Science for Communities

HEALTH RISK ASSESSMENT: DISINFECTION BY-PRODUCTS IN SWIMMING POOL WATER

NOVEMBER 2023

PREPARED FOR:	Te Whatu Ora, National Public Health Service
CLIENT REPORT No:	FW23039
PREPARED BY:	Abhishek Gautam, Risk Assessment, Food and Social Systems Group
REVIEWED BY:	Peter Cressey, Risk Assessment, Food and Social Systems Group

Author	Management Reviewer	Peer Reviewer
		
Ahbishek Gautam	**Daniel Bohnen**	**Peter Cressey**
Senior Scientist, Risk Assessment and Social Systems Group	Manager, Risk Assessment and Social Systems Group	Science Leader, Risk Assessment and Social Systems Group

TABLE OF CONTENTS

ACRONYMS AND ABBREVIATIONS

ADD	Average daily dose
ADI	Acceptable daily intake
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark dose
bw	Body weight
CSF	Cancer slope factor
CDE	Chronic daily exposure
CDI	Chronic daily intake
DBP	Disinfection by-products
DL	Detection limit
ECHA	European Chemicals Agency
EFSA	European Food Safety Authority
ELCR	Excess lifetime carcinogenic risk
ESR	Institute of Environmental Science and Research Limited
EU	European Union
FRC	Free residual chlorine
GC-ECD	Gas chromatography-electron capture detection
GC-MS	Gas chromatography–mass spectrometry
HI	Hazard index
HQ	Hazard quotient
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
Kow	Partition coefficient

LADD	Lifetime average daily dose
LCR	Lifetime cancer risk
LD_{50}	Lethal dose (which causes death in 50% animals)
LOAEL	Lowest observed adverse effect level
MAV	Maximum acceptable value
MCLs	Maximum contaminant levels
MCLG	Maximum contaminant level goal
NOAEL	No observed adverse effect level
NOEL	No observed effect level
NTP	National Toxicology Program
NZ EPA	New Zealand's Environmental Protection Authority
POD	Point of departure
XRF	X-ray fluorescence
RfD	Reference dose
SGOT	Serum glutamic-oxaloacetic transaminase
SGPT	Serum glutamate pyruvate transaminase
SPW	Swimming pool water
SWIMODEL	Swimmer exposure model
USEPA	United States Environmental Protection Agency
WHO	World Health Organization

EXECUTIVE SUMMARY

The purpose of this report is to summarise generic health risk assessment data for incidental exposure to disinfection by-products in swimming pool water such as, trihalomethanes and haloacetic acids. This report will only consider domestic, non-occupational, routine, and incidental exposure to disinfection by-products in swimming pool water from recreational activities.

Disinfection of water is crucial to maintain hygienic conditions in swimming pools and spas. Chlorine and sometimes bromine products are used for water disinfection because of their low cost and effectiveness against many pathogens, especially bacteria and viruses. Chlorine or bromine in swimming pool water reacts with organic matter and residues of personal care products (shampoos, body lotion, sunscreen and other cosmetics) from swimmers, in addition to the natural organic matter from the source water, to form disinfection by-products. Trihalomethanes and haloacetic acids are the major swimming pool disinfection by-products, and their presence has been widely reported in both swimming pool water and drinking-water.

Systemic exposure to disinfection by-products can be by oral, dermal and inhalation routes. Local toxicological effects (skin irritation and rashes) can also occur. Trihalomethanes and haloacetic acids are the disinfection by-products with the most extensive toxicology data sets. Some trihalomethanes and haloacetic acids are carcinogenic, fetotoxic and mutagenic. The Swimmer Exposure Model, developed by the U.S. Environmental Protection Agency has been extensively used in exposure assessments of disinfection by-products in swimming pool water.

Several assessments of health risks (carcinogenic and non-carcinogenic) from disinfection by-products in swimming pool water have been reported in the scientific literature. Risks associated with disinfection by-product exposure have been characterised by estimation of hazard quotient and lifetime cancer risks. Reference doses and cancer slope factors derived by the U.S. Environmental Protection Agency were used in all published assessments.

For non-carcinogenic risks, hazard quotients were less than 1 for all disinfection by-products in most of the studies, indicating a low health risk for the general population. However, two studies reported potential health risks to disinfection by-products. In one study, the hazard quotient was >1 for adults and children following dermal exposure to disinfection by-products which indicated health risk concern due to non-carcinogenic effects. In the second study, the total hazard indexes for the five disinfection by-products following oral, dermal and inhalation exposure was >1 for all children and most adults. This was due to the detection of high levels of disinfection by-products in air and swimming pool water.

The studies summarised indicate that carcinogenic risks were mostly less than the estimated excess lifetime risk of 10^{-5} following the oral, and sometimes dermal, route of exposure to disinfection by-products in swimming pool water. However, the estimated risk was $>10^{-5}$ following inhalation and dermal exposure of trihalomethanes (chloroform) and haloacetic acids, respectively.

1 INTRODUCTION

The purpose of this report is to summarise health risk assessments available in public literature for incidental exposure to disinfectant by-products (DBPs) particularly trihalomethanes (THMs) and haloacetic acids (HAAs) in swimming pool water (SPW). This report will only consider domestic, non-occupational, routine, and incidental exposure to DBPs in SPW through recreational activities.

1.1 SWIMMING POOL WATER

In New Zealand, swimming pools can be indoor, outdoor, public or private. Swimming pool water (SPW) can be a source of pathogenic bacteria, fungi, viruses, cysts, and other microorganisms that can cause infectious diseases in humans.

The contamination of SPW can occur by anthropogenic inputs such as oral and nasal discharges, skin shedding, sweat, urine, hair, personal care products and occasionally faecal matter which can ultimately lead to health issues such as eye, ear, nose and throat ailments, enteric and urinary tract infections (StandardsNewZealand, 2010).

1.2 DISINFECTION OF WATER

Water disinfection means the removal, deactivation or killing of pathogenic microorganisms resulting in termination of growth and reproduction. Disinfection of SPW is crucial to maintain hygienic conditions in pools and spas.

In New Zealand, there are many methods used to disinfect SPW, such as chlorination, ozonation, treatment with copper-silver, UV irradiation, and UV/hydrogen peroxide treatment (StandardsNewZealand, 2010). Chlorine products are most commonly used for water disinfection because of their relatively low cost and effectiveness against many pathogens, especially bacteria and viruses (Genisoglu *et al.*, 2023). The types of chlorine generally used to disinfect SPW are sodium hypochlorite (liquid bleach), calcium hypochlorite, or chlorine gas and stabilised chlorine products (e.g., stabilised chlorine granules, chlorinated isocyanurates, chlorine tablets) (Chowdhury *et al.*, 2014; Ounsaneha *et al.*, 2017).

1.3 DISINFECTION BY-PRODUCTS

DBPs are generated when disinfectants react with organic matter and residues of personal care products (shampoos, body lotion, sunscreen and other cosmetics) from swimmers or natural organic matter from the source water. DBPs may be organic (e.g., chloroform) or inorganic (e.g., chlorate, chlorite, and bromate). However, concentrations of organic DBPs are generally higher than inorganic DBPs in chlorine-treated water (Srivastav and Kaur, 2020). DBPs were first reported in drinking-water (Rook, 1974), and subsequently a large number of studies have been carried out to investigate drinking water DBPs, including their occurrence and toxicity.

Trihalomethanes (THMs) and haloacetic acids (HAAs) are the predominant swimming pool DBPs and have been widely reported in studies of SPW and drinking-water. Other emerging DBPs are haloacetonitriles (HANs), haloketones (HKs), haloaldehydes (HALs), haloamides (HAMs), halonitromethanes (HNMs), and nitrosamines (NAs) (Genisoglu *et al.*, 2023; Peng *et al.*, 2020). A number of the DBPs in these categories are listed in Table 1.

Table 1: DBPs in SPW and drinking water

Category	DBPs
Trihalomethanes (THMs)	Tribromomethane (TBM)
	(DBCM)
	Trichloromethane (TCM) or Chloroform
Haloacetic acids (HAAs)	Monobromoacetic acid (MBAA)
	Monochloroacetic acid (MCAA)
	Dibromoacetic acid (DBAA)
	Dichloroacetic acid (DCAA)
	Bromochloroacetic acid (BCAA)
	Trichloroacetic acid (TCAA)
Haloacetonitriles (HANs)	Chloroacetonitrile (CAN)
	Bromoacetonitrile (BAN)
	Dichloroacetonitrile (DCAN)
	Bromochloroacetonitrile (BCAN)
Haloaldehydes (HALs)	Dichloroacetaldehyde (DCAL)
	Bromochloroacetaldehyde (BDCAL)
	Dibromoacetaldehyde (DBCAL)
	Trichloroacetaldehyde (TCAL) or chloral hydrate
	Bromodichloroacetaldehyde (BDCAL)
Halonitromethanes (HNMs)	Trichloronitromethane (TCNM)
Haloketones (HKs)	1,1-Dichloropropanone
	1,1,1-Trichloropropanone (1,1-TCP)

1.3.1 Factors influencing formation of DBPs

The formation of DBPs depend on a number of factors such as the pH and temperature of the SPW, and the presence of residues of personal care products, and free residual chlorine (FRC) levels or bromine levels (Srivastav and Kaur, 2020).

Organic matter also plays an important role in formation of DBPs. In general, an increase in organic matter in water leads to greater formation of DBPs.

Higher concentration of DBPs were reported in summer months compared to winter months. Hence, higher temperatures in summer may be responsible for the formation of DBPs. THMs and HAAs formation have a key temperature (Tc). When the temperature increases above the Tc, THM levels are reduced. This is likely due to their volatility (Ye *et al.*, 2009).

UV irradiation is also reported to increase the concentration of DBPs in water. In a study, sequential irradiation of river water with either low or medium pressure lamps and free chlorine (from disinfection) was simulated under practical conditions (Liu *et al.*, 2006). There was a statistically significant increase in the formation of chloroform, DCAA, and TCAA as compared to using chlorination only. Increases in levels of chloroform were the most significant.

It is well documented that pH influences the formation of distinct DBPs. For example, HKs decreased with a pH increase above 8. Conversely, the concentration of total THMs notably

rose when the pH increased from 6.0 to 8.5 (Ye *et al.*, 2009). Meanwhile, the content of total Haloacetic acids displayed a slower rate of change within the pH range of 6 to 8.5. Specifically, in the pH range of 6.5 to 7.7, there was a gradual increase in total Haloacetic acid content. However, within the pH range of 7.7-8.5, the concentration of total Haloacetic acid decreased (Ye *et al.*, 2009). Additionally, both THMs and HAAs concentrations exhibit an increase with declining FRC levels (Carter and Joll, 2017; Ye *et al.*, 2009)

Cosmetics can also promote the formation of DBPs in water (Carter and Joll, 2017). This is due to the chemicals (e.g., parabens in sunscreen) present in cosmetics. Trace amounts of chlorinated parabens were detected and quantified as DBPs in SPW (Carter and Joll, 2017; Terasaki and Makino, 2008). Other laboratory studies have also investigated the possible DBP formation from cosmetics under swimming pool conditions. More studies, focused on swimming pools, are required to understand the DBP formation from cosmetics and other personal care products.

1.4 DBPs in SPW

A number of studies have investigated the occurrence of several categories of DBPs, including THMs, HAAs, HANs, HKs and trichloronitromethane (TCNM), in SPW. Some of these studies are summarised below:

1. **Zhang *et al.,* 2023**

   A recent study carried out in Eastern China investigated the quantification of regulated (THMs, HAAs) and emerging (HALs, HANs) DBPs in indoor SPW (n=8) and the corresponding source of water (Zhang *et al.*, 2023). THMs, HANs and HALs were extracted and analysed using gas chromatography-electron capture detection (GC-ECD). HAA analysis was performed using gas chromatography-mass spectrometry (GC-MS) in electron ionisation mode. The limits of quantification (LOQ) and limits of detection (LOD) of DBPs were in the ranges 0.10-0.30 and 0.04-0.10 µg/L, respectively.

   The mean concentrations of THMs, HAAs, HANs, and HALs in the source water were 30.3, 20.0, 13.6 and 10.8 µg/L, respectively. The order of concentration of different DBPs was THMs>HANs>HAAs>HANs. TCM was the dominant THM species with a mean concentration of 19.2 µg/L, followed by BDCM (7.7 µg/L), DBCM (3.5 µg/L), and TBM (0.4 µg/L). Nine HAAs were detected in the source water. Seven HALs were detected in the source water. Among the 7 detected HANs, DCAN and TBAN were the dominant species with a mean concentration of 3.9 and 1.1 µg/L, respectively. Other HANs were detected in very low concentrations, accounting for less than 8% of the total concentration.

