APPENDIX 6.1 METHODOLOGY FOR HUMAN HEALTH RISK ASSESSMENT

Assessment Approach

1.1 The Human Health Risk Assessment (HHRA) was conducted with the following phases:

- Problem Formulation
- Hazard Identification, which consists of
  - Contaminant of Potential Concern (COPC) Identification and Selection of Contaminant of Concern (COC)
  - Potential Human Receptors Identification
- Exposure Assessment
- Dose-response Assessment
- Risk/hazard Characterization

1.2 Problem Formulation phase is added to cater the suggested approach in the Study Brief. The remaining phases of the proposed assessment approach is very similar to the model developed by the National Academy of Sciences (NAS) in the USA in 1983, which is widely used and accepted in the human health risk assessment for the impact due to chemicals. The NAS model also consists of four steps: hazard identification, dose-response assessment, exposure assessment and risk characterization.

Problem Formulation

1.3 The following tasks were accomplished in this phase:

- Establish objective of the assessment
- Establish scope of the assessment
- Establish focus of the assessment
- Construct Site Conceptual Model
- Define assessment endpoint(s)

1.4 The objective, scope and focus of the HHRA have been discussed in **Section 6** of the EIA Report.

Site Conceptual Model

1.5 The SCM adopted in the HHRA was presented graphically in **Figures 6.1**. As seen in the figure, there are 3 types of exposure pathway in terms of completeness and significance, namely “exposure pathway complete and significant”, “exposure pathway complete, but insignificant or significance unknown” and “exposure pathway incomplete”. For the exposure pathway “complete and significant”, it means that contaminants can be up-taken by receptors through that pathway and the amount of uptake can be considerable to contribute to the risk level. This type of exposure pathway was considered in the risk assessment.

1.6 For the exposure pathway “complete, but insignificant or significance unknown”, it means that contaminants can be up-taken by receptors through that pathway but the amount of uptake is not sufficiently large to affect the risk level or the amount of uptake through that pathway is uncertain for determining the risk level. This type of exposure pathway was not considered in the risk assessment. For the “incomplete exposure pathway”, it means that the contaminants cannot be up-taken by the receptor through that pathway because there is no complete route for the contaminants to reach the receptor. This type of exposure pathway was not considered in the risk assessment.

1.7 The SCM was presented in text as shown in **Table 1**.

Table 1 SCM for Human Health Risk Assessment

Contaminant Source:	Effluent from the outfall of SCISTW
Receptor:	Humans (children and adult)
Complete and Significant Exposure Media and Pathway[1]:	• Incidental ingestion of seawater • Ingestion of seafood (contaminated) • Dermal contact of seawater

1.8 For most of the contaminants, the exposure via direct contact (i.e. dermal exposure) is considered to be very low due to their low permeability coefficients from water. Therefore, dermal exposure for most of the contaminants can be considered as a complete but insignificant pathway. However, the fastest penetrating contaminants may pose hazards similar to or greater than direct consumption (ingestion of water) for prolonged dermal exposure time (USEPA 1992). For the sake of conservatism, the risk contributed by dermal exposure was assessed in the HHRA.

Assessment Endpoint

1.9 The assessment endpoint for the HHRA is defined as protection of human health at individual level from chronic exposure of contaminants produced in disinfection process via the incidental ingestion and dermal contact of diluted effluent from SCISTW, and the dietary ingestion of seafood over a relatively long period of time. The measurement endpoint for the HHRA is to evaluate chemical doses that are unlikely to result in significant incremental chronic systematic or carcinogenic effects.

Identification of COPC and Selection of COC

Identification of COPC

1.10 A total number of 35 chemicals were identified as COPCs in the risk assessments. The COPCs included 9 chlorination by-products (CBPs) regulated by USEPA National Primary Drinking Water Standards; 25 priority pollutants[2] (which may contain potential CBPs) regulated by the USA National Pollutant Discharge Elimination System (NPDES)[3]; and total residual chlorine (as disinfectant residue). The list of COPCs was presented in **Table 2**.

[1] Exposure pathways not associated with the HATS discharge, including normal dietary food (non-seafood), potable water consumption, incidental ingestion of soil and inhalation of contaminants in air (under ambient, indoor or workplace conditions), are not considered in the assessment.

[2] The 25 pollutants are regulated in NPDES due to their presence in industrial effluent but not their possible generation in chlorination process. However, a conservative approach is adopted to study all these regulated chlorinated organic substances, which are documented as potential CBPs, in US drinking water and wastewater discharge.

