designation for comparison. The number of samples from the different treatment facilities varied. The lower limit of detection was 0.04 µg/L for most

samples analyzed. In a small number of analyses, the lower limit of detection was 0.01 µg/L or 0.5 µg/L.

Geometric means of the TCP concentrations in tap water samples were calculated per treatment plant, per island, and for all available data for the state of Hawaii. Samples that had no detectable TCP level cannot be assumed to actually be free of the contaminant. Rather than using a value of zero, one-half of the value of the lower limit of detection was used to represent non-detect samples for purposes of calculation of the geometric means. The geometric mean TCP concentrations in TP samples were 0.0338 µg/L on Maui, 0.0432 µg/L on Oahu, 0.0371 µg/L on Kauai, and 0.0367 for the three islands combined (see Table 13, page 38).

Table 14 (page 39) summarizes the TCP concentrations in pre-treated WL water samples taken from source wells at the water treatment facilities. Across the State of Hawaii, detectable TCP was reported in 61% of source well water samples and in 30% of treated tap water samples. Maui had the highest average ground water concentrations of TCP (0.2 µg/L), followed by Oahu (0.1 µg/L) and Kauai (0.04 µg/L).

Appendix C presents summaries of the concentrations in WL and TP samples collected at each water treatment facility.

Analytical Methods and Detection Limit

The lower limit of detection for most water samples analyzed for TCP content in Hawaii between 2000 and 2011 was reported to be 0.04 µg/L (HDOH 2011a). According to the *Directory of Drinking Water Laboratories Certified or Approved by the Hawaii Department of Health* (HDOH 2011b), the laboratories that test the drinking water quality in Hawaii currently use EPA Analytical Method No. 504.1, which is titled *1,2-Dibromoethane (EDB), 1,2-Dibromo-3-chloro-propane (DBCP), and 1,2,3-Trichloropropane (123TCP) in Water by Microextraction and Gas Chromatography*, and the reported detection limit is 0.02 µg/L (U.S. EPA 1995).

Efficacy of Water Treatment

At most of the water treatment facilities, the geometric mean of TCP concentrations was higher in pre-treated well water than in treated tap water. However, the simple comparison between well water and tap water show an increase in TCP concentration after treatment at three sites (HNL-Windward-Pearl Harbor and Wahiawa on Oahu, and Lihue-Kapaa on Kauai; see tables 13 and 14). Comparisons between pre- and post-treated water samples in this manner is not representative of actual treatment efficacy, because the date ranges of the sample collections varied. A more logical method of estimating the efficacy of TCP removal from ground water is a comparison of the TCP concentrations in WL and TP samples that were collected on the same dates.

Table 13. Summary of TCP Analysis Results of Tap Water (TP) Samples Collected at Hawaiian Water Treatment Facilities from 2000 to 2011.

Island	Region or Community	System Owner[a]	No. of Samples Analysed	No. (%) of Samples with Detectable TCP	Range of Detection (µg/L)	Geometric Mean Value[b] (µg/L)	Range of Years of Sample Collections
Maui	Kaanapali	Private	48	14 (29)	N.D. - 0.36	0.0324	2000-2011
	Makawao	MDWS	28	16 (57)	N.D. - 0.174	0.0588	2002-2010
	Lahaina	MDWS	15	5 (33)	N.D. - 0.06	0.0275	2002-2010
	Honokohau	MDWS	21	1 (4.7)	N.D. - 0.05	0.0209	2000 -2004
	Maunaolu	Private	32	4 (13)	N.D - 0.25	0.0248	2004-2011
	Total for Maui	-	*144*	*40 (28)*	*N.D. - 0.36*	*0.0338*	*2000-2011*
Oahu	Kunia Village	Private	62	2 (3.2)	N.D. - 0.13	0.0209	2000-2011
	Hawaii Country Club	Private	56	8 (14)	N.D. - 0.12	0.0242	2000-2011
	HNL-Windward Pearl Harbor	HBWS	4	2 (50)	N.D. - 0.16	0.0990	2010-2011
	Kipapa Acres	Private	65	20 (31)	N.D. - 0.72[c]	0.0509	2000-2011
	Waialua-Haleiwa	HBWS	3	1 (33)	N.D. - 0.11	0.0353	2009-2010
	Wahiawa	HBWS	9	5 (56)	N.D. - 0.09	0.0834	2009-2011
	Waipio Heights	HBWS	49	18 (37)	N.D. - 0.4	0.0469	2004-2010
	Waipahu-Ewa-Waianae	HBWS	373	113 (30)	N.D. - 0.58	0.0390	2000-2011
	Mililani	HBWS	189	54 (29)	N.D. - 0.81[d]	0.0379	2000-2011
	Total for Oahu	-	*810*	*223 (28)*	*N.D. - 0.81*	*0.0432*	*2000-2011*
Kauai	Lihue-Kapaa	KDW	35	31 (89)	N.D. - 0.09	0.0575	2001-2010
	Kalaheo-Koloa	KDW	9	2 (22)	N.D. - 0.05	0.0239	2002-2009
	Total for Kauai	-	*44*	*33 (75)*	*N.D. - 0.09*	*0.0371*	*2001-2010*
State of Hawaii		**-**	***998***	***296 (30)***	***N.D. - 0.81***	***0.0367***	***2000-2011***

N.D. = Not Detected, in most cases the lower limit of detection was 0.04 µg/L; in a small number of analyses, the lower limit of detection was 0.01 µg/L or 0.5 µg/L.
a. MDWS = Maui Department of Water Supply; HBWS = Honolulu Board of Water Supply; KDW = Kauai Department of Water.
b. One-half of the value of the lower limit of detection was used to represent non-detect samples for purposes of calculation of the geometric means.
c. Eleven samples collected at Kipapa Acres exceeded the current MCL of 0.6 µg/L (range = 0.63 to 0.72 µg/L).
d. Five samples collected at Mililani exceeded the current MCL of 0.6 µg/L (range = 0.62 to 0.81 µg/L).

Table 14. Summary of TCP Analysis Results of Non-treated Well Water (WL) Samples Collected at Hawaiian Water Treatment Facilities from 2000 to 2011.

Island	Region or Community	System Owner[a]	No. of Samples Analysed	No. (%) of Samples with Detectable TCP	Range of Detection (µg/L)	Geometric Mean Value[b] (µg/L)	Range of Years of Sample Collections
Maui	Kaanapali	Private	94	80 (85)	N.D. - 1.6	0.415	2000-2008
	Makawao	MDWS	0	0	N.D	N/A	N/A
	Lahaina	MDWS	43	30 (70)	N.D. - 0.09	0.0414	2000-2009
	Honokohau	MDWS	0	0	N.D.	N/A	N/A
	Maunaolu	Private	19	10 (53)	N.D. - 1.32	0.146	2004-2008
	Total for Maui	-	*156*	*120 (77)*	*N.D. - 1.32*	*0.2*	*2000-2009*
Oahu	Kunia Village	Private	102	53 (52)	N.D. - 0.33	0.0571	2000-2008
	Hawaii Country Club	Private	34	25 (74)	N.D. - 0.46	0.138	2000-2008
	HNL-Windward Pearl Harbor	HBWS	27	8 (30)	N.D. - 0.05	0.0254	2000-2009
	Kipapa Acres	Private	0	0	N.D.	N/A	N/A
	Waialua-Haleiwa	HBWS	251	117 (47)	N.D. - 0.82	0.0845	2000-2009
	Wahiawa	HBWS	57	40 (70)	N.D. - 0.23	0.0750	2000-2009
	Waipio Heights	HBWS	230	176 (77)	N.D. - 1.02	0.151	2000-2009
	Waipahu-Ewa-Waianae	HBWS	1021	662 (65)	N.D. - 1.52	0.145	2000-2010
	Mililani	HBWS	203	66 (33)	N.D. - 4.02	0.502	2000-2008
	Total for Oahu	-	*1925*	*1157 (60)*	*N.D. - 4.02*	*0.104*	*2000-2010*
Kauai	Lihue-Kapaa	KDW	27	21 (78)	N.D. - 0.08	0.0449	2002-2011
	Kalaheo-Koloa	KDW	8	2 (25)	N.D. - 0.04	0.0238	2002-2009
	Total for Kauai	-	*35*	*23 (66)*	*N.D. - 0.08*	*0.0401*	*2002-2011*
State of Hawaii		**-**	***2116***	***1300 (61)***	***N.D. - 4.02***	***0.199***	***2000-2011***

N.D. = Not Detected, for all well samples reported, the lower limit of detection was 0.04 µg/L.
a. MDWS = Maui Department of Water Supply; HBWS = Honolulu Board of Water Supply; KDW = Kauai Department of Water.
b. One-half of the value of the lower limit of detection was used to represent non-detect samples for purposes of calculation of the geometric means.

Table 15 (below) summarizes the percent change in TCP concentrations between pre-treatment groundwater/well (WL) samples and post-treatment (TP) samples which were collected on the same dates. This comparison was not possible for six water treatment facilities due to data insufficiencies. Specifically, there were no WL data reported for the facilities at Makawao, Honokohau, and Kipapa Acres, and there were no dates on which both WL and TP samples were collected for the facilities at HNL-Windward-Pearl Harbor, Waialua-Haleiwa, and Wahiawa. For 10 water treatment facilities, however, there were WL and TP samples collected on concordant dates. For calculation of the average TCP concentrations, the samples with no detected TCP were given a value of 0.02 µg/L (half of the lower detection limit), which likely overestimates the actual TCP concentration in those samples.

Table 15. Percent Change in TCP Concentrations by Water Treatment

Island	Region or Community	System Owner[a]	No. of Dates with Corresponding WL & TP Data	Average WL (µg/L TCP)	Average TP (µg/L TCP)	Change in TCP Concentration During Water Treatment[c]
Maui	Kaanapali	Private	32	0.90	0.053	Reduced 94%
	Lahaina	MDWS	11	0.061	0.029	Reduced 57%
	Maunaolu	Private	17	0.53	0.034	Reduced 94 %
Oahu	Kunia Village	Private	39	0.15	0.02	Reduced 88%
	Hawaii Country Club	Private	30	0.24	0.03	Reduced 88%
	Waipio Heights	HBWS	20	0.51	0.056	Reduced 90%
	Waipahu-Ewa-Waianae	HBWS	98	0.63	0.11	Reduced 82%
	Mililani	HBWS	54	2.4	0.099	Reduced 96%
Kauai	Lihue-Kapaa	KDW	11	0.056	0.057	Increased 1.6%
	Kalaheo-Koloa	KDW	8	0.025	0.026	Increased 5%

N.D. = Not Detected, for all well samples reported, the lower limit of detection was 0.04 µg/L.
a. MDWS = Maui Department of Water Supply; HBWS = Honolulu Board of Water Supply; KDW = Kauai Department of Water.
b. One-half of the value of the lower limit of detection was used to represent non-detect samples for purposes of calculation of means.
c. Change (%) = (Average WL – Average TP) ÷ Average WL.

On Maui, the island with the highest average groundwater TCP concentrations, the TCP concentration in water was reduced by 57% to 94%. The two private water treatment facilities on Oahu had greater percent reduction of TCP concentrations than the MDWS facility; however, these private facilities had much higher TCP in the source groundwater. The TP samples for all three facilities had TCP levels close to or below the lower detection limit.

On Oahu, treatment of the water reduced TCP concentration by 82% to 96%.

The two water treatment facilities on Kauai, show a slight increase in average TCP concentrations after treatment in Table 15. However, this comparison is flawed by three factors: 1) the TCP concentrations in the WL samples were very low, close to the lower detection limit of 0.04 µg/L; 2) samples with TCP levels below the limit of detection were given an arbitrary value of 0.02 µg/L which likely overestimates the actual TCP concentrations; and 3) a very small data set with corresponding WL and TP data. Therefore, the slight increase in calculated TCP concentrations at the two Kauai facilities should not be considered of concern. Kauai had the lowest TCP levels in groundwater of the three islands, and TCP levels in the tap water effluent were far below the MCL and close to or below the lower detection limit.

Granular activated carbon (GAC) is the method of filtration used by water treatment facilities to remove TCP from water. Based on the limited data in Table 15, this method appears to be effective in reducing TCP concentrations to levels below the current MCL.

9. Conclusions

A review of the potential human health effects of TCP has been conducted to assist the State of Hawaii in setting a regulatory limit for TCP in household tap water. The available chronic/carcinogenicity studies of TCP provide a poor prediction of the actual health risks to humans exposed to TCP in water. Dosimetric differences in the route of administration and vehicle and the high dose levels used in the 2-year studies produced adverse health effects (e.g., forestomach tumors) in rats and mice that are not likely to be applicable to humans. However, these studies provide the only data on a lifetime exposure to TCP in any species on which to calculate a cancer slope factor.

Previous reviewers have proposed cancer slope factors as low as 0.0588 $(mg/kg\text{-}day)^{-1}$ assuming that pancreatic tumors in male rats are considered the most relevant single endpoint to humans. The U.S. EPA's IRIS program recommended a cancer slope factor of 30 $(mg/kg\text{-}day)^{-1}$, based on overall cancer risk in the most sensitive animals tested, female mice. The IRIS approach appears to overpredict the actual cancer risk in humans from TCP. In the current review, the geometric mean of the cancer slope factors for all tumors in both sexes of rats and mice was estimated to be 5.8 $(mg/kg\text{-}day)^{-1}$. Exposure models and age-dependent adjustment factors were applied to the existing cancer potency values to estimate the human cancer risk for a range of water concentrations of TCP.

Regulations of contaminants in water are generally established with cancer risk estimates in the range of 10^{-6} to 10^{-4}. For the two most conservative cancer slope factors [30 $(mg/kg\text{-}day)^{-1}$ and 5.8 $(mg/kg\text{-}day)^{-1}$], the TCP concentrations associated with cancer risk of 10^{-6} and 10^{-5} are below the lower limit of detection (0.04 µg/L). The 10^{-4} cancer risk level is associated with a range of 0.042 to 22 µg/L TCP in water.

The current Hawaii MCL of 0.6 µg/L TCP is associated with a cancer risk levels of 2.8×10^{-6} to 1.4×10^{-3}, depending on the cancer slope factor. Over the previous 11 years, the concentration of TCP in tap water has rarely exceeded this MCL, and the last occasion was in 2008.

References

Abramson, JH. 2011. WinPepi (Version 11.15) [Software]. Available at http://www.brixtonhealth.com/pepi4windows.html (accessed 19 April 2012).

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for 1,2,3-Trichloropropane. Available at http://www.atsdr.cdc.gov/ToxProfiles/tp57.pdf (accessed 24 Oct 2011).

ATSDR. 2011. Addendum to the Toxicological Profile for 1,2,3-Trichloropropane. Available at http://www.atsdr.cdc.gov/toxprofiles/123-Trichloropropane_addendum.pdf (accessed 24 Oct 2011).

Alaska Department of Environmental Conservation. 2008. Cleanup Level Guidance. Division of Spill Prevention and Response, Contaminated Sites Program. Available at http://dec.alaska.gov/spar/csp/guidance/cleanuplevels.pdf (accessed 15 Dec 2011).

Australian Government. 2011. National Water Quality Management Guidelines, Australian Drinking Water Guidelines 6. Available at http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/adwg_11_06.pdf (accessed 16 Dec 2011).

Boysen G, Pachkowski BF, Nakamura J, Swenberg JA. 2009. The formation and biological significance of N7-guanine adducts. *Mutat. Res*. 678, 76–94.

California Department of Health Services. 2002a. Determination of 1,2,3-Trichloropropane in Drinking Water by Purge and Trap Gas Chromatography/Mass Spectrometry. California Department of Health Services, Division of Drinking Water and Environmental Management, Sanitation and Radiation Laboratories Branch. Available at http://www.cdph.ca.gov/certlic/drinkingwater/Documents/Drinkingwaterlabs/TCPbyPT-GCMS.pdf (accessed 21 March 2012).

California Department of Health Services. 2002b. Determination of 1,2,3-Trichloropropane in Drinking Water by Continuous Liquid-Liquid Extraction and Gas Chromatography/Mass Spectrometry. California Department of Health Services, Division of Drinking Water and Environmental Management, Sanitation and Radiation Laboratories Branch. Available at http://www.cdph.ca.gov/certlic/drinkingwater/Documents/Drinkingwaterlabs/TCPbyLLE-GCMS.pdf (accessed 21 March 2012).

California Department of Public Health (CA-DPH). 2007. Analysis for 1,2,3-Trichloropropane. Available at http://www.cdph.ca.gov/certlic/drinkingwater/Pages/123TCPanalysis.aspx (accessed 21 March 2012).

CA-DPH. 2010. Drinking Water Notification Levels and Response Levels: An Overview. California Department of Public Health, Drinking Water Program. Available at http://www.cdph.ca.gov/certlic/drinkingwater/Documents/Notificationlevels/notificationlevels.pdf (accessed 21 March 2012).

California Office of Environmental Health Hazard Assessment (CA-OEHHA). 2009. Public health Goals for Chemicals in Drinking Water. 1,2,3-Trichloropropane. August 2009. Pesticide and Environmental Toxicology Branch, Office of Environmental Health Hazard Assessment , California Environmental Protection Agency. http://www.oehha.ca.gov/water/phg/pdf/082009TCP_phg.pdf (accessed 16 Dec 2011).

Centers for Disease Control and Prevention (CDC). 2010. Skin permeability calculator. Available at http://www.cdc.gov/niosh/topics/skin/skinpermcalc.html (accessed 8 Feb 2012).

Chandra SA, Nolan MW, Malarkey DE. 2010. Chemical Carcinogenesis of the Gastrointestinal Tract in Rodents: An Overview with Emphasis on NTP Carcinogenesis Bioassays. *Toxicologic Pathology*. 38: 188-197.

Environ. 1985. Analysis of Potential Human Health Risk and Approaches to Settling Action Levels for pesticides in Drinking Water in Hawaii. December 1985 (unpublished report provided to Tetra Tech, Inc., by HDOH).

European Chemicals Agency (ECHA). 2011. Member State Committee Support Document for Identification of 1,2,3-Trichloropropane as a Substance of Very high Concern Because of its CMR properties. Available at http://echa.europa.eu/documents/10162/13638/supdoc_trichloropropane_20110520_en.pdf (accessed 16 Dec 2011).

European Inventory of Existing Commercial Chemical Substances (EINECS). 2012. Result for CAS#: 96-18-4. Available at http://esis.jrc.ec.europa.eu/ (accessed 02 April 2012).

European Union (EU). 1998. Council Directive 98/83/EC of 3 November 1998 on the quality of water intended for human consumption. Available at http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:1998:330:0032:0054:EN:PDF (accessed 9 March 2012).

EU. 2011. European Union's Pesticide Database. http://ec.europa.eu/sanco_pesticides/public/index.cfm (accessed February 2012).

Florida Department of Health (FDOH). 2011. Florida Department of Health Environmental Chemistry Analyte List. Available at http://www.doh.state.fl.us/environment/community/health-advisory/HAL_list.pdf (accessed 23 Dec 2011).