Total HALs were detected at the highest mean concentration in the SPW. The mean concentration of total THMs, HAAs, HANs, and HALs averaged 129.5, 181.6, 54.2 and 277.5 µg/L, respectively. This study does not specify how left censored data were managed in the summation process. The mean concentrations of DBPs in SPW and source water is presented in Table 2.

Table 2: Mean concentrations of DBPs in water

DBP	Mean concentration (µg/L)	
	Source water	SPW
THMs		
TCM	19	92
BDCM	8	20
DBCM	3	10
TBM	0.4	5
HAAs		
DCAA	6	<LOD
TCAA	4	78
BCAA	<LOD	27
BDCAA	<LOD	21
CDBAA	<LOD	23
HANs		
DCAN	4	32
BDCAN	<LOD	11
TBAN	1	<LOD
BCAN	<LOD	7
HALs		
DBAL	2	<LOD
TCAL	1	177
BCAL	<LOD	50
DBCAL	0.6	<LOD
TBAL	0.2	<LOD
DCAL	4	26
BDCAL	3	18

THMs: Trihalomethanes; TCM: Trichloromethane; BDCM: Bromodichloromethane; DBCM: Dibromochloromethane; TBM: Tribromomethane/bromoform; HAAs: Haloacetic acids; DCAA: Dichloroacetic acid; TCAA: Trichloroacetic acid; BCAA: bromochloroacetic acid; BDCAA: Bromodichloroacetic acid; CDBAA: Chlorodibromoacetic acid; HANs: Haloacetonitriles; DCAN: Dichloroacetonitrile; BDCAN: Bromodichloroacetonitrile; TBAN: Tribromoacetonitrile;; BCAN: Bromochloroacetonitrile; HALs: haloaldehydes; DBAL: dibromoacetaldehyde; TCAL: Trichloroacetaldehyde; BCAL: Bromochloroacetaldehyde; DBCAL: dibromochloroacetaldehyde; TBAL: Tribromoacetaldehyde; DCAL: Dichloroacetaldehyde; BDCAL: Bromodichloroacetaldehyde.

It is evident from the data presented in Table 2, that the concentration of DBPs in SPW is approximately 1-2 orders of magnitude higher than those detected in the source water. This is likely to be due to higher disinfectant levels and organic loading in the pool water.

2. Zhao *et al.*, 2020

The environmental occurrence and corresponding predicted human exposure of HAAs (CAA, BAA, DCAA, DBAA, and TCAA) was investigated from SPWs (n = 27) in Shanghai, China (Zhao *et al.*, 2020). The samples were analysed by gas chromatography-mass spectrometry (GC-MS).

The sum of the five HAAs in SPWs ranged from 62 to 407 µg/L, with the mean concentration of 241 µg/L. The concentrations of HAAs were higher than the maximum contaminant level (MCL) regulated by the U.S. Environmental Protection Agency (USEPA) in drinking-waters. DCAA and TCAA were the dominant HAAs in the SPWs, accounting for 27% and 57% of total HAAs, respectively. This is possibly because chlorination is the most commonly used disinfection method and increased chlorine

doses at higher dissolved oxygen concentrations (DOC) favour the formation of di- and tri- haloacetic acid over mono- acetic acids.

3. Sdougkou *et al.,* 2021

This study investigated the presence of various priority and emerging DBP groups (THMs, HAAs, HANs, HNMs, HKs) in a range of swimming pool types (n = 14, indoor, outdoor, only for children and for children/adults) located in the area of Thessaloniki, Northern Greece. GC-ECD was used to determine the levels of DBPs (Sdougkou *et al.*, 2021).

Total HAAs were detected in the highest concentrations followed by THMs, HANs, TCNM and HKs. The concentration range of different groups of DBPs is presented in Table 3.

Table 3: Concentration range for DBPs

DBPs	Concentration (Median; µg/L)
HAAs	178–3640 (680; sum of nine compounds)
THMs	1–410 (89; sum of four compounds)
HANs	0.9–130 (15; sum of four actetonitriles)

HAAs: haloacetic acids; THMs: Trihalomethanes; HANs: haloacetonitriles

TCAA and DCAA were the major individual HAAs detected, with concentration ranges of ~ 100-3000 and 30-50 µg/L, respectively. TCM was the dominant individual THM with a concentration range of 0.8-400 µg/L. DCAN (median 8.1 µg/L) and TCAN (median: 2.1 µg/L) were the dominant compounds in the HANs group. The concentration ranges for DCAN and TCAN were 0.5-80 and 0.9-7 µg/L, respectively. TCNM concentrations ranged from <0.2-7 µg/L (median 1.6 µg/L).

4. Shi *et al.,* 2020

THMs (TCM, TBM, BDCM and DBCM) and HAAs (MCAA, DCAA, TCAA, MBAA and DBAA) were quantified in SPWs from indoor swimming pools (n=16) in Shanghai, China across the four seasons (spring, summer, autumn and winter) (Shi *et al.*, 2020). The samples were extracted by liquid-liquid extraction and analysed using GC-ECD.

All DBPs were detected in all four seasons, except TBM, which was only detected in spring and winter. The concentration of chloroform was highest of the THMs and TCAA and DCAA the highest of the HAAs. The median concentrations of total THMs and total HAAs in this study were 54 and 364 µg/L.

The concentrations of DBPs detected in SPW at the highest concentrations are summarised in Table 4.

Table 4: Concentration of selected DBPs in indoor swimming pools, Shanghai, China

DBPs	Concentration (µg/L)
TCM	5-102
TCAA	7.6-930
DCAA	6.2-480

TCM: Trichloromethane; TCAA: Trichloroacetic acid; DCAA: Dichloroacetic acid
Limit of detection/limit of quantification: TCM: 1.6/5.3 ng/L; TCAA: 6.5/22 ng/L; DCAA: 2.9/10 ng/L

5. Hang *et al.,* 2016

The concentrations of DBPs (THMs, HALs, HANs, HKs and the HNM, TCNM) were determined in public indoor pools (n = 13) in Nanjing, China (Hang *et al.*, 2016). GC-ECD was used to determine the levels of DBPs.

HAAs were detected at the highest concentrations followed by THMs, HANs, HKs and TCNM. The mean concentrations of total HAAs, THMs, HANs, HKs and TCNM from 11 pools detected in SPWs are summarised in Table 5. BCAA, TCM, DCAN, and 1,1,1-TCP were the dominant species among HAAs, THMs, HANs, and HKs, respectively.

Table 5: Mean concentrations of DBPs in public swimming pools

DBPs	Concentration (µg/L)
HAAs	700-1500
THMs	190–380
HANs	100–210
HKs	10–60
TCNM	0–1

6. Peng *et al*, 2020

The occurrence of 29 DBPs was investigated in public swimming pools (16 indoor pools and 25 outdoor pools) from seven districts in Changsha, China (Peng *et al.*, 2020). The source water was also sampled for five of the swimming pools. The USEPA method 551.1 was used to determine the concentrations of four THMs, four HANs, two HKs, and TCNM. HAAs were measured based on modified USEPA method 552.3. Both the methods were followed by analysis using GC-ECD.

The levels of DBPs were higher in the SPWs than the associated source waters. The mean concentrations of the groups of DBPs detected in SPWs are summarised in Table 6.

Table 6: Mean concentrations of DBPs in public swimming pools

DBPs	Concentration (µg/L) ± SD
THMs	26 ± 33.1
HAAs	282.2 ± 394.3
HANs	12.3 ± 15.5
HKs	5.4 ± 5.1
NAs	0.101 ± 0.123
TCNM	1.0 ± 0.13

THMs: Trihalomethanes; HAAs: haloacetic acids; HANs: haloacetonitriles;HKs: Halo ketones; NAs: Nitrosamines; SD: Standard deviation

NAs were the group of DBPs detected at the lowest concentrations in SPWs. THMs and HAAs were the main species detected, and the concentrations of HAAs were the highest.

7. Anchal *et al.,* 2020

THMs were quantified in SPW samples from indoor pools (n=5) in Jharkhand, India. Quantitative analysis of samples (water and air) was performed using GC-ECD (Anchal *et al.*, 2020).

The concentration range of total THMs in SPWs was 163–226 µg/L, with an average concentration of 197 µg/L. Chloroform was the main THMs species detected. The mean concentrations detected are summarised in Table 7. In air, the maximum concentration of chloroform, DBCM and BDCM was 0.032, 0.00012, and 0.0033 µg m^{-3}, respectively.

Table 7: Mean concentrations of trihalomethanes in indoor swimming pools, Jharkhand, India

THMs	Concentration (µg/L) ± SD
Chloroform	191 ± 1.45
DBCM	3 ± 0.85
BDCM	3.2 ± 0.93
Total THM	197 ± 16.13

DBCM: Dibromochloromethane; BDCM: Bromodichloromethane; THM: Trihalomethane; SD: Standard deviation

8. Ounsaneha *et al.,* 2017

The concentrations of HAAs (MCAA, DCAA, TCAA, MBAA, and DBAA) were assessed in swimming pools (1 outdoor and 1 indoor) in Songkhla Province, Thailand (Ounsaneha *et al.*, 2017). The sampling of SPWs was done during summer and rainy seasons and samples were analysed by GC-ECD to determine the levels of HAAs.

The mean concentration of HAAs in the indoor and outdoor pools in the summer and rainy seasons were 151 and 74, and 163 and 101 µg/L, respectively, which was higher than the tap water (source water). The concentrations of HAAs were higher in the outdoor pools as compared to the indoor pools. The authors concluded that the average concentrations of HAA5 (MCAA, DCAA, TCAA, MBAA, and DBAA) in indoor and outdoor swimming pools were higher than US EPA drinking water quality standards.

9. Pándics *et al.,* 2018

In this study, SPW and air was sampled from indoor swimming pools (n = 19) in Budapest, Hungary to determine the concentrations of THMs (Pándics *et al.*, 2018). Air was sampled at a height of 0.4 and 1.5 m above the water level which corresponds to the breathing zone of the swimmers and the staff, respectively. Water was also sampled in parallel to air sampling. The water samples were analysed using GC-MS.

Chloroform was the most abundant THM detected, comprising more than 80% in pool water and air. THMs were detected in all samples of SPW and air. The concentrations of total THMs and chloroform in the air and water samples are summarised in Table 8.

Table 8: Concentration of THMs in SPW and air

THMs	**Concentration**	
Height (cm)	Mean	Median
Water		
NR	36.4 µg/L	31.0 µg/L
Air		
40	56.3 µg/m^3	40.6 µg/m^3
150	55 µg/m^3	44.6 µg/m^3
Chloroform		
Height (cm)	Mean	Median
Water		
NR	40 µg/L	29 µg/L
Air		
40	48.2 µg/m^3	40.6 µg/m^3
150	49 µg/m^3	35 µg/m^3

NR: not relevant for water samples

10. Pieters and Horn, 2020

The presence of THMs was investigated in public and semi-public swimming pools located in the North West province of South Africa (n = 3; 2 indoor and 1 outdoor). SPWs were analysed for THMs using GC-ECD (Pieters and Horn, 2020).

Four THMs were determined in the samples. TCM and BDCM were detected at the highest concentrations in the range of 0.5-32 µg/L and 0.5-36 µg/L, respectively. Other THMs detected were bromoform (0.5-8 µg/L) and dibromochloromethane (0.5-32 µg/L).

11. Abbasnia *et al*, 2018

Water samples from indoor swimming pools in Tehran, Iran were investigated for the presence of THMs. Samples were collected before and after swimming (Abbasnia *et al.*, 2019). The concentration of THMs were determined by GC-ECD.

The mean concentrations of DCBM, DBCM, bromoform and chloroform were 48, 0.52, 1 and 138 µg/L, respectively.

SUMMARY

The studies show that THMs and HAAs are the major DBPs found in SPW. Chloroform is the most dominant THM detected. Among the HAAs, DCAA and TCAA were detected in the highest concentrations. The concentrations of DBPs were always higher in the SPW as compared to the source water. This is because SPW may have a higher organic matter content than the source water. In general, the concentrations of DBPs varied from study to study. Hence, it is not possible to derive typical concentrations for different DBPs.

1.5 QUALITY STANDARDS FOR DBPs IN SPW

There are only a few countries in the world (mostly in Europe) that have established SPW quality standards for DBPs. Due to the carcinogenic nature of THMs, regulations and guidelines has been recommended by several agencies like the World Health Organization (WHO) and USEPA for their control in drinking-water supplies. However, no guideline exists for DBPs (THMs and HAAs) in SPW. Some studies have reported that pool water is more cytotoxic, mutagenic and genotoxic than tap water (Honer *et al.*, 1980; Liviac *et al.*, 2010; Plewa *et al.*, 2011).