[3] The NPDES permit program controls water pollution by regulating point sources that discharge pollutants into water of the United States. Industrial, municipal, and other facilities must obtain permits if their discharges go directly to surface waters.

Table 2 List of Contaminants of Potential Concern

CBPs regulated by USEPA National Primary Drinking Water Standards	Priority Pollutants listed in NPDES Permit Application Testing Requirements (40 CFR 122, Appendix D, Tables II to V), which may contain CBPs	Disinfectant Residue
Chloroform	Methylene chloride	Total residual chloride
Bromodichloromethane	Carbon tetrachloride	
Dibromochloromethane	Chlorobenzene	
Bromoform	1,1-dichloroethane	
Chloroacetic acid	1,2-dichloroethane	
Bromoacetic acid	1,1-dichloroethylene	
Dibromoacetic acid	1,2-dichloropropane	
Dichloroacetic acid	Tetrachloroethylene	
Trichloroacetic acid	1,1,1-trichloroethane	
	1,1,2-trichloroethane	
	Trichloroethylene	
	2-chlorophenol	
	2,4-dichlorophenol	
	p-chloro-m-cresol	
	Pentachlorophenol	
	2,4,6-trichlorophenol	
	Bis(2-chloroethoxy)methane	
	1,4-dichlorobenzene	
	Hexachlorobenzene	
	Hexachlorocyclopentadiene	
	Hexachloroethane	
	1,2,4-trichlorobenzene	
	Alpha-benzene hexachloride	
	Beta-benzene hexachloride	
	Gamma-benzene hexachloride	

1.11 Unlike other conventional human health risk assessments for air pollution source (e.g. incinerator) and contaminated land/groundwater, a look-up table of contaminants/list of possible COPC for CBPs risk assessment in effluent was not identified from local and overseas authorities. Moreover, according to the review of local and overseas practice, list of "regulated CBPs in sewage effluent" was not identified.

1.12 Hence, a conservative approach was adopted in this Study to include all the regulated CBPs in drinking water plus the 25 priority pollutants (may contain potential CBPs) regulated by NPDES as COPCs, although these pollutants are not regulated due to the concern of generation during chlorination process.

1.13 The NPDES practice was adopted because it contains the most comprehensive list of regulated pollutants for effluent discharge, based on the review of practice in the USA, the United Kingdom, Australia, Canada, China and Hong Kong. Moreover, the purpose of NPDES is to ensure the US National Water Quality Criteria are complied by regulating pollutant concentrations in effluent discharge directly to surface water, in order to protect the human health and aquatic life.

1.14 Therefore, the 35 COPCs identified for the risk assessment include all documented potential CBPs/disinfectant residue which are regulated due to their potential to cause impact to human health and/or ecological resources. The list of identified COPCs (which the COCs for risk calculation were selected from the list) was considered sufficiently comprehensive to assess the potential risk to human health due to chronic exposure to the contaminants produced in the disinfection process in the effluent discharges.

1.15 Concerning the chemical species (sodium, bisulphite, sulphite and sulphate) associated with the dechlorination agent - sodium bisulphite, none of them was regulated by the current National Primary Drinking Water Regulations. Therefore, it was considered that the application of the dechlorination

agent would not cause significant impact to human health and the related chemical species were not identified as COPCs in the HHRA.

Selection of COC

1.16 The concentrations of the identified COPCs in chlorinated/dechlorinated (C/D) CEPT effluent from SCISTW (for assessment scenarios 1 to 4), secondary treated effluent from Shatin/Tai Po Sewage Treatment Works (for assessment scenario 5) and ambient seawater (2 sampling locations) were determined by chemical analysis works. The COC selection and determination of COC effluent concentrations for risk assessments were based on the chemical analysis results and the following rules.

Rules of COC Selection

Rule A – COPCs without relevant toxicity values, standards or criteria were not selected as COCs for risk assessments.

Rule B - COPCs detected in the C/D effluent were selected as COCs for risk assessment. The highest value from the replicates of analysis was chosen as the effluent concentrations to use in the risk assessment calculations.

Rule C – Non-detected COPCs with detection limit (for C/D effluent samples) exceeds the Concentration of Interest[4] (COI) were selected as COCs. For these COCs, effluent concentrations used in the risk assessments are one-half of the detection limit, which is a standard approach accepted by USEPA.

Rule D – COPCs with concentration in C/D effluent lower than the ambient seawater concentration were not selected as COCs.