Gelhaus MW, Gift JS, Hogan KA, Kopylev L, Schlosser PM, Kadry AR. 2011. Approaches to cancer assessment in EPA's Integrated Risk Information System. *Toxicology and Applied Pharmacology*. 254:170–180.

Ghanayem BI, Maronpot RR, Matthews HB. 1986. Association of chemically induced forestomach cell proliferation and carcinogenesis. *Cancer Lett*. 32(3):271-278. [cited in IRIS 2009]

Gushow TS, Quast JF. 1984. 1,2,3-Trichloropropane: Acute inhalation toxicity evaluation in male rats and mice. Report to Dow Chemical Co. OTS 0510175. [Unpublished study, cited in ATSDR 1992]

Han H. 2010. Acute 1,2,3-Trichloropane Poisoning: A Case Report and Literature Review. *Basic & Clinical Pharmacology & Toxicology*. 107: 988–990.

Hawaii Department of Health (HDOH). 2005. Hawaii Department of Health Administrative Rules. Rules Relating to Potable Water Systems. Page 20-14. Available on-line at http://oeqc.doh.hawaii.gov/sites/har/AdmRules1/11-20.pdf

HDOH. 2009a. Technical Guidance Manual for the Implementation of the Hawai'i State Contingency Plan. Section 9. Supplemental Guidance for Select Contaminants of Concern. June 21, 2009. Available at http://www.hawaiidoh.org/tgm.aspx (accessed 6 Feb 2012).

HDOH. 2009b. Contaminants Regulated by the Safe Drinking Water Branch (effective 3/4/09). http://hawaii.gov/health/environmental/environmental/water/sdwb/sdwb/pdf/MCL_03-04-09.pdf (accessed 21 Dec 2011)

HDOH. 2011a. Untitled Excel spreadsheet provided to tetra Tech, Inc., by HDOH containing analytical results of TCP measured in 3101 water samples collected at water treatment plants on the islands of Oahu, Maui, and Kauai from 2000 to 2011.

HDOH. 2011b. Directory of Drinking Water Laboratories Certified or Approved by the Hawaii Department of Health, State Laboratories Division. Available at http://hawaii.gov/health/environmental/water/sdwb/sdwb/pdf/LabsNov2011.pdf (accessed 21 March 2012).

Hazleton Laboratories America, Inc. 1977. *104-week dietary study in rats, 1,2-dibromo-3-chloropropane (DBCP). Final report*. Midland, MI, Dow Chemical Co., (unpublished report, project no. 174-122). [cited in WHO 1996].

Health Canada. 2010. Guidelines for Canadian Drinking Water Quality. Available at http://hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/2010-sum_guide-res_recom/sum_guide-res_recom-eng.pdf (accessed 16 Dec 2011).

International Agency for Research on Cancer (IARC). 1995. 1,2,3-Trichloropropane. Volume 63. Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Available at http://monographs.iarc.fr/ENG/Monographs/vol63/volume63.pdf (accessed 16 Dec 2011).

IARC. 1999. Monograph of DBCP. Volume 71. Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide Available at http://monographs.iarc.fr/ENG/Monographs/vol71/mono71-20.pdf (accessed 27 March 2012).

IARC. 2003. Predictive value of rodent forestomach and gastric neuroendocrine tumours in evaluating carcinogenic risks to humans. Technical Publication No. 39. Available at http://monographs.iarc.fr/ENG/Publications/techrep39/IARCrep39.pdf (accessed 3 Feb 2012).

Integrated Risk Information System (IRIS). 1994. IRIS Summary of Dichlorvos (CASRN 62-73-7). Available at http://www.epa.gov/iris/subst/0151.htm (accessed 06 April 2012).

IRIS. 2009. *Toxicological Review of 1,2,3-Trichloropropane (CAS No. 96-18-4)*. EPA/635/R-08/010F. U.S. Environmental Protection Agency, Washington DC. Available at http://www.epa.gov/iris/toxreviews/0200tr.pdf (accessed 24 Oct 2011).

Irwin RD, Haseman JK, Eustis SL. 1995. 1,2,3-Trichloropropane: a multisite carcinogen in rats and mice. *Fundam. Appl. Toxicol*. 25, 241–252.

Jo WK, Weisel CP, Lioy PJ. 1990a. Routes of chloroform exposure and body burdon from showering with chlorinated tap water. *Risk Analysis*. 10(4): 575-580.

Jo WK, Weisel CP, Lioy PJ. 1990b. Chloroform exposure and the health risk associated with multiple uses of chlorinated tap water. *Risk Analysis*. 10(4):581-585.

Johannsen FR, Levinskas GJ, Rusch GM, et al. 1988. Evaluation of the subchronic and reproductive effects of a series of chlorinated propanes in the rat. I. Toxicity of the 1,2,3-trichloropropane. *J Toxicol Environ Health.* 25(3):299-315. [cited in ATSDR 1992]

La, DK; Schoonhoven, R; Ito, N; et al. 1996. The effects of exposure route on DNA adduct formation and cellular proliferation by 1,2,3-trichloropropane. *Toxicol Appl Pharmacol.* 140:108–-114.

Mahmood, NA; Overstreet, D; Burka, LT. 1991 Comparative disposition and metabolism of 1,2,3-trichloropropane in rats and mice. *Drug Metab Dispos.* 19:411–418. [cited in IRIS 2009 and ATSDR 1992]

McOmie WA, Barnes TR. 1949. Acute and subacute toxicity of 1,2,3-trichloropropane in mice and rabbits. *Fed Proc Pharm Exp Therap*. 8:319. [cited in IRIS 2009 and ATSDR 1992]

Merrick, BA; Robinson, M; Condie, LW. 1991. Cardiopathic effect of 1,2,3-trichloropropane after subacute and subchronic exposure in rats. *J Appl Toxicol*. 11:179–187. [cited in IRIS 2009]

Minnesota Department of Health (MDH). 2010. Health-based guidance for water: 1,2,3-Trichloropropane. http://www.health.state.mn.us/divs/eh/risk/guidance/gw/123triclorp.pdf (accessed 23 December 2011).

National Cancer Institute (NCI) 1978. Bioassay of Dibromochloropropane for Possible Carcinogenicity, CAS No. 96-18-4. NCI-CG-TR-28. Available at http://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr028.pdf (accessed 14 March 2012).

NCI. 2012. Surveillance Epidemiology and End Results (SEER) database. Available at http://seer.cancer.gov/statfacts/html/all.html (accessed 19 April 2012).

National Toxicology Program (NTP). 1993. *Toxicology and Carcinogenesis Studies of 1,2,3-Trichloropropane (CAS No. 96-18-4) in F344/N Rats and B6C3F$_1$ Mice (Gavage Studies)*. Technical Report Series No. 384. U.S. Department of Health and Human Services. Available at http://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr384.pdf (accessed 15 Nov 2011).

NTP. 2011. Report on Carcinogens, 12 Edition. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program Available at http://ntp.niehs.nih.gov/ntp/roc/twelfth/roc12.pdf (accessed 11 June 2011).

New Jersey Department of Environmental Protection (NJDEP). 2008. Letter from NJDEP to U.S. EPA regarding Docket ID No. EPA-HQ-OW-2007-1189, Drinking Water Contaminant Candidate List 3-Draft. Available at http://www.state.nj.us/dep/dsr/dw/CCL3%20letter%20final.pdf (accessed 15 Dec 2011).

New Jersey Drinking Water Quality Institute (NJDWQI). 2009. PQL Review, Assessment and Recommendations. March 3, 2009. Available at http://www.state.nj.us/dep/watersupply/last_final_iplq_testing.tt.pdf (accessed 15 Dec 2011).

Proctor, DM; Gatto, NM; Hong, SJ; et al. 2007. Mode-of-action framework for evaluating the relevance of rodent forestomach tumors in cancer risk assessment. *Toxicological Sciences*. 98(2):313–326.

Schaum J, Hoang K, Kinerson R, Moya J, Wang RGM. 1994. Chapter 13. Estimating dermal and inhalation exposure to volatile chemicals in domestic water. In: *Water Contamination and Health: Integration of Exposure Assessment, Toxicology, and Risk Assessment*. Wang RGM (ed). Marcel Decker, Inc. New York. pp 305 – 321.

Silverman L, Schulte HF, First MW. 1946. Further Studies on Sensory Response to Certain Industrial Solvent Vapors. *J Ind Hyg Toxicol*. 28:262–266. [cited in IRIS 2009].

State Drinking Water Branch (SDWB). 2009. Contaminants Regulated by the State Drinking Water Branch (effective 3/4/09). Hawaii Department of Health. Available at http://hawaii.gov/health/environmental/water/sdwb/sdwb/pdf/MCL_03-04-09.pdf

Tardiff RG. 1992. *Reevaluation of the Potential Human health Risk and Approaches to setting Action levels for 1,2,3-Trichloropropane (TCP) in Drinking Water in Hawaii*. November 1992. (unpublished report provided to Tetra Tech, Inc., by HDOH)

Tardiff R. 2001. *Reevaluation of the Potential Human health Risk and Approaches to setting Action levels for 1,2,3-Trichloropropane (TCP) in Drinking Water in Hawaii*. 26 November 2001. (unpublished report provided to Tetra Tech, Inc., by HDOH).

Tardiff RG, Carson ML. 2010. Derivation of a reference dose and drinking water equivalent level for 1,2,3-trichloropropane. *Food and Chemical Toxicology*. 48: 1488–1510.

United Kingdom (UK). 2010a. *The Water Supply (Water Quality) Regulations 2010*. Welsh Statutory Instruments. Water, England and Wales. 2010 No. 994 (W.99). Available at http://www.legislation.gov.uk/wsi/2010/994/pdfs/wsi_20100994_en.pdf (accessed 6 March 2012).

UK. 2010b. *The Water Quality (Scotland) Regulations 2010*. Scottish Statutory Instruments. 2010, No. 95. Available at http://www.legislation.gov.uk/ssi/2010/95/pdfs/ssi_20100095_en.pdf (accessed 6 March 2012).

U.S. Census. 2010. Hawaii. 2010 Census Interactive Population Search. Available at http://2010.census.gov/2010census/popmap/ipmtext.php?fl=15, accessed 18 April 2012.

U.S. EPA. 1992. Draft report: a cross-species scaling factor for carcinogen risk assessment based on equivalence of $mg/kg^{3/4}$/day. Federal Register 57(109):24152-24173.

U.S. EPA. 1995. Method 504.1. 1,2, Dibromoethane (EDB), 1,2-Dibromo-3-chloro-propane (DBCP), and 1,2,3-Trichloropropane (123TCP) in Water by Microextraction and Gas Chromatography. Revision 1.1. Available at https://www.nemi.gov/apex/f?p=237:45:4450649287321603::NO:45:: (accessed 21 March 2012)

U.S. EPA. 1998. IRIS Summary Page for Arsenic, Inorganic (CASRN 7440-38-2). Available at http://www.epa.gov/iris/subst/0278.htm (accessed 19 April 2012).

U.S. EPA. 2004. Risk Assessment Guidance for Superfund (RAGS) Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)

EPA/540/R/99/005, OSWER 9285.7-02EP, PB99-963312, July 2004. Available at http://www.epa.gov/oswer/riskassessment/ragse/pdf/part_e_final_revision_10-03-07.pdf (accessed 23 Feb 2012).

U.S. EPA. 2005a. *Guidelines for carcinogen risk assessment.* Risk Assessment Forum, Washington, DC; EPA/630/P-03/001B. Available at http://www.epa.gov/cancerguidelines (accessed 1 Dec 2011).

U.S. EPA. 2005b. *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens*. EPA/630/R-03/003. Available at http://epa.gov/raf/publications/pdfs/childrens_supplement_final.pdf (accessed 1 Dec 2011).

U.S. EPA. 2009. Drinking Water Contaminant Candidate List 3—Final. Available at http://www.epa.gov/fedrgstr/EPA-WATER/2009/October/Day-08/w24287.pdf (accessed 15 Feb 2012)

U.S. EPA. 2010. Drinking Water Strategy Contaminants as Group(s)-Notice of Public Stakeholder Meeting. Available at https://federalregister.gov/a/2010-22470 (accessed 15 Feb 2012).

U.S. EPA. 2011a. *Exposure Factors Handbook*: 2011 Edition. Available at http://www.epa.gov/ncea/efh/pdfs/efh-complete.pdf (accessed 7 Feb 2012).

U.S EPA 2011b. *Basic Questions and Answers for the Drinking Water Strategy Contaminant Groups Effort*. Available at http://water.epa.gov/lawsregs/rulesregs/sdwa/dwstrategy/upload/FactSheet_DrinkingWaterStrategy_VOCs.pdf (accessed 15 Feb 2012).

U.S. EPA. 2011c. 2011 Edition of the Drinking Water Standards and Health Advisories. EPA 820-R-11-002. Office of Water U.S. Environmental Protection Agency, Washington, DC. Available at http://water.epa.gov/action/advisories/drinking/upload/dwstandards2011.pdf (accessed 12 March 2012).

Villeneuve, DC; Chu, I; Secours, VE; et al. 1985. Results of a 90-day toxicity study on 1,2,3- and 1,1,2- trichloropropane administered via the drinking water. *Sci Total Environ* 47:421–426.

Weber, GL; Sipes, IG. 1992. In vitro metabolism and bioactivation of 1,2,3-trichloropropane. *Toxicol Appl.Pharmacol*. 113:152–158.

World Health Organization (WHO). 1996. 1,2-Dibromo-3-chloropropane in Drinking-water. Background document for development of WHO *Guidelines for Drinking-water Quality*. Available at http://www.who.int/water_sanitation_health/dwq/1,2-Dibromo-3-chloropropane.pdf (accessed 14 March 2012).

WHO. 2003. Concise International Chemical Assessment Document 56. 1,2,3-Trichloropropane. Available at http://www.who.int/ipcs/publications/cicad/en/cicad56.pdf. (accessed 5 March 2012)

WHO. 2011. Guidelines for Drinking-water Quality. 4th Edition. Available at http://whqlibdoc.who.int/publications/2011/9789241548151_eng.pdf (accessed 5 March 2012)

Appendix A. Exposure Models of TCP in Tap Water

The mathematical models used to estimate the exposure to TCP in tap water via the oral, inhalation, and transdermal routes in Section 5 are described in detail below. These models assume a concentration of 1 µg/L (0.001 mg/L) TCP for demonstration purposes. Assumed or measured values of other variables (e.g., shower duration, respiration rate, bathroom volume, etc.) are provided with sources cited.

Because age-dependent adjustment factors apply in the risk assessment, the exposure from each route of administration must be calculated separately for infants of less than 2 years of age, children of ages 2 to 16 years, and adults 16 years and older.

Ingestion of TCP in Tap Water

Ingestion of TCP is most likely to occur by drinking tap water. In its *Exposure Factors Handbook*, the U.S. EPA (2011a) provides data on daily ingestion volumes of tap water (see table below).

Exposure by ingestion of tap water is calculated by the formula:

$Dose_{ing}$ = (Cw x IR x AF)/BW, where the variables are defined as:

Variable	Value	Reference
$Dose_{ing}$ = Daily dose from ingestion of tap water (mg/kg-day)	-	-
C_w = concentration of contaminant in water (mg/L)	Input value	-
IR = Water ingestion rate (L/day)	adult 2.86 child 1.39 infant 0.924	US EPA (2011a)
AF = Absorption factor (no units)	1	Assumed 100% systemic absorption of ingested TCP.
BW = Body weight (kg)	adult 80 child 37 infant 10	US EPA (2011)

The exposure by ingestion of tap water containing 1 µg/L TCP would be calculated by:

(1 µg/L x 2.86 L/day x 1) ÷ 80 kg = 0.036 µg/kg-day = 0.000036 mg/kg-day for an adult

(1 µg/L x 1.39 L/day x 1) ÷ 37 kg = 0.038 µg/kg-day = 0.000038 mg/kg-day for a child

(1 µg/L x 0.924 L/day x 1) ÷ 10 kg = 0.092 µg/kg-day = 0.000092 mg/kg-day for an infant

Inhalation of TCP while Showering or Bathing

Inhalation of TCP from tap water could occur primarily in the bathroom, specifically while showering or bathing. TCP is volatile, so it is likely to be released from the water into the

breathable airspace, especially at the warmer temperatures used for showering and bathing. Because there are no available data on the level of exposure to TCP while showering or bathing, the potential exposure must be estimated by mathematical modeling. There is no official U.S. EPA guideline for modeling exposure to volatile organic compounds in a shower or bath. However, Schaum et al. (1994) published a mathematical model that is useful for estimating the potential inhalation exposure to volatile organic compounds for a person while taking a shower and includes the interval one remains in the bathroom after the shower. Schaum recently retired from U.S. EPA's National Center for Environmental Assessment.

Exposure by inhalation in terms of mg/kg-day is calculated by the formula:

$Dose_{inh} = (C_a \times MV \times (t_1 + t_2) \times EF \times 60 \text{ min/hr}) \div BW$, where the variables are defined as:

Variable	Value	Reference
$Dose_{inh}$ = Daily dose from inhalation at shower (mg/kg-day)	-	-
C_a = concentration of contaminant in air (mg/m^3)	see formula below	Schaum et al. 1994
MV = Minute volume; respiration rate (m^3/minute)	adult 0.0003 child 0.0005 infant 0.001	US EPA (2011a) Assume "Light IntensityActivity"
t_1 = Time in shower/bath (hours)	adult 0.22 child 0.30 infant 0.50	US EPA (201a)
t_2 = Time in bathroom after shower/bath (hours)	adult 0.13 child 0.12 infant 0.12	US EPA (2011a)
EF = Exposure frequency (showers or baths/day)	1	US EPA (2011a)
60 min/hr	Conversion of time units.	
BW = Body weight (kg)	adult 80 child 37 infant 10	US EPA (2011a)

C_a in the formula above is calculated by:

$C_a = ((C_{max} \div 2)t_1 + (C_{max} \times t_2)) \div (t_1 + t_2)$, where the variables are defined as:

Variable	Value	Reference
C_a = concentration of contaminant in air (mg/m^3)	-	-
C_{amax} = maximum concentration of contaminant in air (mg/m^3)	see formula below	Schaum et al. 1994
t_1 = Time in shower/bath (hours)	adult 0.22 child 0.30 infant 0.50	US EPA (2011a)
t_2 = Time in bathroom after shower/bath (hours)	adult 0.13 child 0.12 infant 0.12	US EPA (2011a)

C_{amax} in the formula above is calculated by:

$C_{amax} = (C_w \times f \times F_w \times t_1) \div V_a$, where the variables are defined as:

Variable	Value	Reference
C_{amax} = maximum concentration of contaminant in air (mg/m^3)	-	-
C_w = concentration of contaminant in water (mg/L)	Input value	-
f = fraction volatilized (no units)	0.1	Schaum et al. 1994
Fw = water flow rate (L/hr)	750	Schaum et al. 1994
t_1 = Time in shower/bath (hours)	adult 0.22 child 0.30 infant 0.50	US EPA (2011)
V_a = Volume of bathroom (m^3)	10	Schaum et al. 1994

For tap water that contains 1 µg/L TCP, the modeled inhalation exposure by an adult taking a shower can be estimated by the following series of calculations:

$C_{amax} = (0.001 \text{ mg/L} \times 0.1 \times 750 \text{ L/hr} \times 0.22 \text{ hr}) \div 10 \text{ m}^3 = 0.00165 \text{ mg/m}^3$

$C_a = ((0.00165 \text{ mg/m}^3 \div 2) \times 0.22 \text{ hr} + (0.00165 \text{ mg/m}^3 \times 0.13 \text{ hr})) \div (0.22 \text{ hr} + 0.13 \text{ hr}) = 0.00113 \text{ mg/m}^3$

$Dose_{inh} = (0.00113 \text{ mg/m}^3 \times 0.0003 \text{ m}^3\text{/min} \times (0.22 \text{ hr} + 0.13 \text{ hr}) \times 1\text{/day} \times 60 \text{ min/hr}) \div 80 \text{ kg} = 0.0000000891 \text{ mg/kg-day}$

An adult showering in water that contains 1 µg/L TCP would be exposed to approximately 0.000000089 mg/kg-day TCP by inhalation. Note that this model assumes 100% absorption of inhaled TCP vapors into systemic circulation. The actual absorption fraction may be less than 100%, but no data are available to suggest a lower value.