In New Zealand, the quality of SPW should be maintained as per the pool water standard prepared by the Standards New Zealand Technical Committee P 5826 Pool Water Quality (StandardsNewZealand, 2010). The standard ensures that the chemical and microbiological levels are maintained to safeguard health. The standard is applicable to all treated private/public freshwater and seawater swimming pools, spa pools, and geothermal pools.

Two categories of DBPs (THMs and HAAs) are regulated with concentration limits in drinking-water. The emerging DBPs are not regulated and data on their occurrence and safety are limited.

1.5.1 Europe

There are some countries in Europe where DBPs (THMs) are regulated in SPW (ECHA, 2017; Yang *et al.*, 2018).

Table 9: Maximum contaminant levels for THMs in swimming pool water

Country	**MCL (mg/L)**
Germany	0.02 (chloroform equivalents)
Switzerland	0.03
Netherlands	0.05 ((chloroform equivalents))
Denmark	Under 0.025 or 0.05 depending on the pool types
France	mandatory value: 0.1 guide value: 0.02
Belgium	0.1 (chloroform)
UK	0.1
Finland	0.1

1.5.2 World Health Organization (WHO)

WHO has published guidelines for safe recreational water environments which suggests that guideline values in the WHO Guidelines for Drinking-water Quality can be used to screen for potential risks arising from DBPs from swimming pools and similar environments (WHO, 2006). It is reported that the concentrations of DBPs in SPW and similar environments can exceed the WHO drinking water guideline values of water (WHO, 2006). There are some studies which report that the concentrations of DBPs in SPW is much higher than in the drinking-water (Richardson *et al.*, 2007).

Table 10: WHO Guideline value for DBPs in drinking water (WHO, 2017)

DBPs	Guideline value (mg/L)
Bromodichloromethane	0.06*
Bromoform	0.1
Chloroform	0.3
Dibromoacetonitrile	0.07
Dibromochloromethane	0.1
Dichloroacetate	0.05* (A)
Dichloroacetonitrile	0.02 (B)
Monochloroacetate	0.02
N-Nitrosodimethylamine	0.0001
Trichloroacetate	0.2
Trihalomethanes	The sum of the ratio of the concentration of each to its respective guideline value should not exceed 1

* For substances that are considered to be carcinogenic, the guideline value is the concentration in drinking-water associated with an upper-bound excess lifetime cancer risk of 10^{-5}. Concentrations associated with estimated upper-bound excess lifetime cancer risks of 10^{-4} and 10^{-6} can be calculated by multiplying and dividing, respectively, the guideline value by 10.

A, Provisional guideline value because disinfection is likely to result in the guideline value being exceeded.

B, Provisional guideline value because of uncertainties in the health database.

1.5.3 New Zealand/ Aotearoa

There is no regulation or standards for DBPs in SPW in New Zealand. Taumata Arowai is the water services regulator for New Zealand who has the responsibility for drinking water standards. Drinking Water Standards (Standards) set the maximum acceptable values (MAVs) for a range of contaminants which can affect the safety and quality of drinking water (DrinkingWaterStandards, 2022). They are based on guideline values set by WHO. The MAVs for THMs and HAAs in drinking-water are presented in Table 11.

Table 11: New Zealand MAVs for some DBPs in drinking water

DBPs	MAV (mg/L)
BDCM	0.06
Bromoform	0.1
Carbon tetrachloride	0.005
Chloroform	0.4
DBAN	0.08
DBCM	0.15
1,2-dibromoethane	0.0004
DCAA	0.05
DCAN	0.02
DCM	0.02
MCAA	0.02
TCAA	0.2
THMs*	Σ ratio < 1

MAVs: maximum acceptable values; DBPs: disinfectant by-products; BDCM: Bromodichloromethane; DBAN: Dibromoacetonitrile; DCAA: Dichloroacetic acid; DCAN: Dichloroacetonitrile; MCAA: Monochloroacetic acid; TCAA: Trichloroacetic acid; DCM: Dichloromethane

*The sum of the ratio of the concentration of each THM to its respective MAV must not exceed 1.

1.5.4 United States of America (USA)

There are no regulations or standards for DBPs in SPW in the US. However, there are standards for DBPs in drinking-water. The USEPA has developed the disinfection by-products rules (DBPRs) to limit exposure to DBPs in drinking-water (USEPA, 2006). The DBPRs (stage 1 and stage 2) require public water systems (PWSs) to:

► Comply with established maximum contaminant levels (MCLs) and operational evaluation levels (OELs) for DBPs, and maximum residual disinfection levels (MRDLs) for disinfectant residuals
► Conduct an initial evaluation of their distribution system.

The US EPA sets a maximum contaminant level goal (MCLG) after reviewing data on health effects (USEPA, 2006). The MCLG is the maximum level of a contaminant in drinking-water at which no known or anticipated adverse effect on the health of persons would occur, allowing an adequate margin of safety. After a MCLG is determined, the USEPA sets an enforceable standard which generally becomes an MCL. The MCL is the maximum level allowed of a contaminant in water which is delivered to any user of a PWS.

In addition, PWSs using conventional filtration are required to remove specific percentages of organic material that may react to form DBPs through the implementation of a treatment technique. The MCLs for THMs and HAAs are summarised in Table 12.

Table 12: Maximum contaminant levels (MCL) for DBPs in drinking water

DBPs	MCLG (mg/L)	MCL (mg/L)
Total THMs	-	0.08
Chloroform	0.07	-
BDCM	0	-
DBCM	0.06	-
Bromoform	0	-
HAAs	-	0.06
MCAA	0.07	-
DCAA	0	-
TCAA	0.02	-

MCAA: Monochloroacetic acid: DCAA: Dichloroacetic acid; TCAA: Trichloroacetic acid; BDCM: Bromodichloromethane; DBCM: Dibromochloromethane; MCLG: Maximum Contaminant Level Goal

1.5.5 China

In China, THMs are regulated in drinking water where the sum of the ratio of the concentration of various compounds (chloroform, chlorodibromomethane, bromodichloromethane, and methyl bromide) to their own limits should not exceed 1 (NationalStandardChina, 2006).

1.6 HUMAN HEALTH HAZARD CLASSIFICATIONS OF DBPs

Hazard classification for some of the DBPs are summarised in Table 13.

Table 13: Human health hazard classification of DBPs

DBP (CAS RN)	Hazard classification		
	EU*	USEPA	NZEPA**
Chloroform (67-66-3)	Harmful if swallowed, Toxic if inhaled, Causes skin and serious eye irritation, Suspected of causing cancer, Causes organ damage, Suspected of damaging unborn child.	Likely to be carcinogenic to humans by all routes of exposure under high-exposure conditions	Harmful if swallowed, Toxic if inhaled, Causes skin and serious eye irritation, Suspected of causing cancer, Causes organ damage, Suspected of damaging unborn child.
Bromoform (75-25-2)	Harmful if swallowed, Toxic if inhaled, Causes skin irritation, Causes eye irritation,	Probable human carcinogen, group B2	Harmful if swallowed, Toxic if inhaled, Causes skin irritation, Causes eye irritation,
Bromochloroacetic acid (CAS RN 5589-96-8)	Causes severe skin burns and eye damage	Potential human carcinogen	NA
Chloroacetic acid (CAS RN 79-11-8)	Toxic if inhaled, Toxic in contact with skin, Causes severe skin burns and eye damage.	NA	Fatal if inhaled, Causes severe skin burns and eye damage, Causes serious eye damage.
Dichloroacetic acid (CAS RN 79-43-6)	Causes severe skin burns and eye damage	Anticipated to be a human carcinogen***	Causes severe skin burns and eye damage
Trichloroacetic acid (CAS RN 76-03-9)	Causes severe skin burns and eye damage	Suggestive evidence of carcinogenic potential	Harmful if swallowed, Causes severe skin burns and eye damage,
Chloroacetonitrile (CAS RN 107-14-2)	Toxic if swallowed and inhaled, Toxic in contact with skin.	-	Fatal if swallowed and inhaled, Fatal in contact with skin.

NA: not available

* ECHA

** Chemical Classification and Information Database (CCID)

*** Classification by NTP

2 HAZARD IDENTIFICATION

2.1 PREVIOUS ASSESSMENTS

No previous health impact assessments for DBPs in SPW were found for New Zealand.

2.2 HEALTH EFFECTS – DBPs

2.3 TOXICITY OF DBPs

The toxicity of DBPs present in swimming pool water is not well explored. However, DBPs toxicity have been actively studied in drinking water. THMs and HAAs are the major DBPs found in SPWs in the studies summarised in section 1.4. Some DBPs can be carcinogenic, reprotoxic and mutagenic (Zhao *et al.*, 2020). It is also reported that emerging DBPs, i.e., HAN, HKs, and NAs, are more cytotoxic and genotoxic than THMs and HAAs, and have become emerging concerns (Font-Ribera *et al.*, 2019; Muellner *et al.*, 2007). The following sections summarise toxicity studies on some DPBs found in the literature.

2.3.1 Trihalomethanes (THMs)

THMs are one of the dominating DBPs found in SPWs and drinking water. THMs in swimming pools were first reported by Beech et al. (1980). Since most THMs are volatile in nature, inhalation is the main exposure pathway for THMs.

In mammals, THMs are well absorbed and distributed throughout the body, with high levels found in fat, blood, liver, kidneys, lungs and the nervous system. THMs are metabolised primarily to carbon dioxide and/or carbon monoxide. They are rapidly excreted after oral or inhalation exposure (Florentin *et al.*, 2011).

Chloroform

Chloroform was once used as an anaesthetic in humans but its use was discontinued due to its toxicity (ATSDR, 1997). When administered as an anaesthetic for a prolonged time, it may lead to profound toxaemia and damage to the liver, heart and kidneys. Inhalation of concentrated chloroform vapour causes irritation of exposed mucous surfaces. Narcosis is ordinarily preceded by a stage of excitation which is followed by loss of reflexes, sensation and consciousness (ATSDR, 1997; INCHEM, 1991).

In sub-chronic oral or inhalation toxicity studies in animals, liver and renal toxicity, as well as lesions of the nasal epithelium were observed. In many long-term toxicity studies in animals, chloroform has been reported to cause significant increases in the incidence of liver tumours in male and female mice and significant increases in the incidence of kidney tumours in male rats and mice by the oral and inhalation routes (IARC, 1999b; INCHEM, 1991). Based on these tumours in animals, chloroform is classified as Group B2, probable human carcinogen by the US EPA, and possibly carcinogenic to humans (Group 2B) by IARC, based on "sufficient evidence" of carcinogenicity in animals (IARC, 1999b; IRIS, 2001). In the EU, the European chemical agency (ECHA) has classified chloroform as “suspected of causing cancer in humans” based on cancer effects in animal studies. Tumours are produced only at doses that result in cytotoxicity. Carcinogenic effects in the liver and lungs of animals are attributed to the oxidative metabolite phosgene ($COCl_2$) which is electrophilic and causes cellular toxicity by reaction with tissue proteins and cellular macromolecules, as well as glutathione, free

cysteine, histidine, methionine and tyrosine. The persistent cytotoxicity of phosgene induces regenerative cell proliferation that leads to spontaneous cell mutation and subsequent cancer (ATSDR, 1997; IRIS, 2001).

Chloroform was found to be non-mutagenic in a number of tests in *Salmonella Typhimurium* (Ames test) and *Escherichia coli* (with and without activation), in gene mutation tests in Chinese hamster ovary (CHO) cells and human lymphocytes, in mouse micronucleus tests, and in tests of unscheduled DNA synthesis both *in vitro* and *in vivo* (ATSDR, 1997).

Chloroform was found to be fetotoxic in a number of reproductive and developmental toxicity studies (ATSDR, 1997; IRIS, 2001). However, the effects observed on reproduction and foetus development are most probably due to a toxic effect on the dams, as females appeared more sensitive to the toxic effects than males in these reproductive studies. Chloroform is classified as “suspected of damaging the unborn child” by ECHA.

Bromoform and dibromochloromethane

Data on oral and inhalation absorption of bromoform and dibromochloromethane are very limited. It is expected that the absorption profile of bromoform would be similar to that of chloroform.

Bromoform was previously used as a sedative in children suffering from whooping cough. There have been several deaths reported due to accidental overdoses. Severe central nervous system depression (unconsciousness, stupor, and loss of reflexes) was reported in fatal cases and death was generally due to respiratory failure. The target organs of bromoform and dibromochloromethane toxicity are the liver, kidney, and central nervous system (ATDSR, 2005).