Rules of COC Ambient Seawater Concentration Determination

Rule E – The highest COC concentrations found in the replicates of ambient seawater analysis were used to represent the background concentrations in the risk assessment calculations.

Rule F – For COCs that were not detected in the ambient seawater samples, the background concentration was set as zero.

1.17 Based on the chemical analysis results and above rules, COCs were selected for the risk assessments for Scenarios 1 to 4 and Scenario 5 and presented in **Tables 3** and **4** respectively.

Table 3 Results of COCs Selection for Scenarios 1 to 4

COPC	HHRA	Max. Conc. in C/D CEPT Effluent (µg/L)	Max. Conc. in Ambient Seawater (µg/L)	Note
Total residual chloride	Yes	100	0	
Chloroform	Yes	7	0	
Bromodichloromethane	Yes	<5	0	A
Dibromochloromethane	Yes	<5	0	A
Bromoform		<5	0	
Chloroacetic acid	Yes	4	0	
Bromoacetic acid		<2	0	B
Dibromoacetic acid	Yes	4	0	
Dichloroacetic acid	Yes	45.9	0	
Trichloroacetic acid	Yes	22	0	
Methylene chloride		<20	55	
Carbon tetrachloride		<0.5	0	
Chlorobenzene		<0.5	0	
1,1-dichloroethane		<0.5	0	

[4] The COIs for human health were the standards for drinking/tap water. The list of COIs are presented in Annex A.

COPC	HHRA	Max. Conc. in C/D CEPT Effluent (µg/L)	Max. Conc. in Ambient Seawater (µg/L)	Note
1,2-dichloroethane		<0.5	0	
1,1-dichloroethylene		<0.5	0	
1,2-dichloropropane		<0.5	0	
Tetrachloroethylene	Yes	1.3	0	
1,1,1-trichloroethane		<0.5	0	
1,1,2-trichloroethane		<0.5	0	
Trichloroethylene	Yes	2	0	
2-chlorophenol		<0.5	0	
2,4-dichlorophenol		<0.5	0	
p-chloro-m-cresol		<0.5	0	B
Pentachlorophenol	Yes	<2.5	0	A
2,4,6-trichlorophenol	Yes	2	0	
Bis(2-chloroethoxy)methane		<0.5	0	B
1,4-dichlorobenzene		<0.5	0	
Hexachlorobenzene		<0.5	0	
Hexachlorocyclopentadiene		<2.5	0	
Hexachloroethane		<0.5	0	
1,2,4-trichlorobenzene		<0.5	0	
Alpha-benzene hexachloride	Yes	<0.5	0	A
Beta-benzene hexachloride	Yes	<1	0	A
Gamma-benzene hexachloride	Yes	<1	0	A

Note: A) Detection limit exceeds the concentration of interest for human health
B) No available toxicity data for human health

Table 4 Results of COCs Selection for Scenario 5

COPC	HHRA	Max. Conc. in Secondary Treated Effluent (µg/L)	Max. Conc. in Ambient Seawater (µg/L)	Note
Total residual chloride		<20	0	
Chloroform	Yes	<5	0	A
Bromodichloromethane	Yes	<5	0	A
Dibromochloromethane	Yes	8	0	
Bromoform	Yes	49	0	
Chloroacetic acid		<2	0	
Bromoacetic acid		<2	0	B
Dibromoacetic acid	Yes	10	0	
Dichloroacetic acid	Yes	3	0	
Trichloroacetic acid	Yes	7	0	
Methylene chloride		<20	55	
Carbon tetrachloride		<0.5	0	
Chlorobenzene		<0.5	0	
1,1-dichloroethane		<0.5	0	
1,2-dichloroethane		<0.5	0	
1,1-dichloroethylene		<0.5	0	
1,2-dichloropropane		<0.5	0	
Tetrachloroethylene		<0.5	0	
1,1,1-trichloroethane		<0.5	0	
1,1,2-trichloroethane		<0.5	0	
Trichloroethylene		<0.5	0	
2-chlorophenol		<0.5	0	
2,4-dichlorophenol		<0.5	0	
p-chloro-m-cresol		<0.5	0	B
Pentachlorophenol	Yes	<2.5	0	A
2,4,6-trichlorophenol		<0.5	0	
Bis(2-chloroethoxy)methane		<0.5	0	B
1,4-dichlorobenzene		<0.5	0	