The same calculations for a child taking a shower in water containing 1 µg/L TCP would be as follows:

$C_{amax} = (0.001 \text{ mg/L} \times 0.1 \times 750 \text{ L/hr} \times 0.3 \text{ hr}) \div 10 \text{ m}^3 = 0.00225 \text{ mg/m}^3$

$C_a = ((0.00225 \text{ mg/m}^3 \div 2) \times 0.3 \text{ hr} + (0.00225 \text{ mg/m}^3 \times 0.12 \text{ hr})) \div (0.3 \text{ hr} + 0.12 \text{ hr}) = 0.00145 \text{ mg/m}^3$

$Dose_{inh} = (0.00145 \text{ mg/m}^3 \times 0.0005 \text{ m}^3\text{/min} \times (0.3 \text{ hr} + 0.12 \text{ hr}) \times 1\text{/day} \times 60 \text{ min/hr}) \div 37 \text{ kg} = 0.000000049 \text{ mg/kg-day}$

Thus a child showering in water that contains 1 µg/L TCP would be exposed to approximately 0.000000049 mg/kg-day TCP by inhalation.

The *Exposure Factors Handbook* (U.S. EPA 2011a) indicates that infants < 2 years old typically take baths rather than showers. No model for exposure of an infant taking a bath was identified. Thus, the Schaum et al. (1994) model for exposure in a shower will likely over-predict air concentrations of the volatile contaminant in bath water. However, the model is applied below as a possibly exaggerated estimate of inhalation exposure to an infant taking a bath in water that contains 1 µg/L TCP:

C_{amax} = (0.001 mg/L x 0.1 x 750 L/hr x 0.5 hr) ÷ 10 m^3 = 0.00375 mg/m^3

C_a = ((0.00375 mg/m^3 ÷ 2) x 0.5 hr + (0.00375 mg/m^3 x 0.12 hr)) ÷ (0.5 hr + 0.12 hr) = 0.00223 mg/m^3

$Dose_{inh}$ = (0.00223 mg/m^3 x 0.001 m^3/min x (0.5 hr + 0.12 hr) x 1/day x 60 min/hr) ÷ 10 kg = 0.00000833 mg/kg-day

An infant bathing in water that contains 1 µg/L TCP is thus estimated to be exposed to approximately 0.0000083 mg/kg-day TCP by inhalation.

Limitations and assumptions of this model include:

- The concentration of the contaminant (TCP) is assumed to be zero at the beginning of the shower. The model does not account for ambient TCP in the bathroom air, for example, if someone recently had a shower in the same bathroom.
- The model does not account for the use of an exhaust fan or other ventilation device. It assumes the bathroom is an airtight chamber.
- The time in shower (t_1), time after shower (t_2), and shower frequency were estimated based on values provided in the *Exposure Factors Handbook* (U.S. EPA 2011a). Obviously there is a wide variety in personal habits, and when available the 90th percentile values were used.

Transdermal Absorption of TCP while Showering or Bathing

In addition to inhalation exposure, bathing and showering can lead to transdermal absorption of contaminants in tap water through the skin into systemic circulation. In its *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment* (*RAGS Part E*), the U.S. EPA (2004) describes mathematical models for estimating dermal absorbed dose (DAD) during activities during which individuals are submerged in water, such as swimming or bathing. It is acknowledged that the model of submersion in water probably overestimates the water contact while standing in the flow of water in a shower. A more precise dermal absorption model for the shower scenario was not identified.

Dermal absorbed dose is calculated with the following formula:

DAD = (DA_{event} x EV x ED x EF x SA) ÷ (BW x AT), in which the variables are defined as:

Parameter	Value	Value Source
DAD = Dermally absorbed dose (mg/kg-day)	-	-
DA_{event} = Absorbed dose per event (mg/kg-event)	see formula below	U.S. EPA (2004)
SA = Skin surface area (cm^2)	adult 25000[a] child 15000[b] infant 5100[c]	US EPA (2011a)
EV = Event frequency (events/day)	1	US EPA (2011a)

EF = Exposure Frequency (days/year)	365	Assumed
ED = Exposure Duration (years)	adult 54 child 14 infant 2	Defined by age-dependent adjustment factors
BW = Body weight (kg)	adult 80 child 37 infant 10	US EPA (2011a)
AT = Averaging time (days)	25550	= 70 years x 365 d/year, U.S. EPA (2004)
a. Value used is 95th percentile of males aged 21-30 years. b. Value used is 95th percentile of children aged 11-16 years. c. Value used is 95th percentile of infants aged 1-<2 years.		

DA_{event} (the amount of contaminant absorbed per shower or bath) is calculated with the following formula:

$DA_{event} = 2 \times FA \times K_p \times C_w \times ((6 \times \tau \times t_{event})/\pi)^{0.5}$, in which the variables are identified as:

Parameter	Value	Value Source
DA_{event} = Absorbed dose per event (mg/kg-event)	-	-
FA = Fraction absorbed water (no units)	1	U.S. EPA (2004)
K_p = Dermal permeability coefficient (cm/hr)	9.39 E-3	CDC 2010
C_w = Chemical concentration in water (mg/cm^3)	Input value	-
τ = Lag time (hr)	0.151	Calculated per U.S. EPA (2004)
t_{event} = Event duration (hr)	adult 0.22 child 0.30 infant 0.50	US EPA (2011a)

For tap water that contains 1 µg/L (0.000001 mg/cm^3) TCP, the modeled dermal exposure by an adult taking a shower can be estimated by the following series of calculations:

$DA_{event} = 2 \times 1 \times 0.00939$ cm/h $\times 0.000001$ mg/cm^3 $\times ((6 \times 0.151 \times 0.22$ hr$)/\pi)^{0.5} = 2.4e\text{-}9$ mg/cm^2-event

DAD = (0.0000000024 mg/cm^2-event x 1event/day x 54 yrs x 365 days/yr x 25,000 cm^2) ÷ (80 kg x 25550 days) = 0.00000057 mg/kg-day

Thus an adult showering in water that contains 1 µg/L TCP would be exposed to approximately 0.00000057 mg/kg-day TCP by dermal absorption.

The same calculations for a child taking a shower in water containing 1 µg/L TCP would be as follows:

$DA_{event} = 2 \times 1 \times 0.00939$ cm/h $\times 0.000001$ mg/L $\times ((6 \times 0.151 \times 0.30$ hr$)/\pi)^{0.5} = 0.0000000055$ mg/cm^2-event

DAD = (0.0000000055 mg/cm^2-event x 1event/day x 14 yrs x 365 days/yr x 15,000 cm^2) ÷ (37 kg x 25550 days) = 0.00000045 mg/kg-day

Thus, a child showering in water that contains 1 µg/L TCP would be exposed to approximately 0.00000045 mg/kg-day TCP by dermal absorption.

The *Exposure Factors Handbook* (U.S. EPS 2011a) indicates that infants < 2 years old typically take baths rather than showers. The *RAGS E* dermal absorption dose model (U.S. EPA 2004) also applies to an infant taking a bath in water that contains 1 µg/L TCP in the following calculations:

DA_{event} = 2 x 1 x 0.00939 cm/h x 0.000001 mg/L x ((6 x 0.151 x 0.5 hr)/π)$^{0.5}$ = 0.0000000071 mg/cm^2-event

DAD = (0.0000000071 mg/cm^2-event x 1event/day x 2 yrs x 365 days/yr x 5100 cm^2) ÷ (10 kg x 25550 days) = 0.000000103 mg/kg-day

Thus, an infant bathing in water that contains 1 µg/L TCP would be exposed to approximately 0.0000001 mg/kg-day TCP by dermal absorption.

Total Exposure

The total daily exposure is the sum of exposure from the three routes. Table B1 (below) presents the estimated daily exposure levels of adults, children, and infants to TCP if tap water contains 1 µg/L (1 ppb) from the three anticipated exposure routes.

Table B1. Exposure to TCP (mg/kg-day) from tap water containing 1 µg/L TCP

Route	Adult (≥ 16 yrs)	Child (2-16 yrs)	Infant (≤ 2 yrs)
Oral (drinking)	0.000036	0.000038	0.000092
Inhalation (shower/bath)	0.000000089	0.00000049	0.0000083
Transdermal (shower/bath)	0.0000011	0.00000045	0.0000001
Sum of Exposures	**0.000037**	**0.000039**	**0.0001**

Appendix B. State-Level Regulations of TCO in Drinking Water

US State	Date Searched	State Limits for TCP in Public Drinking Water	Other Info or Comments	Source Hyperlink
Alabama	12/23/2011	No state MCL for TCP	TCP among contaminants which public water systems test for, but which are not regulated	http://www.adem.state.al.us/alEnviroRegLaws/files/Div7Eff5-26-09.pdf
Alaska	12/15/2011	No state MCL for TCP	Groundwater Cleanup Level = 0.0006 mg/L	http://dec.alaska.gov/spar/csp/guidance/cleanuplevels.pdf
Arizona	12/23/2011	No state MCL for TCP	TCP among contaminants that are monitored in public water systems but are not regulated	http://www.azdeq.gov/function/laws/download/1998/sdwmonitor.pdf
Arkansas	12/15/2011	No state MCL for TCP	-	http://www.healthy.arkansas.gov/programsServices/environmentalHealth/Engineering/drinkingWater/Pages/Primary.aspx
California	12/16/2011	No state MCL for TCP; Public Health Goal = 0.0007ug/L	-	http://www.oehha.ca.gov/water/phg/pdf/082009TCP_phg.pdf
Colorado	12/23/2011	No state MCL for TCP	-	http://www.cdphe.state.co.us/regulations/wqccregs/100301primarydrinkingwaternew.pdf
Connecticut	1/2/2012	No state MCL for TCP	MCL has not been established for this chemical.	http://www.ct.gov/dph/lib/dph/agency_regulations/sections/pdfs/title_19._health_and_safety/phc/chapter_ii/19-23._standards_for_quality....pdf
Delaware	1/2/2012	No state MCL for TCP	-	http://dhss.delaware.gov/dhss/dph/hsp/pubdw.html
Florida	12/23/2011	No state MCL for TCP; Health Advisory Level = 40 ug/L	Last update 9/26/2011	http://www.doh.state.fl.us/environment/community/health-advisory/HAL_list.pdf
Georgia	12/23/2011	No state MCL for TCP	-	http://rules.sos.state.ga.us/docs/391/3/5/18.pdf
Hawaii	12/23/2011	State MCL = 0.0006 mg/L	Hawaii is the only US state that has an MCL for TCP	http://hawaii.gov/health/environmental/environmental/water/sdwb/sdwb/pdf/MCL_03-04-09.pdf

US State	Date Searched	State Limits for TCP in Public Drinking Water	Other Info or Comments	Source Hyperlink
Idaho	12/23/2011	No state MCL for TCP	-	http://adm.idaho.gov/adminrules/rules/idapa58/0108.pdf
Illinois	12/23/2011	No state MCL for TCP	-	http://www.epa.state.il.us/water/compliance/drinking-water/publications/regulated-contaminants.pdf
Indiana	1/2/2012	No state MCL for TCP	-	http://www.in.gov/idem/5096.htm
Iowa	1/2/2012	No state MCL for TCP	-	http://www.iowadnr.gov/portals/idnr/uploads/water/wse/2010SAR.pdf
Kansas	12/23/2011	No state MCL for TCP	-	http://www.kdheks.gov/pws/dmcu.html#voc
Kentucky	12/23/2011	No state MCL for TCP	-	http://lrc.ky.gov/kar/401/008/250.htm
Louisiana	1/3/2012	No state MCL for TCP	-	http://new.dhh.louisiana.gov/index.cfm/page/963
Maine	1/3/2012	No state MCL for TCP	-	http://www.maine.gov/dhhs/mecdc/environmental-health/water/rules-policies/rules-policieshome.htm
Maryland	1/2/2012	No state MCL for TCP	-	http://www.mde.maryland.gov/programs/water/water_supply/documents/pws/acrs/wsp-acr-2011for2010.pdf
Massachussetts	1/2/2012	No state MCL for TCP	-	http://www.mass.gov/dep/service/regulations/310cmr22.pdf
Michigan	1/2/2012	No state MCL for TCP	-	http://www.michigan.gov/documents/deq/deq-wb-dwehs-cws-Act399_247583_7.pdf
Minnesota	12/23/2011	No state MCL for TCP; Cancer Health based value = 0.003 ug/L; chronic noncancer health based value = 20 ug/L	-	http://www.health.state.mn.us/divs/eh/water/factsheet/com/voc_soc.pdf http://www.health.state.mn.us/divs/eh/risk/guidance/gw/123triclorp.pdf
Mississippi	1/2/2012	No state MCL for TCP	-	http://www.msdh.state.ms.us/msdhsite/_static/resources/335.pdf
Missouri	1/2/2012	No state MCL for TCP	TCP among contaminants which public water systems test for, but which are not regulated	http://www.sos.mo.gov/adrules/csr/current/10csr/10c60-4.pdf
Montana	12/23/2011	No state MCL for TCP	"State regulations no more stringent than federal regulations or guidelines"	http://data.opi.mt.gov/bills/mca/75/6/75-6-116.htm
Nebraska	12/23/2011	No state MCL for TCP	TCP among contaminants which public water systems test for, but which are not regulated	http://dhhs.ne.gov/Documents/PWSAnnualReport.pdf

US State	Date Searched	State Limits for TCP in Public Drinking Water	Other Info or Comments	Source Hyperlink
Nevada	12/23/2011	No state MCL for TCP	-	http://ndep.nv.gov/bsdw/docs/approved-analytica_methods.pdf
New Hampshire	1/2/2012	No state MCL for TCP	-	http://des.nh.gov/organization/commissioner/legal/rules/documents/env-dw702-706.pdf
New Jersey	1/2/2012	A health-based drinking water guidance value of 0.005 µg/L for 1,2,3-trichloropropane was developed by the NJDEP Division of Science, Research and Technology (DSRT) in 1999.	New Jersey Drinking Water Quality Institute Recommended Changes to Existing Maximum Contaminant Level from 0.0013 ug/L to 0.03 ug/L in 2009.	http://www.nj.gov/dep/watersupply/dwqi_mcl_09_recommend_report_final.pdf
New Mexico	12/23/2011	No state MCL for TCP	-	http://www.nmenv.state.nm.us/dwb/contaminants/
New York	1/2/2012	No state MCL for TCP	TCP among contaminants which public water systems test for, but which are not regulated	http://www.health.ny.gov/regulations/nycrr/title_10/part_5/subpart_5-1_tables.htm
North Carolina	12/23/2011	No state MCL for TCP	-	http://www.ncwater.org/pws/rules/SECTION_1500.pdf
North Dakota	12/23/2011	No state MCL for TCP	-	http://www.ndhealth.gov/MF/forms/acr/2009acr.pdf
Ohio	12/23/2011	No state MCL for TCP	-	http://epa.ohio.gov/portals/28/documents/DWStandardsList.pdf
Oklahoma	12/23/2011	No state MCL for TCP	-	http://www.deq.state.ok.us/wqdnew/pws/index.html
Oregon	12/23/2011	No state MCL for TCP	-	http://public.health.oregon.gov/HealthyEnvironments/DrinkingWater/Rules/Documents/61-0030.pdf
Pennsylvania	1/2/2012	No state MCL for TCP	-	http://www.portal.state.pa.us/portal/server.pt/community/drinking_water_regulations%2C_standards___resources/10544#Standards
Rhode Island	1/2/2012	No state MCL for TCP	-	http://www.health.ri.gov/forms/reporting/waterquality/VolatileOrganicCompounds.xls
South Carolina	12/23/2011	No state MCL for TCP	-	http://www.scdhec.gov/environment/water/docs/dwswp.pdf
South Dakota	12/23/2011	No state MCL for TCP	-	http://denr.sd.gov/des/dw/VOC.aspx

US State	Date Searched	State Limits for TCP in Public Drinking Water	Other Info or Comments	Source Hyperlink
Tennessee	12/23/2011	No state MCL for TCP	-	http://tn.gov/sos/rules/1200/1200-05/1200-05-01.20090606.pdf
Texas	12/23/2011	No state MCL for TCP	-	http://www.tceq.texas.gov/assets/public/compliance/monops/water/02twqmar/10_pws.pdf
Utah	12/23/2011	No state MCL for TCP	-	http://www.drinkingwater.utah.gov/documents/rule_summaries/R309-103_Summary.pdf
Vermont	1/2/2012	No state MCL for TCP	-	http://www.anr.state.vt.us/dec/watersup/wsrule/Vermont_WSR_December_2010.pdf
Virginia	12/23/2011	No state MCL for TCP	Virginia drinking water standards are the same as the Environmental Protection Agency (EPA) standards in the Safe Drinking Water Act.	http://www.vdh.state.va.us/odw/FAQ.htm
Washington	12/24/2011	No state MCL for TCP	TCP among contaminants which public water systems test for, but which are not regulated	http://www.doh.wa.gov/ehp/dw/Publications/331-289_electronic_reporting_8_25_05_web.pdf
West Virginia	12/23/2011	No state MCL for TCP	-	http://www.wvdhhr.org/oehs/eed/c%26e/Documents/2007_req_contaminant_list_1.pdf
Wisconsin	1/2/2012	No state MCL for TCP	-	http://dnr.wi.gov/org/water/dwg/forms/operator.pdf
Wyoming	12/23/2011	No state MCL for TCP	Wyoming is the only State that has not applied to the US Environmental Protection Program for authority to administer the public water supply program. Therefore, Region 8 directly implements the Safe Drinking Water Act in the State of Wyoming.	http://www.epa.gov/Region8/water/dwhome/wyomingdi.html

Appendix C. TCP Concentrations in Water Samples In Hawaii (2000 to 2011)

The following pages and Tables C1 through C16 present the TCP concentrations reported in WL and TP samples at each water treatment facility in Hawaii as reported by the Hawaii Department of Health (HDOH 2011a).

Each table represents the water treatment facility as indicated. The data in each Table is presented chronologically, starting from earliest reported data to most recent. When more than one sample of the same type (WL or TP) was collected on the same day, the range of results is provided.