There is strong evidence that reactive metabolic intermediates and the highly reactive trihalomethyl free radical are responsible for hepatotoxicity of bromoform and dibromochloromethane. Hepatotoxicity is characterised by infiltration, cellular vacuolization and swelling and increases in liver weight at low doses. At higher doses, focal centrilobular necrosis and increases in serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamate pyruvate transaminase (SGPT) levels have been observed. Rats are more sensitive than mice to liver effects of these compounds.

The carcinogenicity of bromoform and dibromochloromethane has been studied in both humans and laboratory animals. The data in humans are inconclusive and are insufficient to establish causal relationships (ATDSR, 2005; IARC, 1999a). Chronic oral exposure to bromoform resulted in increases in the occurrence of rare intestinal tumours (adenomatous polyps and adenocarcinoma) in female rats receiving 200 mg/kg bw/d, 5 days/week for 2 years (ATDSR, 2005; IRIS, 1990; NTP, 1989). The combined tumour incidence was 0/50, 0/50, and 3/50 for males administered 0, 100, or 200 mg/kg and 0/50, 1/50, and 8/50 for females. The IARC classified bromoform as Group 3 not classifiable due to its carcinogenicity to humans due limited evidence of carcinogenicity in experimental animals (IARC, 1999a). In mice, bromoform did not increase the proportion of mice with tumours.

There are mixed results from genotoxicity testing (ATDSR, 2005). Bromoform was mutagenic in Ames test with the *S. typhimurium* strain TA100 without metabolic activation. No studies with other strains were reported. Bromoform also induced DNA damage in *Escherichia coli* with or without metabolic activation. Bromoform is volatile and none of these studies reported

whether the test system used was a closed system that would minimise the loss of the test substance. With mammalian cells, the results were either negative or equivocal.

In rats, bromoform increased chromosomal aberrations in bone marrow cells after intraperitoneal injection and by oral administration but this was not seen in mice after intraperitoneal administration (ATDSR, 2005). Results in mice were inconsistent in bone marrow micronucleus assays. In a micronucleus test in mice, oral administration of bromoform by intragastric gavage did not cause any significant increases in the incidence of micronucleated polychromatic erythrocytes in males and females at any dose level (Stocker *et al.*, 1997). The same response was also seen after intraperitoneal administration (ATDSR, 2005; Hayashi *et al.*, 1988). A significant increase in sister chromatid exchange was observed in the bone marrow cells of mice after receiving bromoform by gavage and intraperitoneal doses (ATDSR, 2005). No conclusion has been reached due to conflicting results on the mutagenicity of bromoform.

Dibromochloromethane induced liver tumours (hepatocellular adenomas and carcinomas) in male and female mice (ATDSR, 2005). The incidence was significantly elevated for hepatocellular adenomas in females (2/50, 4/49, and 11/50 for 0, 50, and 100 mg/kg), hepatocellular carcinoma in males (10/50 and 19/50 for 0 and 100 mg/kg) and combined incidences for hepatocellular adenoma or carcinoma (23/50 and 27/50 for males at 0 and 100 mg/kg and 6/50, 10/49, and 19/50 for females at 0, 50, and 100 mg/kg). USEPA classified bromoform as a “probable human carcinogen, group B2” and dibromochloromethane as a “possible human carcinogen, group C” (IRIS, 1990; 1992).

2.3.2 Haloacetic acids (HAAs)

HAAs are another class of DBPs which are abundantly detected on SPWs and drinking water. DCAA and TCAA are the most abundant HAAs detected in SPW. Brominated acetic acids (including DBAA) occur at the highest concentrations in chlorinated seawater swimming pools.

HAAs are non-volatile and are also not appreciably absorbed through the skin (Xu *et al.*, 2002). Ingestion of SPW is the main route of exposure to HAAs as they have been detected in the urine of swimming pool attendants and swimmers (NTP, 2021a).

HAAs are rapidly absorbed from the gastrointestinal tract and are found in the blood and tissues at approximately equal concentrations. Dihalo-and trihalo acetic acids are metabolised to simpler analogues which ultimately results in the formation of glyoxylate, glycolate, oxylate, glycine, and carbon dioxide (Florentin *et al.*, 2011).

Monochloroacetic acid (MCAA)

MCAA is readily absorbed after ingestion and through skin. It accumulates in the liver and kidneys, followed by accumulation in the brain. MCAA undergoes dehalogenation to form oxalate and glycine and/or dehalogenation and reduction to thiodiacetic acid via glutathione conjugation. It also undergoes enzymatic hydrolysis of the chlorine-carbon bond and forms glycolic acid that can be degraded completely to carbon dioxide (INCHEM, 1998).

Acute exposure to MCAA through inhalation and the dermal route may cause severe damage to the skin and mucous membranes in humans. It is also a skin, eye and respiratory irritant (INCHEM, 1998).

In rodents, chronic exposure through inhalation, and dermal and oral routes caused damage to the respiratory tract, including inflammatory changes in the respiratory organs, inflammatory lesions of the nasal mucosa, metaplasia of the olfactory epithelium, and respiratory congestion (INCHEM, 1998; WHO, 2005a). Myocarditis and death due to myocardial failure in rats and hepatic vacuolar degeneration has been observed in mice after gavage.

Of the available literature, no adverse reproductive, developmental or teratogenic effects were reported (INCHEM, 1998). In addition, there was no evidence of carcinogenicity in rats and mice in two-year drinking water studies (WHO, 2005a).

MCAA was not genotoxic in Ames assay and did not induce chromosomal aberrations in Chinese hamster ovary (CHO) cells with and without metabolic activation (INCHEM, 1998; WHO, 2005a). Several studies report negative results in assays for mutations in bacteria, and positive as well as negative results in tests for mutations and sister chromatid exchanges in eucaryotic cells *in vitro*. Intraperitoneal administration of MCAA in male and female mice significantly increased the rate of chromosomal aberrations in all dose groups after 6-120 h in the bone marrow. No effect was seen 24 hours after oral gavage or subcutaneous injection. Overall, there are mixed results from *in-vitro* assays and there is only one *in-vivo* study available for genotoxicity. Hence, based on these no conclusion could be reached on the genotoxicity of MCAA.

Monobromoacetic acid (MBAA)

Acute oral exposure of MBAA in rats showed clinical symptoms like excess drinking-water intake, hypomobility, laboured breathing and diarrhoea (WHO, 2005b).

No long-term toxicity studies were found in the literature for any exposure route (WHO, 2005b).

MBAA was mutagenic in Ames assay and also produced DNA strand breaks in mouse leukaemia cells (WHO, 2005b). These results have not been confirmed in a valid *in vivo* study.

No test substance related effects on male or female fertility were observed in a 2-generation study in rats receiving MBAA in drinking water up to the maximum concentration that could be administered.

Dichloroacetic acid (DCAA)

DCAA is readily and almost completely absorbed from the gastrointestinal tract of animals and humans (USEPA, 2003). Within 48 hours of administration, DCAA was found in the liver, muscle, skin, blood and intestines. The DCAA metabolism involves oxidative dechlorination to form glyoxylate. The metabolism of DCAA in humans is similar to animals.

Sub-acute and sub-chronic studies in animals revealed that the liver is the primary target organ for DCAA toxicity. In 90-day studies, rats were administered DCAA in drinking-water. Liver effects were observed at the highest dose, including focal hepatocellular enlargement, intracellular swelling, and glycogen accumulation (USEPA, 2003; WHO, 2005c).

Neurotoxicity was also evaluated in a number of studies in animals. In these studies alterations in the gait, mild tremors, hypotonia, and decreased forelimb grip strength were observed in mid and high dose levels after acute, sub-chronic, and chronic exposures (USEPA, 2003).

DCAA is reasonably “anticipated to be a human carcinogen” based on sufficient evidence of carcinogenicity from studies in experimental animals and supporting evidence from mechanistic studies (NTP, 2021b). Liver tumours were observed in rats and mice following administration of DCAA in drinking water. In mice of both sexes, significant increases in benign and malignant liver tumours (hepatocellular adenoma and carcinoma) were observed in several studies (NTP, 2021b; WHO, 2005c).

In a stop-exposure study, male and female mice were exposed at weaning (four weeks of age) to DCAA in drinking water for 10 weeks, followed by no further exposure to this chemical for 80 weeks. Significant increases in benign and malignant liver tumours were reported for both sexes, and tumour incidences approached levels found with near lifetime exposures. DCAA also significantly increased the incidence of hepatocellular carcinoma and combined hepatocellular adenoma and carcinoma in male rats in two drinking-water studies (NTP, 2021b; USEPA, 2003; WHO, 2005c).

The genotoxicity data on DCAA has been extensively reviewed. Most of the evidence indicates that DCAA is a weak mutagen, inducing mutations and chromosome damage *in vitro* (Ames assay, lymphoma mutation assay, CHO assay) and *in vivo* assays (micronuclei assay) but only at high doses (IRIS, 2003; USEPA, 2003). There was no alteration in micronucleus frequencies in male and female mice exposed to DCAA in drinking water for 3 months (NTP, 2007a). Hence, IARC concluded that DCAA is not mutagenic (IARC, 2004). USEPA considers that DCAA might be genotoxic, at least under *in vivo* exposure levels that are associated with detectable increases in tumour incidence at high doses. Overall, the genotoxicity results from *in-vitro* and *in-vivi* studies are considered equivocal. Hence, no conclusion could be reached on the genotoxicity of DCAA.

There are a few studies which evaluate the reproductive and developmental toxicity of DCAA in animals. However, there are no single or multiple generation studies of DCAA reproductive toxicity. In a 14-day study in adult male rats, delayed spermiation and formation of atypical residual bodies were observed after exposure to DCAA in drinking water at doses of 54 mg/kg bw/d and above (Linder *et al.*, 1997). Reduced foetal body weight was observed in the offspring of dams exposed to DCAA on gestation days 6-8. Fetal cardiac malformations were also reported on gestation days 9-11 and 12–15 (IARC, 2004; WHO, 2005c). In sub-chronic toxicity studies, DCAA exposure reduced the absolute weight of the preputial gland and epididymis at all dose levels, but the absolute weight of the testis was not affected (WHO, 2005c). A significant reduction in motile sperm, sperm motion and reduced epididymis sperm head counts was observed at the two highest doses. Impaired spermiation was noted in mid-dose and high-dose animals and was attributed to the retention of late-step spermatids in the seminiferous tubules.

Dibromoacetic acid (DBAA)

In long-term toxicity studies, administration of DBAA in the drinking water caused liver tumours in mice of both sexes and tumours at several other tissue sites (abdominal-pelvic peritoneum, blood, respiratory system, gastrointestinal tract) in rats of both sexes (NTP, 2007b).

DBAA was mutagenic in Ames test with *S. Typhimurium* tester strain TA100, with and without metabolic activation, but not in strain TA98. Increased frequencies of normochromatic erythrocytes (NCEs) were observed in peripheral blood samples from male mice administered 125 to 2000 mg DBAA/L in drinking water for 3 months. There was no increase in NCEs in female mice. No evidence of bone marrow toxicity, as measured by the percentage of immature polychromatic erythrocytes (PCEs) among total erythrocytes, was observed in either

male or female mice (NTP, 2007b). Hence, based on the results from *in-vivo* study, DBAA is not a mutagen.

DBAA is reasonably "anticipated to be a human carcinogen" based on sufficient evidence of carcinogenicity from studies in experimental animals and supporting evidence from mechanistic studies (NTP, 2021a).

There are a few studies which evaluate the reproductive and developmental toxicity of DBAA in animals. After 14-day oral exposures to DBAA, there were histopathologic alterations in the testis and epididymis, decreased testis and epididymis weights, abnormal sperm morphology, adverse effects on sperm motion, decreased testicular sperm head counts, and reduced caput and cauda epididymal sperm counts in the highest dose group (Linder *et al.*, 1994). In the subsequent 79-day oral study, the reproductive ability of male rats rapidly diminished in the high dose group which strongly compromised the reproductive outcome (Linder *et al.*, 1995). In a two-generation drinking water study, DBAA affected the sperm, testes, and epididymides in both the parent and first generation male rats and development of the epididymides in the F1 generation male rat (Christian *et al.*, 2002). Reproductive performance or development of the female rats was not affected after two generation exposure to female rats. As only males were affected, DBAA is considered a male reproductive and developmental toxicant, affecting the testes and sperm production, and producing a low incidence of epididymal malformations in the F1 generation male rats. These effects were observed at a very high dose which may not be relevant to human exposure and hence DBAA is not a reproductive or developmental toxicant in humans.