COPC	HHRA	Max. Conc. in Secondary Treated Effluent (µg/L)	Max. Conc. in Ambient Seawater (µg/L)	Note
Hexachlorobenzene		<0.5	0	
Hexachlorocyclopentadiene		<2.5	0	
Hexachloroethane		<0.5	0	
1,2,4-trichlorobenzene		<0.5	0	
Alpha-benzene hexachloride	Yes	<0.5	0	A
Beta-benzene hexachloride	Yes	<1	0	A
Gamma-benzene hexachloride	Yes	<1	0	A

Note: A) Detection limit exceeds the concentration of interest of human health
B) No available toxicity data for human health

Identification of Potential Human Receptors

1.18 As presented in the SCM for HHRA above, the completed and significant COC exposure pathways are incidental ingestion and dermal contact of seawater, and ingestion of contaminated seafood. Therefore, the potential human receptors (children and adult) are:

- People who swim or engage in other water related activities in the sea area which is contaminated by the selected COCs discharged from the outfall of SCISTW
- People who consume seafood which is contaminated by the selected COCs discharged from the outfall of SCISTW

Exposure Assessment

Human Health Risk Assessment

1.19 This phase of HHRA comprised the following tasks:

- Water quality modelling
- Exposure setting characterization, which consists of the following tasks:
  - Determine exposure points
  - Characterize potential human receptors
  - Calculate the COC exposure

Water Quality Modelling

1.20 The water quality modelling has been conducted in this assignment and the results obtained were used for the risk assessment.

Exposure Setting Characterization

Exposure Points Determination

1.21 For the pathway of incidental ingestion of seawater, three exposure points were identified: (1) the edge of the zone of initial dilution (ZID), which caters the individuals who accidentally drop into the harbour from ships; (2) the edge of the mixing zone and (3) the nearest beach from the SCISTW[5], which caters the individuals frequently bath or swim in the beach, having a potentially higher exposure to contaminated seawater. The identified exposure points are consistent with the previous studies.

1.22 There was no specific exposure point for the contaminated seafood consumption pathway.

[5] In terms of lowest outfall dilution factor calculated by water quality modelling rather than the shortest geological distance.

Potential Human Receptors Characterization

1.23 The following parameters were characterized for both children and adult human receptors:

- Exposure time, duration and frequency for each exposure pathway
- Contaminated water/seafood ingestion rate
- Body weight
- Averaging time

1.24 **Table 5** presented the parameter values of human receptors

Table 5 Parameter Values of Human Receptors

Parameter	Value	Unit
Exposure time for dropping from ships[6]	5	hr/d
Exposure frequency for dropping from ships[7]	1	d/yr
Swimming exposure time	2.6	hr/d
Swimming exposure frequency	124	d/yr
Swimming exposure duration - adult and adult fishermen	52	yr
Swimming exposure duration - children and fishermen children	18	yr
Body weight – adult and adult fishermen	60	kg
Body weight – children and fishermen children	32	kg
Incidental water ingestion rate	50	ml/hr
Averaging time – adult and adult fishermen	52	yr
Averaging time – children and fishermen children	18	yr
Seafood consumption rate – adult	148	g/d
Seafood consumption rate – children	79	g/d
Seafood consumption rate – adult fishermen[7]	300	g/d
Seafood consumption rate – fishermen children[8]	160	g/d
Seafood exposure duration – adult and adult fishermen	52	yr
Seafood exposure duration – children and fishermen children	18	yr
Seafood consumption frequency	350	d/yr
Skin surface area available for contact – adult[9]	20,000	cm^2
Skin surface area available for contact – children[10]	11,600	cm^2
Lifetime (for cancer risk calculation)	70	yr

COC Exposure Calculation

1.25 The COC exposure would be calculated by the following equations, which Equations 1 to 4 are adopted from HATS EEFS Ecological and Health Risk Assessment (2004), Equation 5 is adopted from USEPA (1999b), Equations 6 and 7 are based on the daily dermal intake equation documented in USEPA (1998). It is considered that swimming would be the water related activity with the highest rate of incidental water ingestion and dermal exposure, therefore swimming would be considered to be the representative water related activity for the exposure pathway of incidental ingestion and dermal contact of seawater.

[6] Conservative values are assumed for the purpose of risk assessment.
[7] Adopted from ERM (2005).
[8] Calculated based on the ratio of seafood consumption rate between adult and children and the seafood consumption rate of fishermen adult. Therefore, seafood consumption rate of fishermen children = (79/148 x 300) g/d
[9] Adopted from USEPA (1992).
[10] Adopted from USEPA (1992).