"ND" indicates that TCP was not detected in that sample.

"No data" indicates that no TCP concentration data were reported for that sample type on that date.

Maui

Five water treatment facilities on the island of Maui reported TCP concentrations in water samples. Facilities at Makawao, Lahaina, and Honokohau are operated by the Maui Department of Water Supply, while Kaanapali and Maunaolu are operated privately.

Kaanapali

WL and/or TP samples were collected and analysed on 50 dates between 2/14/2000 and 9/26/2011, although on several of these dates, only one sample type (WL or TP) was collected. No WL sample data were available after 5/19/2008. TCP was detected at concentrations up to 1.58 µg/L on almost all WL sample dates between 2/16/2000 and 5/19/2008. TCP was detected in TP samples on 13 (28%) of 47 dates reported, with concentrations ranging from 0.09 to 0.36 µg/L. TCP concentrations exceeded the MCL of 0.6 µg/L in almost all WL samples, but none of the TP samples had TCP concentrations above the MCL. Based on current regulatory standards, the water treatment facility at Kaanapali appears to effectively protect the public from TCP in effluent water.

A tabular presentation of these data are in Table C1 of Appendix C below.

Table C1. TCP Concentrations in WL and TP Samples Collected at Kaanapali

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
2/14/2000	Wells P4, P5, P6	0.5 to 1.6	Upper Puukolii Reservoir	ND
6/19/2000	Wells P4, P5, P6	ND to 0.79	Upper Puukolii Reservoir	ND
7/25/2000	Wells P4, P5, P6	ND to 1.54	Upper Puukolii Reservoir	ND
10/11/2000	Wells P4, P5, P6	ND to 0.81	Upper Puukolii Reservoir	0.09
10/23/2000	No data	No Data	Upper Puukolii Reservoir	0.11
3/6/2001	Wells P4, P5, P6	0.65 to 1.58	Upper Puukolii Reservoir	0.09
4/16/2001	Wells P4, P5, P6	ND to 0.95	Upper Puukolii Reservoir	0.14
7/18/2001	Wells P4, P6	0.48 to 0.76	Upper Puukolii Reservoir	0.18
10/31/2001	Wells P4, P6	0.42 to 0.62	Upper Puukolii Reservoir	0.09
2/27/2002	Wells P5, P6	0.81 to 1.36	Upper Puukolii Reservoir	ND
5/20/2002	Wells P4, P5, P6	0.52 to 0.82	Upper Puukolii Reservoir	ND
7/22/2002	Wells P4, P5, P6	0.47 to 0.76	Upper Puukolii Reservoir	ND
9/19/2002	Wells P4, P5, P6	ND	Upper Puukolii Reservoir	ND
11/7/2002	Wells P5, P6	0.45 to 0.67	Upper Puukolii Reservoir	ND
2/4/2003	Wells P5, P6	ND to 0.66	Upper Puukolii Reservoir	ND
5/22/2003	Wells P4, P5, P6	0.48 to 0.66	Upper Puukolii Reservoir	ND
8/25/2003	Well P6	0.44	Upper Puukolii Reservoir	ND
11/20/2003	Wells P4, P6	0.46 to 0.98	No data	No data
2/17/2004	Wells P4, P6	ND to 0.57	Upper Puukolii Reservoir	0.11
4/21/2004	Wells P4, P5, P6	0.54 to 0.94	Upper Puukolii Reservoir	0.32
7/27/2004	Wells P4, P5, P6	0.48 to 0.71	Upper Puukolii Reservoir	ND
10/4/2004	No data	No data	Upper Puukolii Reservoir	ND
10/11/2004	Wells P4, P5, P6	0.42 to 0.82	No data	No data
2/9/2005	Wells P4, P5, P6	ND to 1.04	Upper Puukolii Reservoir	ND
4/18/2005	Wells P4, P5, P6	0.62 to 0.93	Upper Puukolii Reservoir	ND
8/8/2005	Wells P4, P5, P6	0.48 to 0.85	Upper Puukolii Reservoir	0.08
10/20/2005	Wells P4, P5, P6	0.52 to 0.92	Upper Puukolii Reservoir	0.15
3/7/2006	Wells P4, P5, P6	0.43 to 0.82	Upper Puukolii Reservoir	ND

Table C1. TCP Concentrations in WL and TP Samples Collected at Kaanapali

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
5/3/2006	Wells P4, P5, P6	0.48 to 1.05	Upper Puukolii Reservoir	ND
8/7/2006	Wells P4, P5, P6	0.56 to 0.94	Upper Puukolii Reservoir	ND
10/25/2006	Wells P4, P5, P6	0.56 to 1.05	Upper Puukolii Reservoir	ND
1/24/2007	Wells P5, P6	0.8 to 0.97	Upper Puukolii Reservoir	ND
4/18/2007	Wells P5, P6	0.67 to 0.88	Upper Puukolii Reservoir	ND
7/31/2007	Wells P5, P6	0.69 to 0.96	Upper Puukolii Reservoir	ND
8/15/2007	Wells P4	0.72	No data	No data
10/9/2007	Wells P4, P5, P6	0.59 to 1.1	Upper Puukolii Reservoir	ND
2/25/2008	No data	No data	Upper Puukolii Reservoir	ND
5/19/2008	Wells P4, P5, P6	0.5 to 0.91	Upper Puukolii Reservoir	ND
7/21/2008	No data	No data	Upper Puukolii Reservoir	0.12
10/27/2008	No data	No data	Upper Puukolii Reservoir	0.18
2/10/2009	No data	No data	Upper Puukolii Reservoir	0.36
4/27/2009	No data	No data	Upper Puukolii Reservoir	ND
8/25/2009	No data	No data	Upper Puukolii Reservoir	ND
10/20/2009	No data	No data	Upper Puukolii Reservoir	ND
3/29/2010	No data	No data	Upper Puukolii Reservoir	ND
5/10/2010	No data	No data	Upper Puukolii Reservoir	ND
8/30/2010	No data	No data	Upper Puukolii Reservoir	ND
3/1/2011	No data	No data	Upper Puukolii Reservoir	ND
5/9/2011	No data	No data	Upper Puukolii Reservoir	ND
9/6/2011	No data	No data	Upper Puukolii Reservoir	ND

Makawao

No WL data were reported for the facility at Makawao. Twenty-eight TP samples samples were collected between 9/16/2002 and 2/22/1020. Of these, TCP was detected in 16 TP samples at concentrations of up to 0.174 µg/L. All TCP concentrations in TP samples were below the MCL. Based on current regulatory standards, the water treatment facility at Makawao appears to effectively protect the public from TCP in effluent water. No WL samples were reported.

A tabular presentation of these data are in Table C2 below.

Table 2C. TCP Concentrations in TP Samples Collected at Makawao

Date	TP Conc. (µg/L)		Date	TP Conc. (µg/L)
9/16/2002	ND		2/4/2005	0.14
10/31/2002	ND		3/15/2005	0.15
10/31/2002	ND		6/15/2005	0.16
2/18/2003	ND		8/10/2005	0.15
2/18/2003	ND		3/9/2007	0.174
5/15/2003	ND		9/30/2008	ND
5/15/2003	0.13		1/12/2009	0.16
5/29/2003	0.13		2/24/2009	0.09
8/27/2003	ND		5/12/2009	0.15
8/27/2003	0.14		5/12/2009	ND
3/15/2004	0.14		8/17/2009	0.15
3/15/2004	ND		8/17/2009	ND
7/28/2004	0.14		2/22/2010	0.17
11/8/2004	0.15		2/22/2010	ND

Lahaina

WL and /or TP samples were collected and analysed on 36 dates between 9/12/2000 and 2/23/2010. WL samples were collected on 31 dates within this range, and of those, TCP was detected on 24 dates at concentrations up to 0.09 ug/L. TP samples were collected on 15 dates, and TCP was detected at up to 0.06 ug/L on 5 days. All TCP concentrations in WL and TP samples were below the MCL.

A tabular presentation of these data are in Table C3 below.

Table C2. TCP Concentrations in WL and TP Samples Collected at Lahaina

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
9/12/2000	Honokohua A (Napili D) Well Head, Napili C Well Head	ND to 0.05	No Data	No data
9/25/2000	Napili C Well Head	0.05	No Data	No data
10/9/2000	Honokohua A (Napili D) Well Head, Napili C Well Head	0.04 to 0.05	No Data	No data
10/25/2000	Honokohua A (Napili D) Well Head	0.04	No Data	No data
11/21/2000	Honokohua A (Napili D) Well Head	ND	No Data	No data
2/21/2001	Honokohua A (Napili D) Well Head	ND	No Data	No data
5/1/2001	Honokohua A (Napili D) Well Head	0.05	No Data	No data
7/16/2001	Honokohua A (Napili D) Well Head, Napili C Well Head	ND to 0.06	No Data	No data
8/28/2001	Napili C Well Head	ND	No Data	No data
10/10/2001	Napili C Well Head	0.06	No Data	No data
11/7/2001	Honokohua A (Napili D) Well Head	ND	No Data	No data
2/25/2002	Napili C Well Head	ND	No Data	No data
5/21/2002	Honokohua A (Napili D) Well Head, Napili C Well Head	ND to 0.06	No Data	No data
7/23/2002	Honokohua A (Napili D) Well Head, Napili C Well Head	0.04 to 0.06	No Data	No data
9/16/2002	Honokohua A (Napili D) Well Head, Napili C Well Head	ND	Air Relief after Alaeloa 1MG Tank	ND
10/31/2002	Honokohua A (Napili D) Well Head, Napili C Well Head	ND to 0.05	No data	No data
2/18/2003	Honokohua A (Napili D) Well Head	ND	No data	No data
5/15/2003	Honokohua A (Napili D) Well Head	0.05	No data	No data
9/15/2003	Honokohua A (Napili D) Well Head	0.06	No data	No data
11/24/2003	Honokohua A (Napili D) Well Head, Napili C Well Head	ND to 0.09	No data	No data
3/17/2004	Honokohua A (Napili D) Well Head, Napili C Well Head	0.06 to 0.07	Air Relief after Alaeloa 1MG Tank	0.05
6/15/2004	Napili C Well Head	0.07	Air Relief after Alaeloa 1MG Tank	ND
7/28/2004	Honokohua A (Napili D) Well Head, Napili C Well Head	0.05 to 0.06	Air Relief after Alaeloa 1MG Tank	ND
11/22/2004	Honokohua A (Napili D) Well Head	0.06	Air Relief after Alaeloa 1MG Tank	ND
11/23/2004	Napili C Well Head	0.07	No data	No data
2/7/2005	Honokohua A (Napili D) Well Head, Napili C Well Head	0.04 to 0.05	Air Relief after Alaeloa 1MG Tank	0.05
6/15/2005	Napili C Well Head	0.06	Air Relief after Alaeloa 1MG Tank	ND

Table C2. TCP Concentrations in WL and TP Samples Collected at Lahaina

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
8/10/2005	Napili C Well Head	0.08	Air Relief after Alaeloa 1MG Tank	ND
11/30/2005	Honokohua A (Napili D) Well Head	0.09	No data	No data
11/22/2006	No data	No data	Air Relief after Alaeloa 1MG Tank	ND
3/9/2007	Napili C Well Head	0.088	Air Relief after Alaeloa 1MG Tank	ND
9/25/2007	No data	No data	Air Relief after Alaeloa 1MG Tank	ND
1/12/2009	Napili C Well Head	0.05	Air Relief after Alaeloa 1MG Tank	0.05
5/11/2009	No data	No data	Air Relief after Alaeloa 1MG Tank	ND
9/21/2009	No data	No data	Air Relief after Alaeloa 1MG Tank	0.06
2/23/2010	No data	No data	Air Relief after Alaeloa 1MG Tank	0.05

Honokohau

In the dataset provided by HDOH, 21 TP samples collected from 2/16/2000 to 11/22/2004 were reported. None of the TP samples had detectable TCP except for one sample containing 0.05 µg/L on 9/16/2002. No TCP concentrations for WL samples at Honokohau were provided. However, given the absence of detectable TCP in the TP samples at Honokohau, the water treatment at this facility appears to effectively protect the public from TCP in the water. No WL Samples were reported.

A tabular presentation of these data are in Table C4 below.

Table C4. TCP Concentrations in TP Samples Collected at Honokohau

Date	TP Conc. (µg/L)	Date	TP Conc. (µg/L)
2/16/2000	ND	10/31/2002	ND
6/7/2000	ND	10/31/2002	ND
7/25/2000	ND	2/18/2003	ND
10/9/2000	ND	5/15/2003	ND
2/21/2001	ND	10/2/2003	ND
5/1/2001	ND	12/10/2003	ND
7/16/2001	ND	3/16/2004	ND
11/29/2001	ND	5/18/2004	ND
2/25/2002	ND	7/28/2004	ND
5/21/2002	ND	11/22/2004	ND
9/16/2002	0.05		

Maunaolu

WL and/or TP samples were collected on 31 dates between 2/19/2004 to 3/7/2011. WL data were available for 18 dates within this range, with the latest samples reported on 7/21/2008. TCP was detected in 10 WL samples at concentrations up to 1.32 µg/L.

TP samples were reported for 30 dates between 2/19/2004 to 3/7/2011, and of these TCP was detected in 4 samples at concentrations up to 0.25 ppb. All TCP concentrations in TP samples were below the current MCL.

A tabular presentation of these data are in Table C5 below.

Table C5. TCP Concentrations in WL and TP Samples Collected at Maunaolu Plantation

Date	WL Collection Location(s)	WL Conc. (µg/L)	TP Location	TP Conc. (µg/L)
2/19/2004	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	ND
3/23/2004	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	No Data
4/21/2004	Maunaolu-Smith Wellhead	0.79	Maunaolu-Smith LAG GAC Effluent	ND
5/10/2004	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	ND
7/27/2004	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	ND
11/3/2004	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	ND
11/15/2004	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	ND
3/17/2005	Maunaolu-Smith Wellhead	0.71	Maunaolu-Smith LAG GAC Effluent	ND
5/25/2005	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
10/20/2005	Maunaolu-Smith Wellhead	0.85	Maunaolu-Smith LAG GAC Effluent	ND
2/7/2006	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	ND
5/23/2006	Maunaolu-Smith Wellhead	ND	Maunaolu-Smith LAG GAC Effluent	ND
8/7/2006	Maunaolu-Smith Wellhead	0.85	Maunaolu-Smith LAG GAC Effluent	ND
10/25/2006	Maunaolu-Smith Wellhead	0.91	Maunaolu-Smith LAG GAC Effluent	ND
1/24/2007	Maunaolu-Smith Wellhead	0.87	Maunaolu-Smith LAG GAC Effluent	ND
4/18/2007	Maunaolu-Smith Wellhead	0.75	Maunaolu-Smith LAG GAC Effluent	ND
7/31/2007	Maunaolu-Smith Wellhead	0.88	Maunaolu-Smith LAG GAC Effluent	0.08
10/9/2007	Maunaolu-Smith Wellhead	1.32	Maunaolu-Smith LAG GAC Effluent	0.2
2/25/2008	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
5/20/2008	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
7/21/2008	Maunaolu-Smith Wellhead	0.93	Maunaolu-Smith LAG GAC Effluent	ND
10/27/2008	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
2/10/2009	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
4/27/2009	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
8/25/2009	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
10/20/2009	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
2/8/2010	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
5/10/2010	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
8/16/2010	No data	No data	Maunaolu-Smith LAG GAC Effluent	ND
2/22/2011	No data	No data	Maunaolu-Smith LAG GAC Effluent	0.04
3/7/2011	No data	No data	Maunaolu-Smith LAG GAC Effluent	0.25

Oahu

Nine water treatment facilities on the island of Oahu reported TCP concentrations in WL and TP water samples. Most of these water treatment facilities are operated by the Honolulu Board of Water Supply. The water facilities at Kunia Village, Hawaii Country Club, and Kipapa Acres are operated privately.

Kunia Village

WL and /or TP samples were collected and analysed on 51 dates between 2/8/2000 and 9/6/2011. Within this range, WL samples were collected on 41 days, and of those TCP was detected on 35 days at concentrations of up to 0.33 µg/L. No TCP was detected in TP samples during the range of dates that had corresponding WL analyses. TCP was detected in two samples, at 0.05 ppm on 10/21/08 and 0.13 ppm on 1/23/2009. No WL data were available after 2/20/2008. TCP concentrations in all WL and TP samples were below the current MCL.

A tabular presentation of these data are in Table C6 below.

Table C6. TCP Concentrations in WL and TP Samples Collected at Kunia Village

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
2/8/2000	Del Monte Kunia Well 4 Pump Manifold	ND	House 817 – Outside Tap	ND
5/11/2000	Del Monte Kunia 3 (Navy) Pump Manifold	0.15	House 817 – Outside Tap	ND
6/9/2000	Del Monte Kunia 3 (Navy) Pump Manifold	ND	House 817 – Outside Tap	ND
9/25/2000	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.08	House 817 – Outside Tap	ND
10/4/2000	Del Monte Kunia Well 4 Pump Manifold	0.08	House 817 – Outside Tap	ND
10/12/2000	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.11 to 0.13	House 817 – Outside Tap	ND
2/22/2001	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.11	House 817 – Outside Tap	ND
5/8/2001	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.09	House 817 – Outside Tap	ND
7/11/2001	Del Monte Kunia 3 (Navy) Pump Manifold	ND to 0.11	House 817 – Outside Tap	ND
8/3/2001	Del Monte Kunia Well 4 Pump Manifold	ND to 0.09	House 817 – Outside Tap	ND
11/30/2001	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.12	House 817 – Outside Tap	ND
12/3/2001	Del Monte Kunia Well 4 Pump Manifold	ND to 0.08	No data	No data
2/2/2002	Del Monte Kunia Well 4 Pump Manifold	ND	No data	No data
2/22/2002	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.17	House 817 – Outside Tap	ND
6/13/2002	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.12	House 817 – Outside Tap	ND
8/21/2002	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.14	House 817 – Outside Tap	ND
12/4/2002	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.15	House 817 – Outside Tap	ND
1/27/2003	Del Monte Kunia Well 4 Pump Manifold	ND	House 817 – Outside Tap	ND
1/30/2003	Del Monte Kunia 3 (Navy) Pump Manifold	ND to 0.15	House 817 – Outside Tap	ND
5/22/2003	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.13	House 817 – Outside Tap	ND
6/3/2003	No data	No data	House 817 – Outside Tap	ND
8/5/2003	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.14	House 817 – Outside Tap	ND
10/7/2003	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.13	House 817 – Outside Tap	ND
12/4/2003	Del Monte Kunia 3 (Navy) Pump Manifold	ND	No data	No data
3/10/2004	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.12	House 817 – Outside Tap	ND

Table C6. TCP Concentrations in WL and TP Samples Collected at Kunia Village

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
3/30/2004	Del Monte Kunia, Well 4 Pump Manifold	ND	No data	No data
5/6/2004	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.15	House 817 – Outside Tap	ND
7/28/2004	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.16	House 817 – Outside Tap	ND
12/16/2004	Del Monte Kunia 3 (Navy) Pump Manifold	ND to 0.15	House 817 – Outside Tap	ND
2/28/2005	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.14 to 0.17	House 817 – Outside Tap	ND
5/24/2005	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.13	House 817 – Outside Tap	ND
8/26/2005	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.22	House 817 – Outside Tap	ND
12/2/2005	Del Monte Kunia 3 (Navy) Pump Manifold	0.22	House 817 – Outside Tap	ND
3/7/2006	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.21	House 817 – Outside Tap	ND
6/20/2006	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	ND to 0.24	House 817 – Outside Tap	ND
8/30/2006	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.2 to 0.23	House 817 – Outside Tap	ND
12/4/2006	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.22 to 0.29	House 817 – Outside Tap	ND
2/20/2007	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.19 to 0.28	House 817 – Outside Tap	ND
4/18/2007	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.16 to 0.27	House 817 – Outside Tap	ND
9/14/2007	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.22 to 0.28	House 817 – Outside Tap	ND
10/11/2007	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.29 to 0.33	House 817 – Outside Tap	ND
2/20/2008	Del Monte Kunia 3 (Navy), Well 4 Pump Manifold	0.21 to 0.23	House 817 – Outside Tap	ND
4/24/2008	No data	No data	House 817 – Outside Tap	ND
7/15/2008	No data	No data	House 817 – Outside Tap	ND
10/21/2008	No data	No data	House 817 – Outside Tap	0.05
1/23/2009	No data	No data	House 817 – Outside Tap	0.13
4/9/2009	No data	No data	House 817 – Outside Tap	ND
9/1/2009	No data	No data	House 817 – Outside Tap	ND
2/17/2010	No data	No data	House 817 – Outside Tap	ND
8/30/2010	No data	No data	House 817 – Outside Tap	ND
9/6/2011	No data	No data	House 817 – Outside Tap	ND

Hawaii Country Club

Corresponding WL and TP samples collected and analysed at the Hawaii Country Club were reported for 30 dates from 2/8/2000 to 2/20/2008. On three dates during this time period, TCP was detected in TP samples at concentrations of 0.05 to 0.12 µg/L. TCP was detected in 24 WL samples during this date range with concentrations ranging from 0.04 to 0.46. TCP was not detected in six WL samples. From 4/24/2008 to 10/3/2011, only TP samples were reported (i.e., no WL samples were reported for this date range), and all TP samples had non-detectable TCP, except for a concentration of 0.09 µg/L in a TP sample collected at the clubhouse restroom on 4/26/2010. For the dates on which WL and TP samples were collected and analysed, it appears that the water treatment center at Hawaii Country Club effectively removed TCP from ground water. TCP concentrations in TP samples collected from 2/8/2000 to 10/3/2011 were all below the current MCL of 0.6 µg/L.