Trichloroacetic acid (TCAA)

TCAA is irritating and corrosive to skin and mucous membranes in humans. The liver was identified as the main target organ for TCAA toxicity after oral exposure in animals. Short-term effects of TCA administration include decreased body weight, increased liver weight, and increases in peroxisome proliferation in the liver and lipid peroxidation in the liver and kidney. Sub-chronic oral exposure to TCA appears to primarily affect liver size and weight, collagen deposition, lipid and carbohydrate metabolism, and peroxisome proliferation.

In chronic toxicity studies, hepatic effects, including increased relative liver weight, increased proliferation, necrosis, inflammation and peroxisome proliferation, and decreased body weight were observed in animals (IRIS, 2011). Apart from liver effects, increased testicular tubular degeneration (a significant dose trend and incidences at 0.5 and 5 g/L TCAA) and increased serum LDH (lactate dehydrogenase) activity, likely caused by increased inflammation and necrosis in the liver, was also observed in a 60- week study in male mice.

In rats, no treatment-related tumours were observed in a study of male F344/N rats exposed to TCAA via drinking water for 104 weeks. In contrast, TCAA is a complete carcinogen that significantly increased the incidence of liver tumours in male B6C3F1 mice exposed via drinking water for 52–104 weeks and female B6C3F1 mice exposed for 51 or 82 weeks. Based on this study, the US EPA concluded that there is suggestive evidence of carcinogenic potential for TCAA based on significantly increased incidences of liver tumours in male B6C3F1 mice exposed via drinking water for 52–104 weeks (IRIS, 2011). However, IARC concluded that TCAA is not classifiable as to its carcinogenicity to humans (Group 3) based on limited evidence in experimental animals for the carcinogenicity of TCAA (IARC, 1995a). TCAA can also act as a liver tumour promoter in rats or mice pretreated with a carcinogenic initiator before chronic exposure to TCAA in drinking water.

2.3.3 Halogenated acetonitriles (HANs)

Data on the toxicology of HANs is limited. In general, HANs are poorly absorbed in animals after oral administration. HANs are metabolised via oxidative dehalogenation and dehydration to carbon dioxide and cyanide. Cyanide is then further metabolised to thiocyanate.

Dibromoacetonitrile (DBAN)

No toxic effects were seen in sub-acute and sub-chronic toxicity studies conducted by the US National Toxicology Program in rats and mice after administration of DBAN in drinking-water (NTP, 2010). No reproductive or developmental effects were observed in a 30-day drinking water study in animals (NTP, 2010).

In a long-term cancer bioassay conducted by the NTP, drinking-water containing DBAN was administered to groups of rats and mice for two years (NTP, 2010). DBAN caused cancer of the oral cavity (buccal, gingival, hard palatine) in male and female rats and of the glandular stomach in male rats. In mice, squamous cell papilloma or carcinoma was observed in the forestomach of males and females. Tumours were found on the skin in female rats and in the liver in male mice. Hence, there is sufficient evidence of carcinogenic activity experimental animals. Hence, IARC classified DBAN as possibly carcinogenic (Group 2B) (IARC, 1995b).

DBAN was found to be weakly mutagenic in three Ames assays in the *S. Typhimurium* TA100 strain in the presence of rat or hamster S9 metabolic activation enzymes and occasionally in TA97, TA1535, and *Escherichia coli* strains in the presence of rat liver S9 mix. In a 3-month drinking water study in mice, no increases in the frequencies of micronucleated NCEs or significant alterations in the percentages of PCEs were seen in peripheral blood of both the sexes (NTP, 2010). Based on the results of *in-vivo* study, DBAN is not considered genotoxic.

Dichloroacetonitrile (DCAN)

There were limited toxicology studies in the literature that evaluated the potential of DCAN toxicity. From the limited studies available, the liver and kidney appear to be the target organs.

In a sub-acute oral toxicity study, exposure to 44 mg/kg bw/d DCAN caused hepatic and renal damage (Dong *et al.*, 2018). This was evident by an increase in activities of serum alanine aminotransferase and alkaline phosphatase, and concentrations of blood serum urea nitrogen and retinol-binding protein. Histopathology of the liver shows alterations including hepatic sinus dilation, extensive haemorrhage, vacuolar degeneration in the liver and glomerulus haemorrhage, and renal tubular swelling. Similar effects were observed in another oral sub-acute toxicity study.

In a sub-chronic toxicity study in rats, DCAN administered with corn oil had no consistent compound-related effects (haematological, serum chemistry or urinary parameters) were observed (WHO, 2004a). Alkaline phosphatase levels were significantly increased in males and females at the high dose and in males also at 33 mg/kg bw/d. Increased relative liver weight was observed in males, beginning at 33 mg/kg bw/d (60% increase), and in females, beginning at 8 mg/kg of body weight per day (17% increase). The relative liver weight was also increased in males (by 12%) at 8 mg/kg bw/d, but this was not statistically significant.

Pregnant Long-Evans rats were dosed with DCAN by oral intubation on Gestation Days 6-18. Embryolethality was observed in the high dose group (Smith *et al.*, 1989; WHO, 2004a). An

increased frequency of malformations of soft tissues, particularly of the cardiovascular and urogenital organs, and some skeletal malformations were observed in foetuses. The frequency of skeletal malformations (fused and cervical ribs) was also dose related and significantly increased at the highest dose (Smith *et al.*, 1989). Based on one dermal carcinogenicity study, no skin tumour was produced after applied topical application of DCAN in six equal doses over a two-week period (IARC, 1999c).

3 DOSE-RESPONSE INFORMATION

In the current context, concerns associated with exposure to DBP in SPW relate to chronic exposure. The risk assessments summarised in this report have used health based guidance values [reference doses (RfD) and cancer slope factors (CSF)] for THMs and HAAs derived by the USEPA. Although the US National Toxicology Program (NTP) has conducted some toxicology studies for emerging DBPs (HANs, HALs, HKs), they have not yet been reviewed and used by the USEPA to derive RfDs and CSFs and hence, the risks associated with these compounds have not been assessed in any of the published assessments.

USEPA has derived oral RfD and oral cancer slope factors (CSF) for some THMs and HAAs. The RfDs and CSFs are summarised below and the toxicology of DBPs is summarised in section 2.3. Oral CSFs were used for dermal exposure and inhalation when data were not available.

3.1.1 Trihalomethanes (THMs)

Table 14: Reference dose and CSF for chloroform, bromoform and dichloromethane

Study/key effect	Point of Departure (POD)	Uncertainty Factor	Reference dose (mg/kg bw/day)	Oral slope factor (mg/kg bw/day)	Reference
Chloroform					
Dog, chronic oral bioassay / Moderate/marked fatty cyst formation in the liver and elevated SGPT	$BMDL_{10}$: 1.2 mg/kg bw/day	100	0.01	0.01	(IRIS, 2001)
Bromoform					
Rat, Sub-chronic oral gavage Bioassay/ Hepatic lesions	NOEL: 25 converted to 18 mg/kg bw/day	1000	0.02	0.008	(IRIS, 1990)
Rat, Sub-chronic oral gavage Bioassay/ absence of histopathological lesions in the liver	NOEL: 25 converted to 18 mg/kg bw/day	1000	TDI: 0.018	-	(WHO, 2004b)
Dibromochloromethane					
Rat, Sub-chronic oral gavage bioassay/ hepatic lesions	NOEL: 30 converted to 21.5	1000	0.02	0.08	(IRIS, 1992)
Rat, Sub-chronic oral gavage bioassay/ hepatic lesions	NOEL: 30 converted to 21.5	1000	0.02	-	(WHO, 2004b)

BMDL: Benchmark dose level, NOEL: No observed effect level, bw: body weight

3.1.2 Haloacetic acids (HAAs)

Table 15: Reference dose and CSF for DCAA and TCAA

Study / key effect	Point of Departure (POD)	Uncertainty Factor	Reference dose (mg/kg bw/day)	Oral slope factor (mg/kg bw/day)	Reference
	Dichloroacetic acid				
Dog, sub-chronic oral bioassay / lesions in testes, cerebrum, cerebellum and liver	LOAEL: 12.52	3000	4E-03	0.05	(IRIS, 2003)
	Trichloroacetic acid				
Mice, 60-week drinking water exposure study/ hepatocellular necrosis in male mice	$BMDL_{10}$: 18	1000	0.02	0.07	(IRIS, 2011)

LOAEL: lowest observed adverse effect level; BMDL: Benchmark dose level, bw: body weight

4 EXPOSURE ASSESSMENT

4.1 EXPOSURE ASSESSMENT APPROACH

4.1.1 Relevant exposure scenarios

The general population is mainly exposed to DBPs in SPW via the oral, inhalation and dermal route. The exposure to DBPs depend on the physical activity of swimmers and level of their effort, average time of swimming, body surface area, inhalation rate and rate of inadvertent ingestion of pool water.

Table 16: Exposure routes considered for DBPs

Population	Product type	Exposure Pathway		
		Inhalation	Dermal	Oral
Adults	DBPs in pool water	X	X	X
Children		X	X	X

The SWIMODEL developed by the USEPA is a well-accepted screening exposure assessment, that uses equations to calculate the total worst-case exposure for swimmers expressed as a mass-based intake value (mg/event). The model focuses on potential chemical intakes only and does not take into account metabolism or excretion of the chemical of concern. The model considers oral, dermal, inhalation, buccal/sublingual, nasal/orbital and aural routes of exposure (USEPA, 2022). The buccal exposure of DBPs is caused by water taken into the mouth but not ingested (spat out). Aural exposure of DBPs occurs through the ear. The health risk assessments summarised in this report have used SWIMODEL to estimate the exposure of DBPs in different populations (USEPA, 2022).

Following are the formulas used to estimate the exposure in swimmers (USEPA, 2015).

Incidental Oral Exposure

Dose (mg/kg/day) = (CW x IGR x ED) / BW

Where:
CW = Chemical Concentration in Water (mg/liter)
IGR = Water Ingestion Rate (liters/hour)
ED = Exposure Duration (Hours/Day)
BW = Body Weight (Kg)

Dermal Exposure

Dose = CW x Kp x SA x ET x CF / BW

Where:
CW = Chemical Concentration in Water (mg/liter)
Kp = Permeability Constant (cm/hr)
SA = Surface Area (cm^2)
ET = Exposure Time (hours/day)
CF = Conversion Factor (0.001 Liter/cm^3)
BW = Body Weight (Kg)

Inhalation exposure and air concentration

$Cvc = Cw \times HLC \times 1000\ liter/m^3$

Where:
Cvc = Chemical vapor concentration (mg/m^3)
Cw = Chemical concentration in water
HLC = Henry's Law Constant (unitless)

$Dose\ (mg/m^3) = Cvc \times ED \times EF \times ET / AT$

Where:
ET = Exposure Time (hours/day)
ED = Exposure duration
EF = Exposure frequency
AT = Average time

The importance of the various exposure routes for particular DBPs depends on characteristics, such as their volatility. Inhalation is the dominant exposure route for THMs as these compounds are highly volatile and occur in the air above the swimming pool. For HAAs, oral and dermal routes are the main exposure routes as they are not volatile.

4.2 RELEVANT STUDIES

The following sections summarise the scope of the exposure assessments carried out. The table below summarises the target population, exposure routes and DBPs considered for risk characterisation. The SWIMODEL was used in all the studies for exposure assessment of DBPs in SPW. It should be noted that the exposure estimates were derived for all studies, but they were not reported. Hence, only three studies are summarised in detail for which exposure estimates were reported.