Non-carcinogen exposure via incidental ingestion of seawater (children or adult)

$ADI_{iw} = (C_{iw} \times IR_w \times ET \times EF \times ED \times 0.001L/ml) / (BW \times AT \times 365\ d/yr)$ Equation 1

Where
ADI_{iw} = average daily COC *i* intake via incidental ingestion of seawater (mg/kg-d)
C_{iw} = COC *i* concentration in water (mg/L)
IR_w = incidental water ingestion rate (ml/hr)
ET = exposure time (hr/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
BW = body weight (kg)
AT = averaging time (yr)

Non-carcinogen exposure via consumption of seafood (children or adult)

$ADI_{is} = (C_{is} \times IR_s \times EF \times ED \times FI \times 0.001kg/g) / (BW \times AT \times 365\ d/yr)$ Equation 2

Where
ADI_{is} = average daily COC *i* intake via consumption of seafood (mg/kg-d)
C_{is} = COC *i* concentration in seafood (mg/kg)
IR_s = seafood consumption rate (g/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
FI = fraction of seafood from ZID (unitless) = ZID area / 1800km^2 (total area for fishing)[11]
BW = body weight (kg)
AT = averaging time (yr)

Carcinogen exposure via incidental ingestion of seawater

$$LADD_{iw} = \frac{\frac{[(C_{iw} \times IR_w \times ET \times EF \times ED \times 0.001L/ml)_{adult}]}{BW_{adult}} + \frac{[(C_{iw} \times IR_w \times ET \times EF \times ED \times 0.001L/ml)_{child}]}{BW_{child}}}{LT \times 365\ d/yr}$$

Equation 3

Where
$LADD_{iw}$ = lifetime average daily COC *i* dose via incidental ingestion of seawater (mg/kg-d)
C_{iw} = COC *i* concentration in water (mg/L)
IR_w = incidental water ingestion rate (ml/hr)
ET = exposure time (hr/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
BW = body weight (kg)
LT = lifetime (yr)

[11] Adopt from SSDS/EIAS DRA (1998) and HATS EEFS E&HRA (2004).

Carcinogen exposure via consumption of seafood (children or adult)

$$LADD_{is} = \frac{\frac{[(C_{is} \times IR_s \times EF \times ED \times FI \times 0.001kg/g)_{adult}]}{BW_{adult}} + \frac{[(C_{is} \times IR_s \times EF \times ED \times FI \times 0.001kg/g)_{child}]}{BW_{child}}}{LT \times 365\ d/yr}$$

Equation 4

Where
$LADD_{is}$ = lifetime average daily COC *i* dose via consumption of seafood (mg/kg-d)
C_{is} = COC *i* concentration in seafood (mg/kg)
IR_s = seafood consumption rate (g/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
FI = fraction of seafood from ZID (unitless)
BW = body weight (kg)
LT = lifetime (yr)

COC Concentration in Contaminated Seafood

1.26 With reference to HATS EEFS Ecological and Health Risk Assessment (2004), it is assumed that all seafood consumed by human receptor is fish and the same assumption is adopted in this HHRA. Therefore, water-to-fish bioconcentration factor is used in the below equation for calculation of COC concentration in seafood.

$$C_{is} = C_{iw} \times BCF_i \times FCM_i$$

Equation 5

Where
C_{is} = COC *i* concentration in seafood (mg/kg)
BCF_i = water-to-fish bioconcentration factor for COC *i* (L/kg)
FCM_i = food chain multiplier of COC *i* (unitless)

Non-carcinogen exposure via dermal contact of seawater (children or adult)

$$DDI_{id} = (D_{ievent} \times EF \times ED \times A_s) / (BW \times AT \times 365\ d/yr)$$

Equation 6

Where
DDI_{id} = average daily COC *i* intake via dermal contact of water (mg/kg-d)
D_{ievent} = dermally absorbed dose per event for COC *i* (mg/cm^2-event)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
A_s = skin surface area available for contact (cm^2)
BW = body weight (kg)
AT = averaging time (yr)

Carcinogen exposure via dermal contact of seawater

$$LADD_{id} = \frac{\frac{[(D_{ievent} \times EF \times ED \times A_s)_{adult}]}{BW_{adult}} + \frac{[(D_{ievent} \times EF \times ED \times A_s)_{child}]}{BW_{child}}}{LT \times 365\ d/yr}$$