A tabular presentation of these data are in Table C7 below.

Table C7. TCP Concentrations in WL and TP Samples Collected at Hawaii Country Club

Date	WL Location	WL Conc. (µg/L)	TP Location	TP Conc. Range (µg/L)
2/8/2000	Hawai CC Well Pumphead	ND	Pro Shop Sink	ND
5/11/2000	Hawai CC Well Pumphead	0.31	Clubhouse Restroom, Pro Shop Sink	0.05
6/9/2000	Hawai CC Well Pumphead	ND	Clubhouse Restroom, Pro Shop Sink	ND
8/14/2000	Hawai CC Well Pumphead	0.27	Clubhouse Restroom, Pro Shop Sink	0.08 to 0.12
10/12/2000	Hawai CC Well Pumphead	ND	Clubhouse Restroom, Pro Shop Sink	ND
2/26/2001	Hawai CC Well Pumphead	0.27	Clubhouse Restroom, Pro Shop Sink	0.07 to 0.09
5/8/2001	Hawai CC Well Pumphead	0.28	Clubhouse Restroom, Pro Shop Sink	ND
7/11/2001	Hawai CC Well Pumphead	0.28	Clubhouse Restroom, Pro Shop Sink	ND
11/28/2001	Hawai CC Well Pumphead	0.27	Clubhouse Restroom, Pro Shop Sink	ND
2/22/2002	Hawai CC Well Pumphead	0.36	Clubhouse Restroom	ND
7/30/2002	Hawai CC Well Pumphead	ND	Clubhouse Restroom	ND
11/14/2002	Hawai CC Well Pumphead	0.29	Clubhouse Restroom	ND
1/27/2003	Hawai CC Well Pumphead	0.32	Clubhouse Restroom	0.08
6/3/2003	Hawai CC Well Pumphead	0.28	Clubhouse Restroom	ND
8/12/2003	Hawai CC Well Pumphead	ND	Clubhouse Restroom	ND
10/7/2003	Hawai CC Well Pumphead	0.25	Clubhouse Restroom	ND
2/9/2004	Hawai CC Well Pumphead	0.31	Clubhouse Restroom	ND
8/17/2004	Hawai CC Well Pumphead	0.29	Clubhouse Restroom	ND
10/14/2004	Hawai CC Well Pumphead	0.36	Clubhouse Restroom	ND
1/25/2005	Hawai CC Well Pumphead	0.29	Clubhouse Restroom	ND
5/23/2005	Hawai CC Well Pumphead	0.27	Clubhouse Restroom	ND
12/2/2005	Hawai CC Well Pumphead	0.17	Clubhouse Restroom	ND
3/8/2006	Hawai CC Well Pumphead	0.26	Clubhouse Restroom	ND
5/18/2006	Hawai CC Well Pumphead	0.27	Clubhouse Restroom	ND
8/25/2006	Hawai CC Well Pumphead	0.04	Clubhouse Restroom	ND
12/4/2006	Hawai CC Well Pumphead	0.38	Clubhouse Restroom	ND
2/20/2007	Hawai CC Well Pumphead	0.39	Clubhouse Restroom	ND

Table C7. TCP Concentrations in WL and TP Samples Collected at Hawaii Country Club

Date	WL Location	WL Conc. (µg/L)	TP Location	TP Conc. Range (µg/L)
4/18/2007	Hawai CC Well Pumphead	0.43	Clubhouse Restroom	ND
7/10/2007	Hawai CC Well Pumphead	0.46	Clubhouse Restroom	ND
2/20/2008	Hawai CC Well Pumphead	ND	Clubhouse Restroom	ND

HNL-Windward-Pearl Harbor

A comparison of WL to TP samples that were collected and analysed at HNL-Windward-Pearl Harbor cannot be performed, because the WL samples were collected between 5/8/2000 and 7/7/2009, while the TP samples were collected between 1/27/2010 and 4/27/2011. Thus, there were no dates on which both WL and TP samples were collected. Of the 27 WL samples collected and analysed, eight (30%) had detectable TCP at concentrations of 0.043 to 0.05 µg/L. Two of the four TP samples collected and analysed at HNL-Windward-Pearl Harbor in 2010 and 2011 contained detectable TCP at 0.12 and 0.16 µg/L, both values below the current MCL. Because no data for WL and TP samples that were collected on or near the same dates were available, the effectiveness of removal of TCP from groundwater at the HNL-Windward-Pearl Harbor water treatment facility cannot be determined.

A tabular presentation of these data are in Table C8 below.

Table C8. TCP Concentrations in WL and TP Samples Collected at HNL-Windward-Pearl Harbor

Date	WL Location	WL Conc. (µg/L)	TP Location	TP Conc. Range (µg/L)
5/8/2000	Pearl City Shaft Pumphead	ND	No data	No data
8/11/2000	Pearl City Shaft Pumphead	ND	No data	No data
9/12/2002	Pearl City Shaft Pumphead	ND	No data	No data
11/6/2003	Pearl City Shaft Pumphead	ND	No data	No data
3/30/2004	Pearl City Shaft Pumphead	0.043	No data	No data
4/21/2004	Pearl City Shaft Pumphead	0.048	No data	No data
8/3/2004	Pearl City Shaft Pumphead	0.05	No data	No data
10/29/2004	Pearl City Shaft Pumphead	0.05	No data	No data
1/25/2005	Pearl City Shaft Pumphead	ND	No data	No data
5/5/2005	Pearl City Shaft Pumphead	ND	No data	No data
7/7/2005	Pearl City Shaft Pumphead	0.04	No data	No data
11/4/2005	Pearl City Shaft Pumphead	ND	No data	No data
2/6/2006	Pearl City Shaft Pumphead	ND	No data	No data
5/17/2006	Pearl City Shaft Pumphead	ND	No data	No data
8/8/2006	Pearl City Shaft Pumphead	ND	No data	No data
10/19/2006	Pearl City Shaft Pumphead	ND	No data	No data
1/26/2007	Pearl City Shaft Pumphead	ND	No data	No data
4/16/2007	Pearl City Shaft Pumphead	ND	No data	No data
7/10/2007	Pearl City Shaft Pumphead	ND	No data	No data
10/2/2007	Pearl City Shaft Pumphead	ND	No data	No data
2/20/2008	Pearl City Shaft Pumphead	ND	No data	No data
4/1/2008	Pearl City Shaft Pumphead	0.04	No data	No data
7/16/2008	Pearl City Shaft Pumphead	0.04	No data	No data

Table C8. TCP Concentrations in WL and TP Samples Collected at HNL-Windward-Pearl Harbor

Date	WL Location	WL Conc. (µg/L)	TP Location	TP Conc. Range (µg/L)
10/28/2008	Pearl City Shaft Pumphead	0.05	No data	No data
1/8/2009	Pearl City Shaft Pumphead	ND	No data	No data
4/14/2009	Pearl City Shaft Pumphead	ND	No data	No data
7/7/2009	Pearl City Shaft Pumphead	ND	No data	No data
1/27/2010	No data	No data	Punanini Chlor, Bridge betweenKaahele & Hekaha	0.12
2/23/2010	No data	No data	Punanini Chlor, Bridge betweenKaahele & Hekaha	0.16
5/26/2010	No data	No data	Punanini Chlor, Bridge betweenKaahele & Hekaha	ND
4/27/2011	No data	No data	Punanini Chlor, Bridge betweenKaahele & Hekaha	ND

Kipapa Acres

No WL sample data were available for Kipapa Acres. TCP analysis results were reported for TP samples collected on 65 dates between 2/22/2000 and 10/3/2011. During this date range, TCP was detected on 20 days at concentrations ranging from 0.04 to 0.72 µg/L. The TCP concentrations in TP water samples exceeded the current MCL of 0.6 µg/L on 11 dates (17%) in this data set, however this has not occurred since 7/19/2005. Without data on source water, it is impossible to assess the performance of the Kipapa Acres water treatment facility in its ability to remove TCP from water. No TP samples collected after July 2005 have exceeded the current MCL for TCP. No WL samples were reported.

A tabular presentation of these data are in Table C9 below.

Table C9. TCP Concentrations in TP Samples Collected at Kipapa Acres

Date	TP Conc. (µg/L)	Date	TP Conc. (µg/L)	Date	TP Conc. (µg/L)
2/22/2000	ND	10/25/2002	0.57	8/11/2006	ND
5/15/2000	0.57	2/7/2003	ND	11/17/2006	ND
5/30/2000	ND	2/7/2003	0	2/26/2007	ND
7/6/2000	ND	5/22/2003	ND	4/26/2007	ND
7/31/2000	ND	5/22/2003	0	7/13/2007	ND
10/13/2000	ND	8/19/2003	ND	10/2/2007	ND
10/13/2000	ND	8/19/2003	0.66	1/30/2008	ND
2/22/2001	ND	10/21/2003	0.69	7/14/2008	ND
2/22/2001	0.55	11/20/2003	ND	10/22/2008	0.04
4/25/2001	ND	2/20/2004	0.71	1/23/2009	ND
4/25/2001	0.47	2/20/2004	ND	4/7/2009	ND
7/16/2001	ND	3/17/2004	ND	7/29/2009	ND
7/16/2001	0.58	4/27/2004	0.69	10/6/2009	ND
11/28/2001	ND	4/27/2004	ND	1/27/2010	ND
11/28/2001	0.65	8/2/2004	0.72	4/15/2010	ND
2/21/2002	ND	12/2/2004	0.65	9/8/2010	ND
2/21/2002	0.67	12/2/2004	ND	10/13/2010	ND
6/13/2002	ND	2/15/2005	0.63	2/7/2011	ND
6/13/2002	ND	5/19/2005	0.71	4/4/2011	0.06
7/15/2002	ND	7/19/2005	0.64	7/5/2011	ND
7/15/2002	0.57	12/1/2005	0.46	10/3/2011	ND
10/25/2002	ND	2/14/2006	ND		

Waialua-Haleiwa

WL samples were reported for 61 dates between 2/4/2000 and 10/5/2009, and TP samples were reported on three dates, 10/5/2009, 2/11/2010 and 4/27/2010. There were no dates on which both WL and TP samples were collected. WL samples contained TCP at concentrations up to 0.11 µg/L. Two of the TP samples had no detectable TCP, while the remaining sample contained 0.11 µg/L TCP. None of the WL or TP samples exceeded the current MCL for TCP.

A tabular presentation of these data are in Table C10 below.

Table C10. TCP Concentrations in WL and TP Samples Collected at Waialua-Haleiwa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
2/4/2000	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
2/10/2000	By Gate, Pumphead	ND	No data	No data
5/11/2000	By Gate, Pumphead	0.63	No data	No data
	By Gate, Pumphead	0.56	No data	No data
5/16/2000	GAC Effluent Tap by Contractor #1	**0.49**	No data	No data
5/16/2000	Toward Waianae Pumphead	0.53	No data	No data
6/1/2000	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
6/8/2000	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
7/6/2000	By Gate, Pumphead	0.56	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
8/3/2000	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.54**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
8/8/2000	Toward Waianae Pumphead	ND	No data	No data
8/9/2000	By Gate, Pumphead	ND	No data	No data
10/18/2000	By Gate, Pumphead	0.56	No data	No data
	By Gate, Pumphead	0.57	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
10/23/2000	GAC Effluent Tap by Contractor #1	**0.56**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.55	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
2/26/2001	GAC Effluent Tap by Contractor #1	**0.53**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.54	No data	No data
	Toward Waianae Pumphead	ND	No data	No data

Table C10. TCP Concentrations in WL and TP Samples Collected at Waialua-Haleiwa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
3/8/2001	By Gate, Pumphead	0.61	No data	No data
	By Gate, Pumphead	0.55	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
4/20/2001	By Gate, Pumphead	0.43	No data	No data
	By Gate, Pumphead	ND	No data	No data
	Toward Waianae Pumphead	0.53	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
7/24/2001	By Gate, Pumphead	0.51	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.52**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.49	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
	By Gate, Pumphead	0.52	No data	No data
11/16/2001	By Gate, Pumphead	0.51	No data	No data
	By Gate, Pumphead	0.53	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.57**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.47	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
2/14/2002	By Gate, Pumphead	0.67	No data	No data
	By Gate, Pumphead	0.67	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.62**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.59	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
6/13/2002	By Gate, Pumphead	0.56	No data	No data
	By Gate, Pumphead	0.59	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
7/16/2002	By Gate, Pumphead	0.49	No data	No data
	By Gate, Pumphead	0.54	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.49	No data	No data
	Toward Waianae Pumphead	ND	No data	No data

Table C10. TCP Concentrations in WL and TP Samples Collected at Waialua-Haleiwa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
12/3/2002	By Gate, Pumphead	0.55	No data	No data
	By Gate, Pumphead	0.67	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.62**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.55	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
1/17/2003	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	0.54	No data	No data
	By Gate, Pumphead	0.61	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.53**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.55	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
4/22/2003	GAC Effluent Tap by Contractor #1	**0.47**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	0.52	No data	No data
5/20/2003	By Gate, Pumphead	0.53	No data	No data
	By Gate, Pumphead	ND	No data	No data
	Toward Waianae Pumphead	0.49	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
8/8/2003	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	0.52	No data	No data
	By Gate, Pumphead	0.48	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.48**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
11/18/2003	By Gate, Pumphead	0.53	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.56**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.51	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
2/24/2004	By Gate, Pumphead	0.57	No data	No data
	By Gate, Pumphead	0.51	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	Toward Waianae Pumphead	0.55	No data	No data
	Toward Waianae Pumphead	ND	No data	No data

Table C10. TCP Concentrations in WL and TP Samples Collected at Waialua-Haleiwa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
3/19/2004	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
5/11/2004	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
6/21/2004	By Gate, Pumphead	0.56	No data	No data
	GAC Effluent Tap by Contractor #1	**0.08**	No data	No data
	GAC Effluent Tap by Contractor #5	**0.08**	No data	No data
7/1/2004	By Gate, Pumphead	0.69	No data	No data
	GAC Effluent Tap by Contractor #1	**0.58**	No data	No data
	Toward Waianae Pumphead	0.56	No data	No data
7/8/2004	By Gate, Pumphead	0.64	No data	No data
	By Gate, Pumphead	0.62	No data	No data
	By Gate, Pumphead	ND	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.13**	No data	No data
	GAC Effluent Tap by Contractor #1	**0.69**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**0.12**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
11/4/2004	By Gate, Pumphead	0.61	No data	No data
	By Gate, Pumphead	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**0.58**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
12/15/2004	By Gate, Pumphead	0.62	No data	No data
	By Gate, Pumphead	ND	No data	No data
	Toward Waianae Pumphead	0.62	No data	No data
	Toward Waianae Pumphead	ND	No data	No data
2/24/2005	By Gate, Pumphead	0.49	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
4/12/2005	By Gate, Pumphead	0.63	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**0.49**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
8/15/2005	By Gate, Pumphead	0.61	No data	No data

Table C10. TCP Concentrations in WL and TP Samples Collected at Waialua-Haleiwa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
	GAC Effluent Tap by Contractor #1	**0.24**	No data	No data
	GAC Effluent Tap by Contractor #1	**0.55**	No data	No data
	GAC Effluent Tap by Contractor #5	**0.16**	No data	No data
11/21/2005	By Gate, Pumphead	0.28	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**0.24**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
2/14/2006	By Gate, Pumphead	0.62	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**0.52**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
5/18/2006	By Gate, Pumphead	0.57	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #1	**0.51**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
11/28/2006	By Gate, Pumphead	0.58	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
	Toward Waianae Pumphead	0.59	No data	No data
1/18/2007	By Gate, Pumphead	0.49	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
	Toward Waianae Pumphead	0.47	No data	No data
4/17/2007	By Gate, Pumphead	0.59	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
	Toward Waianae Pumphead	0.48	No data	No data
7/16/2007	By Gate, Pumphead	0.64	No data	No data
	GAC Effluent Tap by Contractor #1	**0.09**	No data	No data
	GAC Effluent Tap by Contractor #5	**0.07**	No data	No data
	Toward Waianae Pumphead	0.59	No data	No data
10/1/2007	GAC Effluent Tap by Contractor #1	**0.29**	No data	No data
	By Gate, Pumphead	0.66	No data	No data
	GAC Effluent Tap by Contractor #5	**0.24**	No data	No data
	Toward Waianae Pumphead	0.6	No data	No data
12/12/2007	BY GATE PUMPHEAD	0.77	No data	No data
	EAST GAC, BLUE TW LINE	ND	No data	No data
	Toward Waianae Pumphead	0.73	No data	No data
	WEST GAC, BLUE TW LINE	ND	No data	No data
1/31/2008	By Gate, Pumphead	0.62	No data	No data
	GAC Effluent Tap by Contractor #1	**0.49**	No data	No data
	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
2/21/2008	BY GATE PUMPHEAD	0.81	No data	No data
	EAST GAC, BLUE TW LINE	ND	No data	No data
	Toward Waianae Pumphead	0.77	No data	No data
	WEST GAC, BLUE TW LINE	ND	No data	No data
4/2/2008	BY GATE PUMPHEAD	0.82	No data	No data