Table 17: List of studies for exposure assessment

DBP	Population	Exposure route	Exposure estimates	Reference
THMs and HAAs	Adults (>16 years), teens (11-16 years) and children (6-11 years)	Oral ingestion, dermal absorption, inhalation, buccal and aural exposure	N.R	(Zhang *et al.*, 2023)
HAAs	Adults	Oral and inhalation	Yes	(Zhao *et al.*, 2020)
THMs, HAAs, HANs, HKs	Children (3–<6; 6–<11, 11–<16 years) adults (>18 years)	Oral, dermal and inhalation	N.R	(Sdougkou *et al.*, 2021)
THMs, HAAs	Children (9–17 years), adults (>18 years)	Oral, dermal and inhalation	Yes	(Shi *et al.*, 2020)
THMs, HAAs, HANs, HKs and TCNM	Children and adults	Oral, dermal and inhalation	N.R	(Hang *et al.*, 2016)
NAs, THMs, HAAs	Adults	Oral, dermal and inhalation	N.R	(Peng *et al.*, 2020)
THMs	Adults	Oral, dermal and inhalation	N.R	(Anchal *et al.*, 2020)
HAAs	Children and adults	Oral, dermal and inhalation	N.R	(Ounsaneha *et al.*, 2017)
THM	Children (7–<10, 11–14 years), adults (>18 years)	Oral ingestion, dermal absorption, inhalation, buccal and aural exposure	N.R	(Pándics *et al.*, 2018)
THMs	Children and adults	Oral, dermal	N.R	(Pieters and Horn, 2020)
THMs	Adults	Oral, dermal	Yes	(Abbasnia *et al.*, 2019)

THMs: Trihalomethanes; HAAs: haloacetic acids; HANs: haloacetonitriles; HALs: haloaldehydes; NAs: Nitrosamines; HKs: Haloketones; TCNM: Trichloronitromethane; N.R: not reported

1. **Zhao *et al.,* 2020**

In this study, chronic daily exposures of five HAAs was estimated in adults for oral and inhalation routes of exposure following USEPA guidelines and the SWIMODEL (USEPA, 1989; 2022). The chronic daily intake (CDE) for each HAA was in the range of 1.3–22.6 x10^{-6} mg/kg bw/d. Swimmers were exposed to greater amounts of TCAA and DCAA (i.e., CDE of 1.1 x 10^{-5} mg/kg bw/d for DCAA and 2.3 x 10^{-5} mg/kg bw/d for TCAA for males), due to their several times' higher concentrations compared to other HAA compounds in SPWs. Dermal absorption was the main exposure pathway during pool activities (i.e., accounting for 58.5% of the total CDE for males). The CDE of each HAA in drinking waters followed the same decreasing order of CAA > BAA > DCAA > TCAA > DBAA as its concentrations (Zhao *et al.*, 2020).

2. **Shi *et al.,* 2020**

In this study, quantitative exposure assessments based on the exposure parameters in the early-life stage were performed, as childhood may be a particularly sensitive period for the development of cancer (Shi *et al.*, 2020). The swimmers were divided into two age groups: 9-17 years old as a pooled children/adolescent group, and ≥18 years old as the adult group. The ADD through oral, dermal and inhalation of each age group was then calculated based on corresponding exposure parameters and equations in SWIMODEL (USEPA, 1989; 2022).

Table 18: Average daily dose for THMs and HAAs

Exposure routes	**ADD (mg/kg/d)**	
Age: 9–17 years		Age: ≥18 years
THMs		
Inhalation	3 x 10^{-2}	2.66 x 10^{-2}
Dermal	2.7 x 10^{-5}	3.5 x 10^{-5}
Ingestion	2.7 x 10^{-6}	3.7 x 10^{-5}
HAAs		
Inhalation	1 x 10^{-6}	7.24 x 10^{-7}
Dermal	6 x 10^{-5}	7.65 x 10^{-5}
Ingestion	6.5 x 10^{-5}	3 x 10^{-6}

From the results, it is evident that inhalation was the main exposure route for THMs in both age groups. For HAAs, dermal and ingestion were the highest and of similar order of magnitude. TCM and TCAA were the highest contributors for THMs and HAAs, respectively.

3. **Abbasnia *et al.*, 2018**

CDI for THMs was estimated in an adult population for dermal contact and ingestion exposure using USEPA SWOMODEL (USEPA, 1989; 2022).

The CDIs of THMs after dermal contact were high which may be due to the low Kow (partition coefficient) of DBPs, causing higher permeability. The mean values of CDIs for chloroform, DCBM, DBCM, and bromoform were 2.12 x 10^{-6}, 7.35 x 10^{-7}, 8.12 x

10^{-9}, and 1.28 x 10^{-8} mg/kg-d through ingestion pathways and were 3.95 x 10^{-5}, 1.56 x 10^{-6}, 1.88 x 10^{-8} and 3.08 x 10^{-8} through dermal pathways (Abbasnia *et al.*, 2019).

5 RISK CHARACTERISATION

5.1 SUMMARY OF RISK CHARACTERISATION IN LITERATURE STUDIES

Non-carcinogenic risks and carcinogenic risks were estimated in the studies summarised here. For non-cancer risks, hazard quotient (HQ) was estimated using the following equation:

$$\mathrm{HQ} = \frac{\mathrm{Exposure}}{\mathrm{RfD}}$$

Where, exposure may be represented as CDE/I (chronic daily exposure/intake), LADD (lifetime average daily dose) and ADD (average daily dose). RfD is the chronic reference dose (mg/kg bw/d).

HQ ≤1 indicates that there would be no adverse health effects whereas HQ ≥1 indicates possible adverse health effects. For the combined effect of multiple chemicals, a hazard index (HI) can be derived which is the sum of HQs for each chemical considered. This is particularly used in a combined assessment of THMs and HAAs.

For carcinogenic risks, lifetime cancer risk (LCR) or cancer risk (CR) was estimated using the equation:

LCR = CDE x CSF

Where, CSF (mg/kg bw/d) is cancer slope factor. The appropriate slope factors from USEPA were used to approximate human health risks through different exposure pathways.

In New Zealand, carcinogenic risks are benchmarked against an estimated excess LCR of 10^{-5}. This means there is a risk of one additional cancer per 100,000 people (TaumataArowai, 2021). This is considered to be a level of negligible risk. Hence, risk estimates greater than 1 in 100,000 (1 x 10^{-5}) are regarded as a health concern.

1. **Zhang *et al.*, 2023**

   The total non-carcinogenic risks for exposure to THMs, HAAs and HANs for both competitive and non-competitive swimmers and for all age groups considered were less than 1, indicating a low non-carcinogenic health risk. The non-carcinogenic risk due to exposure to HANs was lower than for THMs and HAAs.

   The total carcinogenic risk of DBPs (THMS and HAAs) in swimming pools was estimated. The risks for competitive children, teens and adults of DBPs in swimming pool were 2.94 ×10^{-6} , 6.13 ×10^{-6} and 7.82 ×10^{-5}, respectively. Carcinogenic risks of non-competitive teens and adults were lower than that of the competitive swimmers due to low exposure frequency. The health risk of DBPs in swimming pool was much higher than their source water.

2. **Zhao *et al*., 2020**

Carcinogenic and non-carcinogenic risks to humans exposed to HAAs in SPWs was estimated by (Zhao *et al.*, 2020). The total risk of DCAA and TCAA in SPWs was ~2 x 10^{-6}. The non-carcinogenic risk (HI) of humans exposed in SPWs was 0.005 (less than 1).

3. **Sdougkou *et al*., 2021**

Carcinogenic and non-carcinogenic risks were estimated for THMs and HAAs as these were the dominant DBPs detected in the SPWs. The risks were estimated in children (3 to <6, 6 to <11, 11 to <16 years of age) and adults.

HQ values in children and adults ranged from 7 x 10^{-8} to 3 x 10^{-1} for both total THMs and total HAAs from ingestion, dermal and inhalation routes of exposure. These HQ are all less than 1 which suggests that the non-cancer risks were of low concern to public health.

Carcinogenic risks via oral and dermal exposure routes were low. The risk for THMs in adults and children following oral and dermal exposure ranged from 8 x 10^{-8} to 3 x 10^{-11} and 7 x 10^{-7} to 1 x 10^{-10}, respectively. However, the risks were higher (4 x 10^{-9} to 4 x 10^{-6}) for THMs following inhalation exposure, occasionally exceeding negligible risk level (NSRL) (10^{-6}). For HAAs, ingestion and dermal contact posed higher risk (3 x 10^{-8} to 3 x 10^{-6}) than the inhalation route (1 x 10^{-8} to 1 x 10^{-11}).

4. **Shi *et al*., 2020**

LCR was determined for individual DBP species (THMs and HAAs) for specific exposure routes for two age groups (9-17 and ≥18 years) (Shi *et al.*, 2020). The age-dependent adjustment factors (ADAF) were introduced to assess the early life carcinogenic risks, with an ADAF of 3 used for the 9–17 age group and an ADAF of 1 for the ≥18 age group. The CSF used to estimate LCR come from the studies (bioassay or epidemiological) that involve only adult exposures. Hence, when considering childhood exposure, ADAFs should be applied to CSF for estimating the risk for different age groups (USEPA, 2005). However, the basis of these ADAFs is not clear in the source document.

ADD was multiplied by slope factor (SF) or inhalation unit risk (IUR) to calculate the LCR. The total LCRs for THMs and HAAs through three routes (inhalation, dermal absorption and ingestion) were 3.8 x 10^{-4} and 7.4 x 10^{-6}, respectively. The LCR for THMs is higher than HAAs. The LCR value of TBM for ingestion was the lowest i.e 1 x 10^{-10} and the LCR of TCM for inhalation was the highest i.e. 2.7 x 10^{-4}.

5. **Hang *et al*., 2016**

Carcinogenic and non-carcinogenic risks were estimated for THMs (TCM, BDCM, TBM) and HAAs (DCAA and TCAA for non-cancer risks only) as these were the dominant DBPs detected in the SPWs (Hang *et al.*, 2016). For non-carcinogenic risks, HQ was calculated from oral, dermal and inhalation exposures to DBPs. LCR was determined for individual DBP species for specific exposure routes. Estimates were derived for three pool groups (A: ozonation followed by chlorination, B: chlorinated pool and C-M: typical pools with chlorination) and for athletic and non-athletic adult males, females and children.

The carcinogenic risk estimates and HQs in this study were relatively high due to the high levels of DBPs in SPW and air. The total HIs for the five DBPs (TCM, BDCM, TBM, DCAA, TCAA) were <1 for athletic adults which does not raise a health concern. However, most of the HIs for non-athletic adults and for all children were >1, indicating a health concern. The LCR was in the range of 10^{-1} to 10^{-4} for all pools.

6. Peng *et al.*, 2020

In this study, the total LCRs posed by inhalation, dermal absorption and ingestion of four THMs, two HAAs (DCAA and TCAA), and seven NAs were assessed (Peng *et al.*, 2020).

The carcinogenic risks through dermal exposure were higher than those through ingestion. HAAs were the main contributor for carcinogenic risks. The total carcinogenic risks following ingestion, dermal and inhalation exposure is presented in Table 19. The mean total risk for women was higher than men for all routes of exposure. This was due to the differences in skin surface area and body weight.

Table 19: Mean carcinogenic risks from ingestion and dermal exposure to NAs, HAAs, and THMs

Carcinogenic risk (mg/kg/d)		
Population	Men	Women
Ingestion & dermal		
Total risk	1.5×10^{-6}	1.7×10^{-6}
Inhalation		
Total risk	4.2×10^{-4}	5.1×10^{-4}

7. Anchal *et al*., 2020

Carcinogenic and non-carcinogenic risks to adults exposed to THMs in SPWs was estimated (Anchal *et al.*, 2020). For non-carcinogenic risks, a hazard quotient (HQ) was estimated for oral, and dermal exposures.

All the HQs were less than 0.01 for both males and females after oral and dermal exposure which indicates a low health concern for non-cancer risks.

The total carcinogenic risk of THM exposure was calculated by adding up the estimated individual risk for the three exposure routes. Since chloroform was the predominant THM detected in the SPW, carcinogenic risk was estimated for chloroform only. The estimated average carcinogenic risk through all the three exposure routes is presented in Table 20.

Table 20: Carcinogenic risk from chloroform

Exposure pathways	Estimated risk	
	Male	**Female**
Oral	9.41 E-08	9.81 E-08
Dermal	4.82 E-07	5.16 E-07
Inhalation	7.49 E-03	13.71 E-03

The LCR due to THMs from the ingestion and dermal routes of exposure was very low ($<10^{-6}$). The carcinogenic risk from inhalation exposure is more than the negligible risk level (10^{-6}) thus indicating possible concerns on human health.

8. Ounsaneha *et al*., 2017

Carcinogenic and non-carcinogenic risks for HAAs were estimated using USEPA SWIMODEL (USEPA, 1989; 2022). For carcinogenic risks, LCR was determined for individual HAA and total HAAs. For non-carcinogenic risks, HQ was calculated for individual HAA and total HAAs following oral, dermal and inhalation exposures.

The HQs for indoor and outdoor pools were in the range of 3.31×10^{-2} and 1.72×10^{-1}. In the summer season, all HQ values were higher than during the rainy season and the HQ values in outdoor pools were also significantly higher than in indoor pools (Ounsaneha *et al.*, 2017).

The overall LCR for total HAA exposure in SPWs was 8×10^{-6}–6×10^{-5}. LCR values were significantly higher in the outdoor pool as compared to the indoor pools due to the fact that UV radiation promotes the formation of DBPs and thus increases the exposure (Liu *et al.*, 2006).

9. Pándics *et al*., 2018

In this study, total excess lifetime carcinogenic risk (ELCR) associated with chloroform was estimated in different age groups (adults, 7–10 and 11–14 year olds).
The total ELCRs of chloroform from swimming exposure of recreational and elite swimmers in different age groups by all exposure routes are summarised in Table 21.