Table C10. TCP Concentrations in WL and TP Samples Collected at Waialua-Haleiwa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
	EAST GAC, BLUE TW LINE	ND	No data	No data
	Toward Waianae Pumphead	0.7	No data	No data
	WEST GAC, BLUE TW LINE	ND	No data	No data
5/6/2008	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
7/31/2008	EAST GAC, BLUE TW LINE	ND	No data	No data
	Toward Waianae Pumphead	0.72	No data	No data
	WEST GAC, BLUE TW LINE	ND	No data	No data
8/1/2008	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
8/12/2008	BY GATE PUMPHEAD	0.77	No data	No data
11/5/2008	EAST GAC, BLUE TW LINE	0.05	No data	No data
	GAC Effluent Tap by Contractor #1	**0.1**	No data	No data
11/10/2008	GAC Effluent Tap by Contractor #1	**0.05**	No data	No data
	GAC Effluent Tap by Contractor #5	**0.07**	No data	No data
2/5/2009	EAST GAC, BLUE TW LINE	0.31	No data	No data
	GAC Effluent Tap by Contractor #1	**0.14**	No data	No data
	GAC Effluent Tap by Contractor #5	**0.16**	No data	No data
	WEST GAC, BLUE TW LINE	0.27	No data	No data
4/8/2009	WEST GAC, BLUE TW LINE	0.57	No data	No data
4/21/2009	EAST GAC, BLUE TW LINE	ND	No data	No data
	GAC Effluent Tap by Contractor #1	**0.29**	No data	No data
	GAC Effluent Tap by Contractor #5	**0.33**	No data	No data
	WEST GAC, BLUE TW LINE	ND	No data	No data
7/16/2009	EAST GAC, BLUE TW LINE	ND	No data	No data
	WEST GAC, BLUE TW LINE	ND	No data	No data
7/17/2009	GAC Effluent Tap by Contractor #1	**ND**	No data	No data
	GAC Effluent Tap by Contractor #5	**ND**	No data	No data
10/5/2009	No data	No data	HALEIWA GAC,UNDR MTLCVR NR TRFFC LIGHTBX	ND
2/11/2010	No data	No data	HALEIWA GAC,UNDR MTLCVR NR TRFFC LIGHTBX	ND
4/27/2010	No data	No data	HALEIWA GAC,UNDR MTLCVR NR TRFFC LIGHTBX	0.11

Wahiawa

TCP analysis results were reported for WL samples collected on 41 dates between 2/8/2000 and 7/17/2009. TCP was detected on all but four dates at concentrations up to 0.21 µg/L. TP samples were reported for 9 dates between 9/24/2009 and 4/6/2011. TCP was detected in five of these samples at concentrations up to 0.1 µg/L. No WL or TP samples collected at Wahiawa exceeded the current MCL for TCP.

A tabular presentation of these data are in Table C11 below.

Table C11. TCP Concentrations in WL and TP Samples Collected at Wahiawa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
2/8/2000	BASEYARD, PUMPHEAD	ND	No data	No data
5/12/2000	BASEYARD, PUMPHEAD	0.23	No data	No data
5/31/2000	BASEYARD, PUMPHEAD	ND	No data	No data
8/8/2000	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.15	No data	No data
10/20/2000	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.16	No data	No data
2/27/2001	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.16	No data	No data
4/20/2001	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.16	No data	No data
7/26/2001	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.14	No data	No data
11/16/2001	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.14	No data	No data
2/19/2002	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.11	No data	No data
6/19/2002	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.12	No data	No data
7/16/2002	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.13	No data	No data
12/4/2002	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.13	No data	No data
1/30/2003	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.14	No data	No data
5/2/2003	WELLHEAD, BASEYARD, PUMPHEAD	ND to 0.11	No data	No data
8/8/2003	WELLHEAD, BASEYARD, PUMPHEAD	ND	No data	No data
11/20/2003	BASEYARD, PUMPHEAD	ND	No data	No data
2/20/2004	BASEYARD, PUMPHEAD	0.12	No data	No data
6/29/2004	BASEYARD, PUMPHEAD	0.11	No data	No data
8/3/2004	BASEYARD, PUMPHEAD	0.12	No data	No data
11/4/2004	WELLHEAD, BASEYARD, PUMPHEAD	0.12 to 0.21	No data	No data
2/3/2005	WELLHEAD	0.15	No data	No data
5/24/2005	WELLHEAD	0.14	No data	No data
8/15/2005	WELLHEAD	0.15	No data	No data

Table C11. TCP Concentrations in WL and TP Samples Collected at Wahiawa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
5/18/2006	BASEYARD, PUMPHEAD	0.12	No data	No data
8/7/2006	BASEYARD, PUMPHEAD	0.14	No data	No data
10/27/2006	BASEYARD, PUMPHEAD	0.14	No data	No data
2/1/2007	BASEYARD, PUMPHEAD	0.13	No data	No data
4/17/2007	BASEYARD, PUMPHEAD	0.14	No data	No data
7/16/2007	WELLHEAD, BASEYARD, PUMPHEAD	0.11 to 0.16	No data	No data
10/6/2007	WELLHEAD	0.09	No data	No data
12/12/2007	BASEYARD, PUMPHEAD	0.17	No data	No data
1/31/2008	BASEYARD, PUMPHEAD	0.12	No data	No data
2/29/2008	WELLHEAD	0.1	No data	No data
4/3/2008	WELLHEAD, BASEYARD, PUMPHEAD	0.11 to 0.13	No data	No data
7/31/2008	BASEYARD, PUMPHEAD	0.13	No data	No data
8/1/2008	WELLHEAD	0.11	No data	No data
11/10/2008	WELLHEAD	0.1	No data	No data
2/17/2009	WELLHEAD	0.12	No data	No data
4/22/2009	WELLHEAD	0.11	No data	No data
7/17/2009	WELLHEAD	0.13	No data	No data
9/24/2009	No data	No data	WAHIWA II, MPSNK BTWN RR WHWA BWS BASEYD	ND
10/5/2009	No data	No data	WAHIAWA I CHLOR, MOP SINK IN CTRL BLDG	ND
10/6/2009	No data	No data	WAHIWA II, MPSNK BTWN RR WHWA BWS BASEYD	0.1
1/27/2010	No data	No data	WAHIAWA I CHLOR, MOP SINK IN CTRL BLDG	0.06
1/27/2010	No data	No data	WAHIWA II, MPSNK BTWN RR WHWA BWS BASEYD	0.09
4/27/2010	No data	No data	WAHIAWA I CHLOR, MOP SINK IN CTRL BLDG	0.08
4/27/2010	No data	No data	WAHIWA II, MPSNK BTWN RR WHWA BWS BASEYD	0.09
4/6/2011	No data	No data	WAHIAWA I CHLOR, MOP SINK IN CTRL BLDG	ND
4/6/2011	No data	No data	WAHIWA II, MPSNK BTWN RR WHWA BWS BASEYD	ND

Waipio Heights

WL and/or TP samples were reported for 112 dates between 2/28/2000 and 4/15/2010. During this time, WL samples were collected on 108 days and TP samples were collected on 28 days. TCP was detected in WL samples on 100 dates at concentrations up to 1.02 µg/L and in 11 TP samples up to 0.33 µg/L. There were 20 days on which both WL and TP samples were collected. On each of these dates except one, the concentration of TCP was lower in TP samples than in WL samples. On 4/21/2009, the WL samples contained 0.24 to 0.31 µg/L TCP, and the TP samples contained 0.25 to 0.33 µg/L TCP. All TCP levels in TP samples were below the current MCL.

A tabular presentation of these data are in Table C12 below.

Table C12. TCP Concentrations in WL and TP Samples Collected at Waipio Heights

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
2/28/2000	Waipio Heights Wells	ND to 0.6	No Data	No data
3/1/2000	Waipio Heights Wells	ND	No Data	No data
5/11/2000	Waipio Heights Wells	ND to 0.08	No Data	No data
5/25/2000	Waipio Heights Wells	ND to 0.71	No Data	No data
6/15/2000	Waipio Heights Wells	0.11	No Data	No data
7/6/2000	Waipio Heights Wells	0.27	No Data	No data
7/14/2000	Waipio Heights Wells	ND	No Data	No data
7/20/2000	Waipio Heights Wells	0.04 to 0.25	No Data	No data
8/8/2000	Waipio Heights Wells	ND to 0.61	No Data	No data
8/10/2000	Waipio Heights Wells	0.11	No Data	No data
10/11/2000	Waipio Heights Wells	0.26	No Data	No data
10/13/2000	Waipio Heights Wells	ND to 0.65	No Data	No data
10/19/2000	Waipio Heights Wells	0.25	No Data	No data
10/20/2000	Waipio Heights Wells	0.12 to 0.21	No Data	No data
2/20/2001	Waipio Heights Wells	0.37	No Data	No data
2/21/2001	Waipio Heights Wells	0.52	No Data	No data
2/22/2001	Waipio Heights Wells	ND to 0.65	No Data	No data
3/1/2001	Waipio Heights Wells	0.17	No Data	No data
4/20/2001	Waipio Heights Wells	0.17	No Data	No data
5/9/2001	Waipio Heights Wells	ND to 0.54	No Data	No data
7/10/2001	Waipio Heights Wells	0.16 to 0.18	No Data	No data
7/12/2001	Waipio Heights Wells	ND to 0.57	No Data	No data
7/16/2001	Waipio Heights Wells	ND	No Data	No data
7/20/2001	Waipio Heights Wells	ND to 0.24	No Data	No data
7/27/2001	Waipio Heights Wells	0.16 to 0.18	No Data	No data
7/30/2001	Waipio Heights Wells	ND to 0.28	No Data	No data
8/21/2001	Waipio Heights Wells	0.49	No Data	No data

Table C12. TCP Concentrations in WL and TP Samples Collected at Waipio Heights

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
10/23/2001	Waipio Heights Wells	ND to 0.25	No Data	No data
11/28/2001	Waipio Heights Wells	ND to 0.61	No Data	No data
12/5/2001	Waipio Heights Wells	0.16	No Data	No data
2/21/2002	Waipio Heights Wells	0.083 to 0.32	No Data	No data
2/26/2002	Waipio Heights Wells	ND to 0.08	No Data	No data
3/7/2002	Waipio Heights Wells	0.67	No Data	No data
6/18/2002	Waipio Heights Wells	ND to 0.58	No Data	No data
7/2/2002	Waipio Heights Wells	0.18 to 0.49	No Data	No data
7/15/2002	Waipio Heights Wells	ND	No Data	No data
7/17/2002	Waipio Heights Wells	0.06	No Data	No data
7/29/2002	Waipio Heights Wells	0.63	No Data	No data
10/18/2002	Waipio Heights Wells	0.09 to 0.32	No Data	No data
10/25/2002	Waipio Heights Wells	0.19 to 0.23	No Data	No data
10/31/2002	Waipio Heights Wells	ND to 0.58	No Data	No data
1/16/2003	Waipio Heights Wells	0.19 to 0.23	No Data	No data
2/20/2003	Waipio Heights Wells	ND to 0.34	No Data	No data
2/25/2003	Waipio Heights Wells	ND to 0.046	No Data	No data
4/30/2003	Waipio Heights Wells	ND to 0.45	No Data	No data
5/2/2003	Waipio Heights Wells	0.55	No Data	No data
5/28/2003	Waipio Heights Wells	0.21	No Data	No data
8/18/2003	Waipio Heights Wells	ND to 0.39	No Data	No data
8/21/2003	Waipio Heights Wells	ND to 0.55	No Data	No data
10/8/2003	Waipio Heights Wells	0.12 to 0.39	No Data	No data
10/21/2003	Waipio Heights Wells	ND to 0.51	No Data	No data
12/3/2003	Waipio Heights Wells	ND	No Data	No data
2/11/2004	Waipio Heights Wells	0.046 to 0.42	No Data	No data
2/20/2004	Waipio Heights Wells	ND to 0.52	No Data	No data
3/17/2004	Waipio Heights Wells	ND	No Data	No data
3/19/2004	Waipio Heights Wells	ND	No Data	No data
4/18/2004	Waipio Heights Wells	0.23	No Data	No data
4/28/2004	Waipio Heights Wells	0.44	No Data	No data
5/28/2004	Waipio Heights Wells	ND to 0.61	No Data	No data
7/9/2004	Waipio Heights Wells	0.19 to 0.88	GAC EFF	ND
8/10/2004	Waipio Heights Wells	ND to 0.48	No Data	No data
10/14/2004	Waipio Heights Wells	0.23 to 0.47	GAC EFF	ND
10/15/2004	Waipio Heights Wells	0.38	No data	No data
10/18/2004	Waipio Heights Wells	0.83	GAC EFF	ND
11/22/2004	Waipio Heights Wells	0.45	No data	No data
12/7/2004	Waipio Heights Wells	ND	No data	No data
12/16/2004	Waipio Heights Wells	ND	No data	No data
1/25/2005	Waipio Heights Wells	0.32	No data	No data
1/26/2005	Waipio Heights Wells	ND to 0.28	No data	No data
2/9/2005	Waipio Heights Wells	0.42	GAC EFF	ND
3/15/2009	No data	No data	GAC EFF	ND
4/8/2005	Waipio Heights Wells	0.45	GAC EFF	ND
5/19/2005	Waipio Heights Wells	0.34	No data	No data
5/23/2005	Waipio Heights Wells	0.26	No data	No data
7/12/2005	Waipio Heights Wells	0.35	No data	No data

Table C12. TCP Concentrations in WL and TP Samples Collected at Waipio Heights

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
7/15/2005	Waipio Heights Wells	0.27 to 0.49	GAC EFF	ND
11/14/2005	Waipio Heights Wells	ND to 0.12	GAC EFF	ND
11/16/2005	Waipio Heights Wells	0.13 to 0.49	GAC EFF	ND
2/3/2006	Waipio Heights Wells	0.26 to 0.29	No data	No data
2/6/2006	Waipio Heights Wells	0.37	No data	No data
2/9/2006	Waipio Heights Wells	0.48 to 0.98	GAC EFF	ND
5/3/2006	Waipio Heights Wells	0.46 to 1.02	GAC EFF	ND
5/15/2006	Waipio Heights Wells	0.35	No data	No data
5/25/2006	Waipio Heights Wells	0.28 to 0.29	No data	No data
8/10/2006	Waipio Heights Wells	0.24 to 0.36	No data	No data
8/11/2006	Waipio Heights Wells	0.25 to 0.46	GAC EFF	ND
8/22/2006	Waipio Heights Wells	0.44	No data	No data
10/27/2006	Waipio Heights Wells	0.29 to 0.77	GAC EFF	ND
1/19/2007	Waipio Heights Wells	0.29 to 0.38	No data	No data
2/13/2007	Waipio Heights Wells	0.39 to 0.44	GAC EFF	0.1
4/10/2007	Waipio Heights Wells	0.26 to 0.35	No data	No data
4/26/2007	Waipio Heights Wells	0.41 to 0.42	GAC EFF	0.24 to 0.28
7/13/2007	Waipio Heights Wells	0.27 to 0.45	GAC EFF	ND
10/3/2007	Waipio Heights Wells	0.27 to 0.38	GAC EFF	ND
11/27/2003	Waipio Heights Wells	0.26 to 0.34	No data	No data
1/31/2008	Waipio Heights Wells	0.26 to 0.36	GAC EFF	ND
3/3/2008	Waipio Heights Wells	0.28	No data	No data
4/2/2008	Waipio Heights Wells	0.27 to 0.28	GAC EFF	ND
5/20/2008	Waipio Heights Wells	0.33	No data	No data
7/14/2008	Waipio Heights Wells	0.29 to 0.38	No data	No data
7/29/2008	Waipio Heights Wells	0.25	GAC EFF	ND to 0.08
9/2/2008	Waipio Heights Wells	ND to 0.36	No data	No data
10/1/2008	Waipio Heights Wells	0.27 to 0.37	No data	No data
10/22/2008	No data	No data	GAC EFF	0.09 to 0.15
1/20/2009	No data	No data	GAC EFF	0.19 to 0.25
1/21/2009	Waipio Heights Wells	0.26 to 0.27	No data	No data
4/21/2009	Waipio Heights Wells	0.24 to 0.31	GAC EFF	0.25 to 0.33
7/6/2009	No data	No data	GAC EFF	0.04
8/27/2009	No data	No data	GAC EFF	0.26
10/6/2009	No data	No data	GAC EFF	0.24 to 0.25
2/17/2010	No data	No data	GAC EFF	0.22
4/15/2010	No data	No data	GAC EFF	0.23

Waipahu-Ewa-Waianae

WL and /or TP samples were collected and analysed on 259 dates between 2/7/2000 and 4/12/2011. Within this range, WL samples were collected on 240 days, and of those TCP was detected on 210 days at concentrations of up to 1.52 µg/L. TP samples were reported for 112 dates, and TCP was detected on 47 TP sample dates at concentrations up to 0.58 µg/L. in all WL and TP samples were below the current MCL.

A tabular presentation of these data are in Table C13 below.

Table C13. TCP Concentrations in WL and TP Samples Collected at Waipahu-Ewa-Waianae

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
2/7/2000	Wai[ahu Wells Kunia Wells	ND 0.9 to 1.2	Waipahu Wells, Hoaeae-Kunia Blend	ND ND
2/17/2000	Kunia Wells	1	No data	No data
3/2/2000	Kunia Wells	0.7	No data	No data
5/10/2000	Hoaeae Wells Waipahu Wells Kunia Wells	0.27 to 0.48 ND to 0.41 0.74	Waipahu Wells	ND to 0.15
5/11/2000	Kunia Wells	ND to 0.08	No data	No data
5/15/2000	Waipahu Wells	0.53	No data	No data
5/24/2000	Kunia Wells	ND to 0.77	No data	No data
5/30/2000	Waipahu Wells	ND	Waipahu Wells	ND
6/7/2000	Kunia Wells	ND	Hoaeaa-Kunia Blend	ND
6/13/2000	Kunia Wells	ND to 0.75	No data	No data
7/6/2000	Waipahu Wells	0.42 to 0.61	Waipahu Wells	0.13 to 0.36
7/7/2000	Hoaeae Wells Kunia Wells	ND to 0.54 ND to 0.83	No data	No data
7/13/2000	Kunia Wells Waipahu Wells	ND to 0.93 0.58	Hoaeae-Kunia Blend	0.34
7/20.2000	Hoaeae Wells Waipahu Wells	ND to 0.54 0.54	No data	No data
7/28/2000	Kunia Wells	ND to 0.18	Hoaeae-Kunia Blend Waipahu Wells	0.35 0.18 to 0.39
8/2/2000	Kunia Wells Waipahu Wells	ND to 0.87 ND	Waipahu Wells	ND
8/4/2000	Kunia Wells	0.81	No data	No data
8/7/2000	Kunia Wells	ND to 0.82	No data	No data
10/4/2000	Kunia Wells Waipahu Wells	ND to 0.94 0.59	Waipahu Wells	0.11 to 0.35
10/12/2000	Hoaeae Wells Waipahu Wells	ND to 0.52 0.59	Hoaeae-Kunia Blend	0.32
10/13/2000	Kunia Wells Waipahu Wells	0.86 ND to 0.42	Waipahu Wells	ND
10/19/2000	Kunia Wells Waipahu Wells	ND to 0.72 0.56	Waipahu Wells	0.17 to 0.47
2/21/2001	Hoaeae Wells	0.28 to 0.53	No data	No data
2/22/2002	Kunia Wells Waipahu Wells	ND to 1.07 ND to 0.38	Hoaeae-Kunia Blend Waipahu Wells	0.29 ND
3/1/2001	Kunia Wells	ND to 0.84	No data	No data
3/6/2001	Waipahu Wells	0.54 to 0.66	Waipahu Wells	ND

Table C13. TCP Concentrations in WL and TP Samples Collected at Waipahu-Ewa-Waianae

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
3/9/2001	Kunia Wells	ND	No data	No data
4/11/2001	Kunia Wells	ND to 0.96	Hoaeae-Kunia Blend	0.36
4/12/2001	Hoaeae Wells Waipahu Wells Kunia Wells	0.32 to 0.42 ND to 0.61 ND	Waipahu Wells	ND
4/18/2001	Waipahu Wells	0.64	No data	No data
4/23/2001	Hoaeae Wells Waipahu Wells Kunia Wells	0.39 to 0.48 ND to 0.41 ND to 0.93	Waipahu Wells	ND
7/10/2001	Hoaeae Wells Waipahu Wells	0.32 to 0.42 0.48 to 0.52	Waipahu Wells	ND to 0.05
7/11/2001	Hoaeae Wells Kunia Wells	0.41 to 0.42 ND to 0.89	Hoaeae-Kunia Blend	0.34
7/12/2001	Kunia Wells	ND to 1.14	No data	No data
7/16/2001	Waipahu Wells	ND to 0.48	Waipahu Wells	ND to 0.18
7/20/2001	Kunia Wells	ND to 1.02	No data	No data
7/23/2001	Kunia Wells	ND to 0.23	No data	No data
7/30/2001	Waipahu Wells	0.48	No data	No data
8/13/2001	Kunia Wells	0.72	No data	No data
8/20/2001	Kunia Wells	ND to 0.82	No data	No data
10/25/2001	Waipahu Wells	0.049 to 0.23	No data	No data
11/1/2001	Hoaeae Wells Waipahu Wells Kunia Wells	0.04 to 0.48 ND ND to 0.82	Hoaeae-Kunia Blend	0.45
11/2/2001	Kunia Wells	ND to 0.97	No data	No data
11/7/2001	Hoaeae Wells Waipahu Wells Kunia Wells	0.35 to 0.43 ND to 0.33 ND to 0.73	Waipahu Wells	ND to 0.47
11/9/2001	Kunia Wells	ND to 0.92	No data	No data
11/26/2001	Kunia Wells	ND	No data	No data
11/28/2001	Waipahu Wells Kunia Wells	ND ND	No data	No data
2/12/2002	Waipahu Wells Kunia Wells	ND to 0.67 ND to 1.17	Waipahu Wells	ND
2/14/2002	Kunia Wells	ND to 0.89	No data	No data
2/20/2002	Waipahu Wells Kunia Wells	0.65 ND to 1.02	Hoaeae-Kunia Blend	ND
2/21/2002	Hoaeae Wells Kunia Wells	0.33 to 0.44 ND	No data	No data
2/22/2002	Hoaeae Wells	ND to 0.41	No data	No data
2/26/2002	Kunia Wells	ND	No data	No data
5/21/2002	Hoaeae Wells Kunia Wells	0.34 to 0.52 ND	Hoaeae-Kunia Blend	ND
5/24/2002	Kunia Wells	ND to 1.16	No data	No data
5/31/2002	Waipahu Wells Kunia Wells	NF to 0.37 0.17	Waipahu Wells	ND
6/12/2002	Waipahu Wells	0.05 to 0.55	Waipahu Wells	ND
6/13/2002	Waipahu Wells Kunia Wells	0.49 0.18	No data	No data
6/21/2002	Kunia Wells	0.17	No data	No data
7/2/2002	Hoaeae Wells Waipahu Wells Kunia Wells	0.29 to 0.49 ND to 0.53 ND to 0.72	Waipahu Wells	ND
7/3/2002	Kunia Wells	ND to 0.85	Hoaeae-Kunia Blend	0.06

Table C13. TCP Concentrations in WL and TP Samples Collected at Waipahu-Ewa-Waianae

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
7/12/2002	Kunia Wells	ND	No data	No data
7/15/2002	Waipahu Wells	ND to 0.46	Waipahu Wells	ND
7/18/2002	Kunia Wells	ND to 1.14	No data	No data
7/19/2002	Kunia Wells	0.16 to 0.17	No data	No data
9/19/2002	Hoaeae Wells	ND	No data	No data
9/24/2002	No data	No data	Hoaeae-Kunia Blend	ND
10/15/2002	No data	No data	Waipahu Wells	ND
10/18/2002	Waipahu Wells Kunia Wells	ND to 0.43 ND to 1.11	Waipahu Wells	ND to 0.06
10/25/2002	Waipahu Wells	ND to 0.56	Waipahu Wells	ND to 0.08
10/31/2002	Hoaeae Wells Waipahu Wells Kunia Wells	0.42 0.53 to 0.57 ND to 1.23	Hoaeae-Kunia Blend	0.28
11/6/2002	Kunia Wells	ND to 0.29	No data	No data
11/14/2002	Hoaeae Wells	ND to 0.36	No data	No data
11/25/2002	Hoaeae Wells Kunia Wells	0.57 ND to 0.85	No data	No data
1/7/2003	Kunia Wells	ND	No data	No data
1/9/2003	Waipahu Wells	ND to 0.37	Waipahu Wells	ND to 0.29
1/10/2003	Hoaeae Wells Waipahu Wells Kunia Wells	ND to 0.47 0.05 to 0.65 ND to 0.85	Waipahu Wells	0.05 to 0.12
1/14/2003	Kunia Wells	ND to 0.47	No data	No data
1/24/2003	Hoaeae Wells Waipahu Wells Kunia Wells	0.32 to 0.34 ND to 0.047 ND to 1.08	Hoaeae-Kunia Blend	0.07
1/30/2003	Kunia Wells	0.78	No data	No data
2/7/2003	Hoaeae Wells	ND to 0.35	No data	No data
2/21/2003	Hoaeae Wells	ND	No data	No data
5/5/2003	Hoaeae Wells	0.31 to 0.49	No data	No data
5/9/2003	Hoaeae Wells Kunia Wells	0.39 ND to 0.76	Hoaeae-Kunia Blend	0.05
5/16/2003	Hoaeae Wells Kunia Wells	0.041 ND to 1.22	No data	No data
5/21/2003	Kunia Wells	ND	No data	No data
5/28/2003	Waipahu Wells	ND to 0.55	No data	No data
6/2/2003	Kunia Wells	ND to 0.18	Waipahu Wells	ND
8/4/2003	Waipahu Wells	ND to 0.38	Waipahu Wells	ND
8/5/2003	Waipahu Wells	0.49 to 0.53	Waipahu Wells	ND
8/6/2003	Hoaeae Wells	ND to 0.54	No data	No data
8/11/2003	Hoaeae Wells Kunia Wells	ND to 0.04 ND to 0.93	No data	No data
8/12/2003	Kunia Wells	ND	No data	No data
8/14/2003	Kunia Wells	ND to 0.71	No data	No data
8/19/2003	Kunia Wells	0.64	No data	No data
10/6/2003	Kunia Wells	ND to 0.91	No data	No data
10/7/2003	Kunia Wells	ND	No data	No data
10/8/2003	Hoaeae Wells Waipahu Wells	0.34 to 0.56	No data	No data
10/16/2003	Kunia Wells	ND to 0.18	No data	No data
10/20/2003	Hoaeae Wells	0.18	No data	No data
11/4/2003	Kunia Wells	0.18	No data	No data

Table C13. TCP Concentrations in WL and TP Samples Collected at Waipahu-Ewa-Waianae

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
11/5/2003	Waipahu Wells	0.51	No data	No data
11/10/2003	Waipahu Wells	0.32 to 0.42	Waipahu Wells	ND
11/21/2003	Waipahu Wells	ND	Waipahu Wells	ND
1/13/2004	Hoaeae Wells Kunia Wells	ND to 0.45 ND to 0.78	Hoaeae-Kunia Blend	0.35
1/15/2004	Waipahu Wells	ND to 0.51	Waipahu Wells	ND
1/25/2004	Waipahu Wells	0.44	No data	No data
2/2/2004	Kunia Wells	ND	No data	No data
2/6/2004	Waipahu Wells Kunia Wells	ND to 0.57 ND to 1.04	Waipahu Wells	ND
2/20/2004	Kunia Wells	ND	No data	No data
2/26/2004	Waipahu Wells	ND to 0.59	Waipahu Wells	ND to 0.12
3/2/2004	Hoaeae Wells Kunia Wells	ND to 0.42 ND to 0.04	No data	No data
3/16/2004	Kunia Wells	0.19	No data	No data
3/26/2004	No data	No data	Waipahu Wells	ND
4/28/2004	Waipahu Wells	ND to 0.65	Waipahu Wells	ND to 0.18
4/29/2004	Hoaeae Wells Kunia Wells	0.32 to 0.49 ND to 0.91	No data	No data
4/30/2004	Kunia Wells	0.21	Hoaeae-Kunia Blend	0.07
5/6/2004	Kunia Wells	ND to 0.27	No data	No data
5/14/2004	Waipahu Wells	ND	Waipahu Wells	ND
5/26/2004	Kunia Wells	ND	No data	No data
6/2/2004	Waipahu Wells Kunia Wells	0.46 0.83	Waipahu Wells	ND
6/21/2004	Waipahu Wells	0.37	Waipahu Wells	ND
6/29/2004	Kunia Wells	0.18	No data	No data
7/1/2004	Kunia Wells	1.41	No data	No data
7/9/2004	Waipahu Wells	ND to 0.64	Waipahu Wells	ND
7/19/2004	Waipahu Wells	ND to 0.52	Waipahu Wells	ND
7/22/2004	Waipahu Wells	0.09 to 0.49	No data	No data
7/27/2004	Hoaeae Wells Kunia Wells	ND to 0.51 ND to 0.62	Hoaeae-Kunia Blend	ND
7/28/2004	Kunia Wells	ND to 1.14	No data	No data
8/2/2004	Kunia Wells	ND to 0.19	No data	No data
8/10/2004	Kunia Wells	ND	No data	No data
10/15/2004	Waipahu Wells	ND to 0.76	Waipahu Wells	ND
10/29/2004	Hoaeae Wells Waipahu Wells	ND to 0.74	No data	No data
11/10/2004	Kunia Wells	ND to 1.22	No data	No data
11/29/2004	Hoaeae Wells Kunia Wells	ND 0.18 to 0.21	No data	No data
12/3/2004	Waipahu Wells	ND	Waipahu Wells	ND
12/14/2004	Kunia Wells	ND	No data	No data
12/16/2004	Kunia Wells	0.19	No data	No data
12/17/2004	No data	No data	Hoaeae-Kunia Blend	ND
1/25/2005	Waipahu Wells	ND to 0.66	Waipahu Wells	ND
1/28/2005	Kunia Wells	ND to 0.89	No data	No data
2/3/2005	Waipahu Wells	ND to 0.54	Waipahu Wells	ND to 0.07
2/6/2005	Hoaeae Wells Kunia Wells	0.47 to 0.54 0.21	No data	No data

Table C13. TCP Concentrations in WL and TP Samples Collected at Waipahu-Ewa-Waianae

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
2/7/2005	Hoaeae Wells Waipahu Wells Kunia Wells	ND to 0.41 ND to 0.65 0.17	Waipahu Wells	ND
2/9/2005	Kunia Wells	ND to 1.01	No data	No data
2/10/2005	Kunia Wells	1.25	No data	No data
2/23/2005	Kunia Wells	0.19	No data	No data
2/28/2005	Kunia Wells	ND	No data	No data
4/8/2005	Waipahu Wells	ND to 0.64	Waipahu Wells	ND to 0.24
4/14/2005	Hoaeae Wells Kunia Wells	ND to 0.61 ND to 0.94	No data	No data
5/5/2005	No data	No data	Waipahu Wells	0.16 to 0.24
5/16/2005	Kunia Wells	ND to 1.02	No data	No data
5/19/2005	Kunia Wells	ND to 0.21	No data	No data
5/23/2005	Kunia Wells	1.33	No data	No data
7/12/2005	Waipahu Wells Kunia Wells	ND to 0.43 ND to 0.22	Waipahu Wells	ND
7/15/2005	Waipahu Wells	ND to 0.67	No data	No data
8/5/2005	Waipahu Wells Kunia Wells	ND to 0.51 0.87 to 0.89	No data	No data
8/8/2005	Hoaeae Wells Kunia Wells	ND to 0.68 ND	Waipahu Wells	ND
8/18/2005	Kunia Wells	ND to 1.42	No data	No data
8/23/2005	Waipahu Wells	0.46	Waipahu Wells	ND
10/14/2005	Waipahu Wells Kunia Wells	0.35 to 0.54 0.12 to 0.21	Waipahu Wells	ND
10/27/2005	Hoaeae Wells Waipahu Wells Kunia Wells	ND to 0.32 0.25 ND to 0.48	Waipahu Wells	ND
11/1/2005	Hoaeae Wells	ND to 0.23	No data	No data
11/3/2005	Waipahu Wells	ND	Waipahu Wells	ND
11/4/2005	Kunia Wells	ND to 1.43		
11/17/2005	Kunia Wells	ND to 0.48	Waipahu Wells	ND
1/31/2006	Waipahu Wells	0.48 to 0.64	Waipahu Wells	ND to 0.08
2/3/2006	Hoaeae Wells Waipahu Wells Kunia Wells	0.37 to 0.45 0.56 ND to 1.21	No data	No data
2/6/2006	Hoaeae Wells Kunia Wells	0.29 to 0.49 ND to 1.33	No data	No data
2/8/2006	Waipahu Wells	0.73	Waipahu Wells	0.18
5/2/2006	Hoaeae Wells Waipahu Wells	0.04 to 0.49 0.44 to 0.76	Waipahu Wells	ND to 0.15
5/3/2006	Waipahu Wells	0.77	Waipahu Wells	0.21 to 0.25
5/15/2006	Waipahu Wells Kunia Wells	0.47 ND to 0.97	Waipahu Wells	0.14
5/16/2006	Kunia Wells	ND to 1.04	No data	No data
5/18/2006	Kunia Wells	1.31	No data	No data
6/14/2006	Kunia Wells	0.81	No data	No data
8/4/2006	Waipahu Wells	0.46 to 0.48	Waipahu Wells	ND
8/8/2006	Kunia Wells	1.01	No data	No data
8/10/2006	Kunia Wells	0.16 to 1.05	No data	No data
8/14/2006	Hoaeae Wells Waipahu Wells	0.41 to 0.64 0.72 to 0.8	Waipahu Wells	ND
10/19/2006	Waipahu Wells	0.53 to 0.94	Waipahu Wells	ND to 0.07

Table C13. TCP Concentrations in WL and TP Samples Collected at Waipahu-Ewa-Waianae

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
11/2/2006	Hoaeae Wells	ND to 0.46	No data	No data
11/13/2006	Hoaeae Wells Kunia Wells	0.39 to 0.48 0.17 to 1.05	No data	No data
11/14/2006	Kunia Wells	ND to 1.49	No data	No data
11/17/2006	Kunia Wells	0.57	Hoaeae Wells	ND
12/1/2006	Kunia Wells	ND	No data	No data
1/19/227	Kunia Wells	ND to 0.94	No data	No data
1/22/2007	Waipahu Wells Kunia Wells	0.47 to 0.9 0.88 to 1.52	Waipahu Wells	ND
1/26/2007	Hoaeae Wells Waipahu Wells	ND to 0.68 0.85	No data	No data
1/29/2007	Waipahu Wells	0.49	No data	No data
1/30/2007	Hoaeae Wells	0.49	No data	No data
2/2/2007	Kunia Wells	ND to 1.27	No data	No data
2/20/2007	Kunia Wells	0.19 to 0.22	No data	No data
2/23/2007	Waipahu Wells	0.43	No data	No data
3/9/2007	Waipahu Wells	0.78	No data	No data
4/5/2007	Waipahu Wells	0.33 to 0.73	Waipahu Wells	ND to 0.08
4/10/2007	Hoaeae Wells Waipahu Wells	0.06 to 0.62 0.54	No data	No data
4/12/2007	Kunia Wells	ND to 1.49	No data	No data
4/13/2007	Kunia Wells	ND to 1.43	No data	No data
4/23/2007	Waipahu Wells	0.33 to 0.41	Waipahu Wells	ND
5/7/2007	Waipahu Wells	0.44	No data	No data
7/2/2007	Waipahu Wells	0.29 to 0.42	Waipahu Wells	ND
7/3/2007	Waipahu Wells	0.44 to 0.45	No data	No data
7/5/2007	Hoaeae Wells Waipahu Wells Kunia Wells	0.09 to 0.64 0.64 to 0.84 0.07 to 1.01	Waipahu Wells	ND
7/6/2007	Kunia Wells	ND to 1.44	No data	No data
7/10/2007	Waipahu Wells Kunia Wells	ND to 0.72 ND to 1.11	Waipahu Wells	ND
7/11/2007	Waipahu Wells	0.44 to 0.68	Waipahu Wells	0.21 to 0.24
7/19/2007	Kunia Wells	0.22	Waipahu Wells	0.26 to 0.31
10/2/2007	Hoaeae Wells Kunia Wells	0.2 to 0.69 ND to 1.3	No data	No data
10/3/2007	Hoaeae Wells Waipahu Wells	0.5 0.27 to 0.4	Waipahu Wells	ND
10/4/2007	Waipahu Wells	ND to 0.72	Waipahu Wells	ND to 0.58
10/8/2007	Kunia Wells	1.12	No data	No data
10/10/2007	Waipahu Wells	0.86 to 0.93	Waipahu Wells	ND
10/11/2007	Waipahu Wells	0.68 to 1.04	No data	No data
10/17/2004	Waipahu Wells	0.72	No data	No data
11/29/2007	Kunia wells	0.22	No data	No data
1/28/2008	Waipahu Wells	ND to 0.72	Waipahu Wells	ND
1/30/2008	No data	No data	Waipahu Wells	ND
1/31/2008	No data	No data	Waipahu Wells	ND
2/11/2008	Hoaeae Wells Waipahu Wells	ND to 0.5 0.68 to 0.81	Waipahu Wells	ND
2/20/2008	Kunia Wells	ND to 1.39	No data	No data
2/25/2008	Waipahu Wells	0.38 to 0.84	No data	No data
3/24/2008	Hoaeae Wells	0.46	No data	No data