Table 21: Total ELCR in recreational and elite swimmers

Recreational swimmers								
SWIMODEL					**ConsExpo**			
Age (yrs)	**7-10**	**11-14**	**Adult**	**Total**	**7-10**	**11-14**	**Adult**	**Total**
Sum of all routes	2.5×10^{-6}	1.16×10^{-6}	4.10×10^{-6}	7.75×10^{-6}	2×10^{-6}	1.2×10^{-6}	3.4×10^{-6}	6.5×10^{-6}
Elite swimmers								
Sum of all routes	2.15×10^{-6}	6.5×10^{-6}	5.10×10^{-5}	6×10^{-5}	3×10^{-6}	6.5×10^{-6}	4×10^{-5}	4×10^{-5}

From the data, the total ELCRs in all age groups exceeded 10^{-6} for both recreational and elite swimmers. The risk for elite swimmers was even higher for adults. Although ConsExpo only allows estimation of oral, dermal and inhalation exposure based on

concentrations in water, the results were similar to those calculated by SWIMODEL that also included estimates based on air concentrations.

10. Pieters and Horn, 2020

The non-cancer risks for THMs were characterised by estimating HIs for each sample (two pools, various locations and within the pool area), for each compound for adults and children (Pieters and Horn, 2020).

Overall, the carcinogenic and non-carcinogenic risks were higher through dermal exposure than ingestion in both age groups. Children were more at risk as compared to adults. The HQ was >1 for both age groups following dermal exposure which indicated high health risk concern due to non-cancerous effects. The HQ was <1 for both age groups following ingestion of SPWs. The CR following dermal exposure was the highest in both the age groups and was $>10^{-6}$, indicating high risk to the general public. The CR following ingestion was mostly $>10^{-6}$ in both age groups, indicating a high risk to the general public.

11. Abbasnia *et al*, 2018

LCR was estimated for THMs in SPW (Abbasnia *et al.*, 2019). The carcinogenic risks by the dermal route of exposure were higher than by the ingestion route. Also, the risks of chlorinated THMs were higher than brominated THMs. The mean of total LCR for swimmers through ingestion and dermal pathway was 4.63×10^{-8} and 6.5×10^{-8} due to low exposure to THMs.

SUMMARY

From the available literature there seems to be no concern for non-cancer risk to public health due to exposure of THMs and HAAs in SPW. The HQ in most studies summarised was <1, which indicated no health concern to adults and children. However, two studies reported potential health risks to DBPs. In one study, the HQ was >1 for adults and children following dermal exposure to chloroform (Pieters and Horn, 2020). In a second study, the total HIs for the five DBPs (TCM, BDCM, TBM, DCAA, TCAA) following oral, dermal and inhalation exposure was >1 for all children and most adults (Hang *et al.*, 2016). This was due to the presence of DBPs in high concentrations in air and SPW.

In New Zealand, carcinogenic risks are benchmarked against an estimated excess LCR of 10^{-5}. This means there is a risk of one additional cancer per 100,000 people (TaumataArowai, 2021). This is considered to be a level of negligible risk. Hence, a risk estimate greater than 1 in 100,000 (1×10^{-5}) are regarded as a health concern. The studies summarised indicate that carcinogenic risks were mostly less than the estimated excess LCR of 10^{-5} following the oral and sometimes dermal route of exposure to DBPs. However, the estimated risk was $>10^{-5}$ following inhalation and sometimes dermal exposure of THMs (chloroform) and HAAs, respectively. The results should be interpreted with caution as the outcome of the risk assessment could be significantly over or under-estimated due to a number of uncertainties such as actual exposure frequency, local water sanitation, human body constitution, seasonal variation (summer vs winter), physical protection, etc., affecting the accuracy of the results. Nevertheless, total carcinogenic risk due to DBPs in swimming pools should not be considered as negligible and measures should be taken to minimise the exposure to DBPs without compromising disinfection efficiency.

6 CONCLUSIONS

The purpose of this report is to summarise generic health risk assessment data for incidental exposure to disinfection by-products in swimming pool water such as, trihalomethanes and haloacetic acids. This report only considers domestic, non-occupational, routine, and incidental exposure to disinfection by-products in swimming pool water from recreational activities.

Disinfection of water is crucial to maintain hygienic conditions in swimming pools and spas. Chlorine and sometimes bromine products are used for water disinfection because of their low cost and effectiveness against many pathogens, especially bacteria and viruses. Chlorine or bromine in swimming pool water reacts with organic matter and residues of personal care products (shampoos, body lotion, sunscreen and other cosmetics) from swimmers, in addition to the natural organic matter from the source water, to form disinfection by-products. Trihalomethanes and haloacetic acids are the major swimming pool disinfection by-products, and their presence has been widely reported in both swimming pool water and drinking-water. Other emerging DBPs detected in swimming pool water are haloacetonitriles, haloketones, haloaldehydes, halonitromethanes, and nitrosamines. The formation of disinfection by-products depend on number of factors such as pH, temperature, the presence of residues of personal care products, and residual-free chlorine and bromide levels.

Quality standards for swimming pool water exist for microbiological hazards but there are fewer quality standards for chemical hazards in swimming pool water. Only a few countries (mostly in Europe) have established swimming pool water quality standards for disinfection by-products, although many countries have quality standards for disinfection by-products in drinking-water. The emerging disinfection by-products are not regulated and data on their safety and occurrence are limited. In New Zealand, there is no regulation or standards around disinfection by-products in swimming pool water. Maximum acceptable values for a range of trihalomethanes and haloacetic acids in drinking water standards are in place, based on guideline values set by the World Health Organization. As per the guidance, these values can be used to screen for potential risks arising from disinfection by-products from swimming pools and similar environments.

Systemic exposure to disinfection by-products can be by oral, dermal and inhalation routes. Local toxicological effects (skin irritation and rashes) can also occur. Trihalomethanes and haloacetic acids are the disinfection by-products with the most extensive toxicology data sets. Some trihalomethanes and haloacetic acids are carcinogenic, fetotoxic and mutagenic. The exposure of swimmers to disinfection by-products depends on the physical activity and average time of swimming, body surface area, inhalation rate and rate of inadvertent ingestion of pool water. The Swimmer Exposure Model, developed by the U.S. Environmental Protection Agency has been extensively used in exposure assessments of disinfection by-products in swimming pool water. It considers the primary routes of exposure to disinfection by-products (dermal, oral or inhalation) but also considers buccal/sublingual, nasal/orbital and aural as supplemental exposure routes.

The relative importance of the various exposure routes depends on the physicochemical properties of the disinfection by-products. For example, trihalomethanes such as chloroform are volatile, and inhalation is the dominant route of exposure. For haloacetic acids, dermal and oral routes are the main exposure routes as they are non-volatile.

For non-carcinogenic risks, hazard quotients were less than 1 for all disinfection by-products in most of the studies, indicating a low health risk for the general population. However, two studies reported potential health risks to disinfection by-products. In one study, the hazard quotient was >1 for adults and children following dermal exposure to disinfection by-products which indicated health risk concern due to non-carcinogenic effects. In the second study, the total hazard indexes for the five disinfection by-products following oral, dermal and inhalation exposure was >1 for all children and most adults. This was due to the detection of high levels of disinfection by-products in air and swimming pool water.

The excess lifetime cancer risks were less than 10^{-5} in most of the studies, following oral and dermal routes of exposure to disinfection by-products. However, in some studies the estimated carcinogenic risk was >10^{-5} following inhalation and dermal exposure of trihalomethanes and haloacetic acids, respectively. The studies summarised indicate that carcinogenic risks were mostly less than the estimated excess lifetime risk of 10^{-5} following the oral and sometimes dermal route of exposure to disinfection by-products. However, the estimated carcinogenic risk was >10^{-5} following inhalation and dermal exposure of trihalomethanes (chloroform) and haloacetic acids, respectively. The results should be interpreted with caution as the outcome of the risk assessment could be significantly over or under-estimated due to a number of uncertainties such as actual exposure frequency, local water sanitation, human body constitution, seasonal variation (summer vs winter), physical protection, etc., affecting the accuracy of the results. Nevertheless, total carcinogenic risk due to disinfection by-products in swimming pools should not be considered as negligible and measures should be taken to minimise the exposure to disinfection by-products without compromising disinfection efficiency.

7 REFERENCES

Abbasnia A, Ghoochani M, Yousefi N, Nazmara S, Radfard M, Soleimani H, Yousefi M, Barmar S, Alimohammadi M. (2019) Prediction of human exposure and health risk assessment to trihalomethanes in indoor swimming pools and risk reduction strategy. Human and Ecological Risk Assessment: An International Journal; 25(8): 2098-2115.

Anchal P, Kumari M, Gupta SK. (2020) Human health risk estimation and predictive modeling of halogenated disinfection by-products (chloroform) in swimming pool waters: a case study of Dhanbad, Jharkhand, India. Journal of Environmental Health Science and Engineering; 18: 1595-1605.

ATDSR. (2005) Toxicological profile for bromoform and dibromochloromethane. USA: Agency for Toxic Substances and Disease Registry Centers for Disease Control and Prevention.

ATSDR. (1997) Toxicological profile for chloroform. USA: Agency for Toxic Substances and Disease Registry Centers for Disease Control and Prevention.

Beech JA, Diaz R, Ordaz C, Palomeque B. (1980) Nitrates, chlorates and trihalomethanes in swimming pool water. American Journal of Public Health; 70(1): 79-82.

Carter RA, Joll CA. (2017) Occurrence and formation of disinfection by-products in the swimming pool environment: A critical review. Journal of Environmental Sciences; 58: 19-50.

Chowdhury S, Alhooshani K, Karanfil T. (2014) Disinfection byproducts in swimming pool: occurrences, implications and future needs. Water Research; 53: 68-109.

Christian M, York R, Hoberman A, Frazee J, Fisher L, Brown W, Creasy D. (2002) Oral (drinking water) two-generation reproductive toxicity study of dibromoacetic acid (DBA) in rats. International journal of Toxicology; 21(4): 237-276.

Dong Y, Li F, Shen H, Lu R, Yin S, Yang Q, Li Z, Wang S. (2018) Evaluation of the water disinfection by-product dichloroacetonitrile-induced biochemical, oxidative, histopathological, and mitochondrial functional alterations: Subacute oral toxicity in rats. Toxicology and Industrial Health; 34(3): 158-168.

DrinkingWaterStandards. (2022) DrinkingWaterStandards. Accessed at: https://www.legislation.govt.nz/regulation/public/2022/0168/latest/whole.html. Accessed: 2 October 2023.

ECHA. (2017) Guidance on the biocidal products regulation. volume V, guidance on disinfection by-products. Helsinki, Finland: European Chemicals Agency Helsinki.

Florentin A, Hautemanière A, Hartemann P. (2011) Health effects of disinfection by-products in chlorinated swimming pools. International Journal of Hygiene and Environmental Health; 214(6): 461-469.

Font-Ribera L, Marco E, Grimalt JO, Pastor S, Marcos R, Abramsson-Zetterberg L, Pedersen M, Grummt T, Junek R, Barreiro E. (2019) Exposure to disinfection by-products in swimming

pools and biomarkers of genotoxicity and respiratory damage–The PISCINA2 Study. Environment International; 131: 104988.

Genisoglu M, Minaz M, Tanacan E, Sofuoglu SC, Kaplan-Bekaroglu SS, Kanan A, Ates N, Sardohan-Koseoglu T, Yigit NO, Harman BI. (2023) Halogenated by-products in chlorinated indoor swimming pools: a long-term monitoring and empirical modeling study. Acs Omega; 8(12): 11364-11372.

Hang C, Zhang B, Gong T, Xian Q. (2016) Occurrence and health risk assessment of halogenated disinfection byproducts in indoor swimming pool water. Science of the Total Environment; 543: 425-431.

Hayashi á, Kishi á, Sofuni T, Ishidate Jr M. (1988) Micronucleus tests in mice on 39 food additives and eight miscellaneous chemicals. Food and Chemical Toxicology; 26(6): 487-500.

Honer W, Ashwood-Smith M, Warby C. (1980) Mutagenic activity of swimming-pool water. Mutation Research/Genetic Toxicology; 78(2): 137-144.

IARC. (1995a) Trichloroacetic acid_IARC monographs Geneva: INTERNATIONAL AGENCY FOR RESEARCH ON CANCER.

IARC. (1995b) Dibromoacetonitrile_IARC monographs. Geneva: INTERNATIONAL AGENCY FOR RESEARCH ON CANCER.

IARC. (1999a) Bromoform_IARC monographs Geneva: INTERNATIONAL AGENCY FOR RESEARCH ON CANCER.

IARC. (1999b) Chloroform_IARC monographs Geneva: INTERNATIONAL AGENCY FOR RESEARCH ON CANCER.

IARC. (1999c) Dichloroacetonitrile_IARC monographs. Geneva: INTERNATIONAL AGENCY FOR RESEARCH ON CANCER.

IARC. (2004) Dichloroacetic acid_Ic. Geneva: INTERNATIONAL AGENCY FOR RESEARCH ON CANCER.

INCHEM. (1991) International programme on chemical safety poisons information monograph 121 chemical_choloroform. Accessed at: https://www.inchem.org/documents/pims/chemical/pim121.htm#SectionTitle:7.2%20%20Toxicity. Accessed: 2 October 2023.

INCHEM. (1998) International programme on chemical safety poisons information monograph 121 chemical_monochloroacetic acid. Accessed at: https://inchem.org/documents/pims/chemical/pim352.htm#SubSectionTitle:7.2.5%20%20Acceptable%20daily%20intake%20(ADI). Accessed: 2 October 2023

IRIS. (1990) Bromoform CASRN 75-25-2 | DTXSID1021374. Accessed at: https://iris.epa.gov/ChemicalLanding/&substance_nmbr=214. Accessed: 2 October 2023

IRIS. (1992) Dibromochloromethane CASRN 124-48-1 | DTXSID1020300. Accessed at: https://iris.epa.gov/ChemicalLanding/&substance_nmbr=222. Accessed: 2 October 2023.

IRIS. (2001) Chloroform; CASRN 67-66-3. Accessed at: https://iris.epa.gov/static/pdfs/0025_summary.pdf. Accessed: 2 October 2023.

IRIS. (2003) Dichloroacetic acid; CASRN 79-43-6. Accessed at: https://iris.epa.gov/ChemicalLanding/&substance_nmbr=654. Accessed: 2 October 2023

IRIS. (2011) Trichloroacetic acid (TCA); CASRN 76-03-9. Accessed at: https://iris.epa.gov/static/pdfs/0655_summary.pdf. Accessed: 13 October 2023.

Linder RE, Klinefelter GR, Strader LF, Suarez JD, Roberts NL, Dyer CJ. (1994) Spermatotoxicity of dibromoacetic acid in rats after 14 daily exposures. Reproductive Toxicology; 8(3): 251-259.

Linder RE, Klinefelter GR, Strader LF, Narotsky MG, Suarez JD, Roberts NL, Perreault SD. (1995) Dibromoacetic acid affects reproductive competence and sperm quality in the male rat. Fundamental and Applied Toxicology; 28(1): 9-17.

Linder RE, Klinefelter GR, Strader LF, Suarez JD, Roberts NL. (1997) Spermatotoxicity of dichloroacetic acid. Reproductive Toxicology; 11(5): 681-688.

Liu W, Cheung L-M, Yang X, Shang C. (2006) THM, HAA and CNCl formation from UV irradiation and chlor (am) ination of selected organic waters. Water Research; 40(10): 2033-2043.

Liviac D, Wagner ED, Mitch WA, Altonji MJ, Plewa MJ. (2010) Genotoxicity of water concentrates from recreational pools after various disinfection methods. Environmental Science & Technology; 44(9): 3527-3532.

Muellner MG, Wagner ED, McCalla K, Richardson SD, Woo Y-T, Plewa MJ. (2007) Haloacetonitriles vs. regulated haloacetic acids: are nitrogen-containing DBPs more toxic? Environmental Science & Technology; 41(2): 645-651.

NationalStandardChina. (2006) GB 5749-2006 Standards for drinking water quality. China: Ministry of Health.

NTP. (1989) Toxicology and carcinogenesis tudies of tribromomethane (bromoform) in F344/N rats and B6C3F1 mice (gavage studies). Accessed at: https://ntp.niehs.nih.gov/sites/default/files/ntp/htdocs/lt_rpts/tr350.pdf. Accessed: 2 October 2023.

NTP. (2007a) Toxicology studies of dichloroacetic acid in genetically modified (FVB Tg.AC Hemizygous) mice (dermal and drinking water studies) and carcinogenicity studies of dichloroacetic acid in genetically modified [B6.129-Trp53tm1Brd (N5) Haploinsufficient] mice (drinking water studies). Accessed at: https://ntp.niehs.nih.gov/publications/reports/gmm/gmm11. Accessed: 2 October 2023.

NTP. (2007b) Toxicology and carcinogenesis studies of dibromoacetic acid in F344/N rats and B6C3F1 mice (drinking water studies). Accessed at: https://ntp.niehs.nih.gov/publications/reports/tr/500s/tr537. Accessed: 2 October 2023.

NTP. (2010) Toxicology and carcinogenesis studies of dibromoacetonitrile (CAS NO. 3252-43-5) in f344/n rats and b6c3f1 mice (drinking water studies). Accessed at:

https://ntp.niehs.nih.gov/publications/reports/tr?type=Technical%20Report. Accessed: 2 October 2023.

NTP. (2021a) 15th report on carcinogens. Haloacetic acids found as water disinfection by-products. Accessed at: https://ntp.niehs.nih.gov/sites/default/files/ntp/roc/content/profiles/haloacetic_acids.pdf. Accessed: 2 October 2023.

NTP. (2021b) Haloacetic acids found as water disinfection by-products. Accessed at: https://ntp.niehs.nih.gov/sites/default/files/ntp/roc/content/profiles/haloacetic_acids.pdf. Accessed: 2 october 2023.

Ounsaneha W, Kraisin P, Suksaroj TT, Suksaroj C, Rattanapan C. (2017) Health risk assessment from haloacetic acids exposure in indoor and outdoor swimming pool water. EnvironmentAsia; 10(2): 177-185.

Pándics T, Hofer Á, Dura G, Vargha M, Szigeti T, Tóth E. (2018) Health risk of swimming pool disinfection by-products: a regulatory perspective. Journal of Water and Health; 16(6): 947-957.

Peng F, Peng J, Li H, Li Y, Wang B, Yang Z. (2020) Health risks and predictive modeling of disinfection byproducts in swimming pools. Environment International; 139: 105726.

Pieters R, Horn S. (2020) Chemicals of concern in recreational waters. Occurrence and assessment of potential human health risks of chemicals in public swimming pools. Water Research Commission, Pretoria. WRC Report; (2804/1): 20.

Plewa MJ, Wagner ED, Mitch WA. (2011) Comparative mammalian cell cytotoxicity of water concentrates from disinfected recreational pools. Environmental Science & Technology; 45(9): 4159-4165.

Richardson SD, Plewa MJ, Wagner ED, Schoeny R, DeMarini DM. (2007) Occurrence, genotoxicity, and carcinogenicity of regulated and emerging disinfection by-products in drinking water: a review and roadmap for research. Mutation Research/Reviews in Mutation Research; 636(1-3): 178-242.

Sdougkou A, Kapsalaki K, Kozari A, Pantelaki I, Voutsa D. (2021) Occurrence of disinfection by-products in swimming pools in the area of Thessaloniki, Northern Greece. Assessment of multi-pathway exposure and risk. Molecules; 26(24): 7639.

Shi Y, Ma W, Han F, Geng Y, Yu X, Wang H, Kimura SY, Wei X, Kauffman A, Xiao S. (2020) Precise exposure assessment revealed the cancer risk and disease burden caused by trihalomethanes and haloacetic acids in Shanghai indoor swimming pool water. Journal of Hazardous Materials; 388: 121810.

Smith M, Randall J, Stober J, Read E. (1989) Developmental toxicity of dichloroacetonitrile: a by-product of drinking water disinfection. Toxicological Sciences; 12(4): 765-772.

Srivastav AL, Kaur T. (2020) Factors affecting the formation of disinfection by-products in drinking water: human health risk. In: (eds). Disinfection by-products in drinking water. Elsevier.

StandardsNewZealand. (2010) NZS 5826:2010; Pool water quality. Standards New Zealand.

Stocker KJ, Statham J, Howard WR, Proudlock RJ. (1997) Assessment of the potential in vivo genotoxicity of three trihalomethanes: chlorodibromomethane, bromodichloromethane and bromoform. Mutagenesis; 12(3): 169-173.

TaumataArowai. (2021) Drinking water standards for New Zealand (draft) 20 December 2021. Accessed at: https://te-puna-korero.taumataarowai.govt.nz/regulatory/drinking-water-standards/user_uploads/drinking-water-standards.pdf. Accessed: 2 October 2023.

Terasaki M, Makino M. (2008) Determination of chlorinated by-products of parabens in swimming pool water. International Journal of Environmental and Analytical Chemistry; 88(13): 911-922.

USEPA. (1989) Risk assessment guidance for superfund (RAGS) Part A. Accessed at: https://www.epa.gov/sites/default/files/2015-09/documents/rags_a.pdf. Accessed: 13 September 2023.

USEPA. (2003) Toxicological review of dichloroacetic acid (CAS No. 79-43-6) Washington, DC U.S. Environmental Protection Agency.

USEPA. (2005) Supplemental guidance for assessing susceptibility from early-life exposure to carcinogens. Risk Assessment Forum, US Environmental Protection Agency.

USEPA. (2006) Drinking water requirements for states and public water systems. Accessed at: https://www.epa.gov/dwreginfo/stage-1-and-stage-2-disinfectants-and-disinfection-byproducts-rules. Accessed: 29 August 2023.

USEPA. (2015) Evaluation of swimmer exposures using the SWIMODEL algorithms and assumptions. Accessed at: https://www.epa.gov/sites/default/files/2016-11/documents/swimodel_final.pdf. Accessed: 2 Octover 2023.

USEPA. (2022) Swimmer exposure assessment model (SWIMODEL). Accessed at: https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/swimmer-exposure-assessment-model-swimodel. Accessed: 13 Spetember 2023.

WHO. (2004a) Halogenated acetonitriles in drinking-water background document for development of WHO guidelines for drinking-water quality. World Health Organisation.

WHO. (2004b) Trihalomethanes in drinking-water. Background document for development of WHO guidelines for drinking-water quality. Geneva: World Health Organization.

WHO. (2005a) Monochloroacetic acid in srinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. World Health Organization:

WHO. (2005b) Brominated acetic acids in drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality World Health Organization.

WHO. (2005c) Dichloroacetic acid in drinking-water. World Health Organization.

WHO. (2006) Guidelines for safe recreational water environments. Volume 2, Swimming pools and similar environments. 1-146.

WHO. (2017) Guidelines for drinking-water quality. WHO chronicle; 38(4): 104-108.

Xu X, Mariano TM, Laskin JD, Weisel CP. (2002) Percutaneous absorption of trihalomethanes, haloacetic acids, and haloketones. Toxicology and Applied Pharmacology; 184(1): 19-26.

Yang L, Chen X, She Q, Cao G, Liu Y, Chang VW-C, Tang CY. (2018) Regulation, formation, exposure, and treatment of disinfection by-products (DBPs) in swimming pool waters: A critical review. Environment International; 121: 1039-1057.

Ye B, Wang W, Yang L, Wei J, Xueli E. (2009) Factors influencing disinfection by-products formation in drinking water of six cities in China. Journal of Hazardous Materials; 171(1-3): 147-152.

Zhang D, Dong S, Chen L, Xiao R, Chu W. (2023) Disinfection byproducts in indoor swimming pool water: Detection and human lifetime health risk assessment. Journal of Environmental Sciences; 126: 378-386.

Zhao H, Yang L, Li Y, Xue W, Li K, Xie Y, Meng S, Cao G. (2020) Environmental occurrence and risk assessment of haloacetic acids in swimming pool water and drinking water. RSC Advances; 10(47): 28267-28276.

Science for Communities

INSTITUTE OF ENVIRONMENTAL
SCIENCE AND RESEARCH LIMITED

Kenepuru Science Centre
34 Kenepuru Drive, Kenepuru, Porirua 5022
PO Box 50348, Porirua 5240
New Zealand
T: +64 4 914 0700 **F:** +64 4 914 0770

Mt Albert Science Centre
120 Mt Albert Road, Sandringham, Auckland 1025
Private Bag 92021, Auckland 1142
New Zealand
T: +64 9 815 3670 **F:** +64 9 849 6046

NCBID – Wallaceville
66 Ward Street, Wallaceville, Upper Hutt 5018
PO Box 40158, Upper Hutt 5140
New Zealand
T: +64 4 529 0600 **F:** +64 4 529 0601

Christchurch Science Centre
27 Creyke Road, Ilam, Christchurch 8041
PO Box 29181, Christchurch 8540
New Zealand
T: +64 3 351 6019 **F:** +64 3 351 0010

www.esr.cri.nz