Table C13. TCP Concentrations in WL and TP Samples Collected at Waipahu-Ewa-Waianae

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
	Kunia Wells	0.21 to 0.23		
4/1/2008	Hoaeae Wells Waipahu Wells Kunia Wells	0.09 0.75 0.23	Waipahu Wells	ND
4/2/2008	Waipahu Wells	0.72	No data	No data
4/24/2008	Kunia Wells	0.4 to 0.46	No data	No data
5/5/2008	Waipahu Wells	0.48 to 0.96	No data	No data
5/9/2008	Hoaeae Wells Kunia Wells	0.74 0.05 to 1.18	No data	No data
5/22/2008	Hoaeae Wells Kunia Wells	0.38 0.22	No data	No data
7/14/2008	No data	No data	Waipahu Wells	ND
7/15/2008	Kunia Wells	ND	No data	No data
7/21/2008	Waipahu Wells	0.75	No data	No data
7/22/2008	Waipahu Wells	0.62 to 0.96	Waipahu Wells	ND to 0.11
7/29/2008	No data	No data	Waipahu Wells	0.17
7/31/2008	Hoaeae Wells	0.21	No data	No data
8/8/2008	Kunia Hills	1.02	No data	No data
9/10/2008	Kunia Wells	1.09	No data	No data
10/1/2008	Hoaeae Wells	0.32	Waipahu Wells	ND to 0.36
10/21/2008	Kunia Wells	ND	Waipahu Wells	0.51
1/12/2009	Kunia Wells	ND	No data	No data
1/21/2009	Waipahu Wells	0.48	Waipahu Wells	ND
1/23/2009	Kunia Wells	ND	Waipahu Wells	ND
2/11/2009	Hoaeae Wells	ND	No data	No data
4/1/2009	No data	No data	Waipahu Wells	ND
4/9/2009	Kunia Wells	ND	No data	No data
4/15/2009	Hoaeae Wells	0.11	No data	No data
4/21/2009	No data	No data	Waipahu Wells	ND
4/28/2009	Kunia Wells	0.08 to 0.11	No data	No data
7/6/2009	Hoaeae Wells	0.27	Waipahu Wells	ND to 0.09
7/7/2009	Kunia Wells	0.12 to 0.31	No data	No data
9/1/2009	No data	No data	Kunia Wells	0.24
10/6/2009	No data	No data	Waipahu Wells	ND to 0.14
10/7/2009	No data	No data	Kunia Wells	0.25 to 0.35
10/20/2009	No data	No data	Hoaeae Wells Waipahu Wells	0.07 0.08 to 0.23
2/17/2010	No data	No data	Hoaeae Wells Waipahu Wells Kunia Wells	0.18 ND 0.17 to 0.3
4/13/2010	No data	No data	Hoaeae Wells	0.21
4/21/2010	No data	No data	Waipahu Wells	0.11 to 0.4
4/26/2010	No data	No data	Kunia Wells	ND
4/12/2011	No data	No data	Waipahu Wells	ND

Mililani

WL and /or TP samples were collected and analysed on 91 dates between 4/11/2001 and 5/12/2011. WL samples were collected on 75 dates within this range (the latest WL samples was collected on 3/3/2008), and of these 66 had detectable TCP up to 4.02 ug/L. All WL samples with detectable TCP have concentrations above the MCL of 0.6 ug/L, and most WL samples had TCP concentrations above 2.0 ug/L.

TP samples were collected at Mililani on 70 dates in the range described above. TCP was detected on 37 dates. The TCP concentrations exceeded the current MCL on four dates: 1.15 ug/L on 11/22/2006; 0.62 ug/L on 4/16/2008; 0.7 ug/L on 4/21/2008; and 0.81 ug/L on 7/21/2008. The date of the highest TCP concentration in a TP sample (1.15 ug/L on 11/22/2006) corresponded with the highest TCP concentration in a WL sample at 4.02 ug/L on the same date. There were no corresponding WL sample data for the 2008 dates for comparison. Aside from these four incidents, the TCP levels in TP samples were kept below the current MCL.

A tabular presentation of these data are in Table C14 below.

Table C14. TCP Concentrations in WL and TP Samples Collected at Mililani

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
5/12/2000	Miliani Wells Pumpheads	ND	GAC Efferent Lines	0.35
5/15/2000	Miliani Wells Pumpheads	ND	No data	No data
6/8/2000	Miliani Wells Pumpheads	2.87	No data	No data
7/20/2000	Miliani Wells Pumpheads	ND	GAC Efferent Lines	ND
9/8/2000	Miliani Wells Pumpheads	ND	No data	No data
10/10/2000	Miliani Wells Pumpheads	ND	GAC Efferent Lines	ND
10/23/2000	Miliani Wells Pumpheads	3.29	No data	No data
2/21/2001	Miliani Wells Pumpheads	2.17 to 2.59	GAC Efferent Lines	0.32 to 0.35
3/27/2001	Miliani Wells Pumpheads	2.16	No data	No data
4/18/2001	Miliani Wells Pumpheads	3.57 to 2.44	GAC Efferent Lines	ND
4/20/2001	Miliani Wells Pumpheads	2.49	No data	No data
5/10/2001	Miliani Wells Pumpheads	1.55	No data	No data
7/25/2001	Miliani Wells Pumpheads	1.85 to 2.74	GAC Efferent Lines	0.04 to 0.06
7/26/2001	Miliani Wells Pumpheads	1.67	No data	No data
8/3/2001	Miliani Wells Pumpheads	2.29	No data	No data
10/25/2001	Miliani Wells Pumpheads	1.72 to 2.37	GAC Efferent Lines	ND
10/29/2001	Miliani Wells Pumpheads	2.42	GAC Efferent Lines	0.39 to 0.45
11/15/2001	Miliani Wells Pumpheads	0.37 to 2.44	No data	No data
1/16/2002	No data	No data	GAC Efferent Lines	ND
2/13/2002	Miliani Wells Pumpheads	1.57 to 2.03	GAC Efferent Lines	ND

Table C14. TCP Concentrations in WL and TP Samples Collected at Mililani

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
2/19/2002	Miliani Wells Pumpheads	2.21 to 2.29	GAC Efferent Lines	ND
6/13/2002	Miliani Wells Pumpheads	1.59 to 2.55	GAC Efferent Lines	ND
6/18/2002	Miliani Wells Pumpheads	2.03 to 3.97	No data	No data
6/27/2002	Miliani Wells Pumpheads	2.29	No data	No data
7/11/2002	Miliani Wells Pumpheads	2.22 to 2.76	No data	No data
7/17/2002	Miliani Wells Pumpheads	2.29 to 2.51	GAC Efferent Lines	0.06 to 0.11
7/18/2002	Miliani Wells Pumpheads	1.51 to 2.29	GAC Efferent Lines	ND to 0.07
11/6/2002	Miliani Wells Pumpheads	ND to 2.07	GAC Efferent Lines	ND
12/2/2002	Miliani Wells Pumpheads	ND to 2.75	GAC Efferent Lines	ND
1/16/2003	Miliani Wells Pumpheads	ND to 3.08	GAC Efferent Lines	ND
1/30/2003	Miliani Wells Pumpheads	ND to 3.25	No data	No data
2/3/2003	Miliani Wells Pumpheads	ND to 2.13	No data	No data
2/20/2003	Miliani Wells Pumpheads	ND to 2.42	GAC Efferent Lines	ND
5/27/2003	Miliani Wells Pumpheads	ND to 2.92	GAC Efferent Lines	ND to 0.17
6/3/2003	Miliani Wells Pumpheads	ND to 1.98	GAC Efferent Lines	ND
8/18/2003	Miliani Wells Pumpheads	ND to 2.07	GAC Efferent Lines	ND
8/19/2003	Miliani Wells Pumpheads	ND to 2.14	GAC Efferent Lines	ND
11/17/2003	Miliani Wells Pumpheads	ND to 3.61	GAC Efferent Lines	ND
11/20/2003	Miliani Wells Pumpheads	ND to 2.05	No data	No data
11/20/2003	Miliani Wells Pumpheads	ND to 1.21	GAC Efferent Lines	ND to 0.16
1/15/2004	Miliani Wells Pumpheads	ND to 2.57	GAC Efferent Lines	ND
2/26/2004	Miliani Wells Pumpheads	ND to 2.68	GAC Efferent Lines	ND to 0.06
3/5/2004	Miliani Wells Pumpheads	ND	GAC Efferent Lines	ND to 0.41
4/27/2004	Miliani Wells Pumpheads	ND to 2.29	GAC Efferent Lines	ND to 0.28
4/30/2004	Miliani Wells Pumpheads	ND to 3.34	GAC Efferent Lines	ND to 0.33
5/4/2004	Miliani Wells Pumpheads	ND to 0.73	GAC Efferent Lines	ND
6/2/2004	Miliani Wells Pumpheads	ND to 1.92	No data	No data
7/19/2004	Miliani Wells Pumpheads	ND to 1.96	GAC Efferent Lines	ND
7/29/2004	Miliani Wells Pumpheads	ND to 2.09	GAC Efferent Lines	ND
8/10/2004	Miliani Wells Pumpheads	ND	No data	No data
11/12/2004	Miliani Wells Pumpheads	ND to 2.89	GAC Efferent Lines	ND
12/2/2004	Miliani Wells Pumpheads	ND to 1.52	GAC Efferent Lines	ND
1/26/2005	Miliani Wells Pumpheads	0.87 to 2.35	GAC Efferent Lines	0.09
2/3/2005	Miliani Wells Pumpheads	2.13	GAC Efferent Lines	0.07
2/17/2005	Miliani Wells Pumpheads	1.68 to 2.87	GAC Efferent Lines	ND
5/6/2005	Miliani Wells Pumpheads	1.97 – 2.81	GAC Efferent Lines	ND
5/18/2005	Miliani Wells Pumpheads	1.64 to 2.56	GAC Efferent Lines	0.14 to 0.17
8/22/2005	Miliani Wells Pumpheads	ND to 3.38	GAC Efferent Lines	ND
8/24/2005	Miliani Wells Pumpheads	ND	GAC Efferent Lines	ND
11/3/2005	Miliani Wells Pumpheads	0.91 to 3.26	GAC Efferent Lines	ND
11/8/2005	Miliani Wells Pumpheads	ND to 3.37	GAC Efferent Lines	ND
2/9/2006	Miliani Wells Pumpheads	0.65 to 3.62	GAC Efferent Lines	ND to 0.25
6/1/2006	Miliani Wells Pumpheads	1.79 to 3.92	GAC Efferent Lines	ND to 0.23
8/23/2006	Miliani Wells Pumpheads	2.85 to 3.34	GAC Efferent Lines	ND
8/24/2006	Miliani Wells Pumpheads	1.71 to 3.12	GAC Efferent Lines	ND
11/17/2006	Miliani Wells Pumpheads	1.88	GAC Efferent Lines	ND
11/22/2006	Miliani Wells Pumpheads	2.51 to 4.02	GAC Efferent Lines	0.14 to 1.15
12/13/2006	Miliani Wells Pumpheads	2.48	No data	No data
2/1/2007	Miliani Wells Pumpheads	0.06 to 3.19	GAC Efferent Lines	ND to 0.34

Table C14. TCP Concentrations in WL and TP Samples Collected at Mililani

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. Range (µg/L)
2/26/2007	Miliani Wells Pumpheads	1.09	No data	No data
4/24/2007	Miliani Wells Pumpheads	0.84 to 3.0	GAC Efferent Lines	ND
4/27/2007	Miliani Wells Pumpheads	2.85	GAC Efferent Lines	ND
7/16/2007	Miliani Wells Pumpheads	2.28 to 2.92	GAC Efferent Lines	ND
10/4/2007	Miliani Wells Pumpheads	1.9 to 2.55	GAC Efferent Lines	0.06 to 0.12
1/30/2008	Miliani Wells Pumpheads	2.04 to 2.36	GAC Efferent Lines	0.17 to 0.46
3/3/2008	Miliani Wells Pumpheads	2.39	No data	No data
4/16/2008	No data	No data	GAC Efferent Lines	0.09 to 0.62
4/21/2008	No data	No data	GAC Efferent Lines	0.65 to 0.7
5/5/2008	No data	No data	GAC Efferent Lines	ND
7/14/2008	No data	No data	GAC Efferent Lines	ND
7/21/2008	No data	No data	GAC Efferent Lines	0.79 to 0.81
10/22/2008	No data	No data	GAC Efferent Lines	ND
10/20/2009	No data	No data	GAC Efferent Lines	ND
1/23/2009	No data	No data	GAC Efferent Lines	ND
4/7/2009	No data	No data	GAC Efferent Lines	ND
7/7/2009	No data	No data	GAC Efferent Lines	0.07 to 0.32
8/27/2009	No data	No data	GAC Efferent Lines	ND
10/6/2009	No data	No data	GAC Efferent Lines	ND
2/11/2010	No data	No data	GAC Efferent Lines	0.1 to 0.11
4/15/2010	No data	No data	GAC Efferent Lines	0.2 to 0.23
4/11/2011	No data	No data	GAC Efferent Lines	ND

Kauai

Both facilities reporting TCP concentrations in water on the island of Kauai are operated by the Kauai Department of Water.

Lihue-Kapaa

WL and /or TP samples were collected and analysed on 42 dates between 2/8/2001 and 9/28/2010. WL samples were collected on 18 dates within this range, and of these 14 had detectable TCP up to 0.08 ug/L. TP samples were collected on 35 dates, and of these 32 had detectable TCP up to 0.09 ug/L. All TCP concentrations in WL and TP samples were below the current MCL of 0.6 ug/L.

WL and TP samples were collected on the same day 11 times in the reported date range. On each of these dates the TCP concentration in the TP water was approximately the same as the WL water, suggesting little removal of TCP during treatment.

A tabular presentation of these data are in Table C15 below.

Table C15.TCP Concentrations in WL and TP Samples Collected at Lihue-Kapaa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
2/28/2001	No data	No data	BY GATE	0.09
5/2/2001	No data	No data	BY GATE	0.08
7/18/2001	No data	No data	BY GATE	ND
10/17/2001	No data	No data	BY GATE	0.08
7/23/2002	No data	No data	BY GATE	0.09
10/31/2002	Tap on well discharge line	ND to 0.08	BY GATE	0.08
1/30/2003	No data	No data	BY GATE	0.07
5/22/2003	No data	No data	BY GATE	0.07
8/21/2003	No data	No data	BY GATE	0.07
11/24/2003	No data	No data	BY GATE	0.08
1/27/2004	No data	No data	BY GATE	0.08
4/19/2004	No data	No data	BY GATE	0.08
7/22/2004	No data	No data	BY GATE	0.07
11/3/2004	No data	No data	BY GATE	0.08
3/1/2005	No data	No data	BY GATE	0.07
5/3/2005	No data	No data	BY GATE	0.08
8/23/2005	No data	No data	BY GATE	0.07
11/16/2005	No data	No data	BY GATE	0.06
3/7/2006	No data	No data	BY GATE	ND
6/1/2006	Hanamalulu 3 Wellhead	ND	No data	No data
6/21/2006	No data	No data	BY GATE	0.06
8/9/2006	Hanamalulu 3 Wellhead	ND	BY GATE	0.05
10/25/2006	Hanamalulu 3 Wellhead	ND	No data	No data
11/16/2006	No data	No data	BY GATE	0.05

Table C15.TCP Concentrations in WL and TP Samples Collected at Lihue-Kapaa

Date	WL Collection Location(s)	WL Conc. Range (µg/L)	TP Location	TP Conc. (µg/L)
1/24/2007	Hanamalulu 3 Wellhead	0.05	BY GATE	0.06
2/12/2007	Hanamalulu 3 Wellhead	ND	No data	No data
4/24/2007	Hanamalulu 3 Wellhead	0.05	BY GATE	0.05
7/31/2007	No data	No data	BY GATE	ND
8/1/2007	Hanamalulu 3 Wellhead	0.05	No data	No data
10/10/2007	Hanamalulu 3 Wellhead	0.06	BY GATE	0.06
3/18/2008	Hanamalulu 3 Wellhead	0.06	BY GATE	0.06
4/8/2008	Tap on well discharge line	0.06	BY GATE	0.05
8/6/2008	Hanamalulu 3, Hanamulu 4 Wellheads	0.05 to 0.06	BY GATE	0.05
10/27/2008	Hanamalulu 3, Hanamulu 4 Wellheads	0.05 to 0.06	BY GATE	0.06
2/23/2009	Hanamalulu 3, Hanamulu 4 Wellheads	0.05	BY GATE	0.06
6/16/2009	Hanamalulu 3, Hanamulu 4 Wellheads	ND to 0.05	No data	No data
6/30/2009	No data	No data	BY GATE	0.05
7/15/2009	No data	No data	BY GATE	0.07
12/1/2009	Hanamulu 4 Wellhead	0.07	No data	No data
6/21/2010	Hanamulu 4 Wellhead	0.06	No data	No data
9/28/2010	Hanamalulu 3, Hanamulu 4 Wellheads	0.05 to 0.07	BY GATE	0.05

Kalaheo-Koloa

WL and TP samples were collected and analysed on eight dates between 11/26/2002 and 2/23/2009. On all but two dates, there was no TCP detected in WL or TP samples. On 1/29/2004, TCP was detected at 0.04 µg/L in both the WL and TP samples. On 2/3/2009 TCP was measured at 0.04 µg/L in the WL sample and at 0.05 µg/L in the TP sample. Based on these data, the water treatment facility has not been challenged with TCP concentrations much higher than the detection limit ground water, and thus has been able to prevent excessive TCP from occurring in the effluent tap water.

A tabular presentation of these data are in Table C16 below.

Table C16. TCP Concentrations in WL and TP Samples Collected at Kaleheo-Kola

Date	WL Collection Location(s)	WL Conc. (µg/L)	TP Collection Location	TP Conc. (µg/L)
11/26/2002	Lawai Well 1, Sink Inside Central Bldg	ND	Lawai Well 1,Tap at Dump Sump	ND
1/29/2004	Lawai Well 1, Sink Inside Central Bldg	0.04	Lawai Well 1,Tap at Dump Sump	0.04
2/6/2004	Lawai Well 1, Sink Inside Central Bldg	ND	Lawai Well 1,Tap at Dump Sump	ND
8/16/2004	Lawai Well 1, Sink Inside Central Bldg	ND	Lawai Well 1,Tap at Dump Sump	ND
10/27/2005	Lawai Well 1, Sink Inside Central Bldg	ND	Lawai Well 1,Tap at Dump Sump	ND
3/22/2006	Lawai Well 1, Sink Inside Central Bldg	ND	Lawai Well 1,Tap at Dump Sump	ND
4/9/2008	Lawai Well 1, Sink Inside Central Bldg	ND	Lawai Well 1,Tap at Dump Sump	ND
2/3/2009	Lawai Well 1, Sink Inside Central Bldg	0.04	Lawai Well 1,Tap at Dump Sump	0.05