Equation 7

Where
$LADD_{id}$ = lifetime average daily COC *i* dose via dermal exposure of seawater (mg/kg-d)
D_{ievent} = dermally absorbed dose per event for COC *i* (mg/cm^2-event)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
A_s = skin surface area available for contact (cm^2)
BW = body weight (kg)
LT = lifetime (yr)

For organic substances, D_{ievent} will be calculated using the equations below:

If $t_{event} < t^*$, then $D_{ievent} = 2x\ Kp \times C_{iw}(6 \times T \times t_{event})^{1/2} / 1000$ Equation 8a

If $t_{event} > t^*$, then $D_{ievent} = Kp \times C_{iw} \times [(t_{event} / (1+B)) + 2 \times T\ (1+3B / 1+B)] / 1000$ Equation 8b

Where
Kp = permeability coefficient from water for contaminant (cm/hr)
C_{iw} = contaminant *i* concentration in water (mg/L)
t_{event} = duration of event (hr/event)
T = lag time (hr)
T* = time to reach steady-state (hr)
B = parameter to describe relative contribution of permeability coefficients in stratum corneum and viable epidermis

For inorganic substances, D_{ievent} will be calculated using the equation below:

$D_{ievent} = (2 \times Kp \times C_{iw} \times t_{event}) / 1000$ Equation 8c

1.27 A number of variables in the above equations needed to be defined for the exposure assessment. For COC concentrations at exposure points, they were determined by water quality modelling. The simulation periods for water quality modelling covered two 15-day full spring-neap cycles for dry and wet seasons respectively. The dry and wet seasons results were averaged to represent the annual mean results, which were used for exposure calculation.

1.28 Other defined variables were presented in **Tables 6** and **7**.

Table 6 Bioconcentration Factor and Food Chain Multiplier of COC

COC	Water-to-fish Bioconcentration Factor	FCM[a]
Total residual chlorine	N/A	N/A
Bromoform	13.3[b]	1.0
Bromodichloromethane	8.26[b]	1.0
Chloroform	6.92[b]	1.0
Dibromochloromethane	10.4[b]	1.0
Chloroacetic acid	0.26[c]	1.0
Dibromoacetic acid	0.82[c]	1.0
Dichloroacetic acid	1.13[c]	1.0
Trichloroacetic acid	5.75[c]	1.0
Tetrachloroethylene	82.8[b]	1.0
Trichloroethylene	14.1[b]	1.0
Pentachlorophenol	671[b]	3.2
2,4,6-trichlorophenol	56.1[b]	1.0
Alpha-BHC	168[b]	1.0
Beta-BHC	168[b]	1.0
Gamma-BHC	168[d]	1.0

N/A: Not Available

Note: [a] FCMs were developed using K_{ow} values reported in USEPA (1995), as in USEPA (1999b).
[b] BCF values documented in USEPA (2005).
[c] No recommended BCF value identified. Regression equation was used to calculate the BCF values (Bintein *et al.* (1993), as in USEPA (1999b)).
[d] Same BCF adopted from isomer.

Table 7 Parameters related to Dermal Exposure

COC	Kp (cm/hr)	T (hr)	t* (hr)	B
Total residual chlorine[a]	1E-3	-	-	-
Bromoform[a]	2.6E-3	3	7.3	2.3E-2
Bromodichloromethane[a]	1.3E-1	4.7E-1	1.1	9.3E-3
Chloroform[a]	5.8E-3	8.7E-1	2.1	1.2E-2
Dibromochloromethane[a]	3.9E-3	1.6	3.9	1.7E-2
Chloroacetic acid[b]	7.24E-4	3.3E-1	0.8	1.7E-4
Dibromoacetic acid[b]	3.15E-4	1.89	4.5	5.9E-4
Dichloroacetic acid[b]	1.40E-3	5.3E-1	1.3	8.3E-4
Trichlroacetic acid[b]	3.09E-3	8.9E-1	2.1	5E-3
Tetrachloroethylene[a]	3.7E-1	9E-1	4.3	2.5E-1
Trichloroethylene[a]	2.3E-1	5.5E-1	1.3	2.6E-2
Pentachlorophenol[a]	6.5E-1	3.7	1.7E+1	7.2E+1
2,4,6-trichlorophenol[a]	5.9E-2	1.4	9.2	4.9E-1
Alpha-BHC[b]	0.016	5.19	36.7	0.60
Beta-BHC[b]	0.015	5.19	34.8	0.52
Gamma-BHC[a]	0.014	5.20	35.0	0.52

Note: [a] parameter values were adopted from USEPA (1992).
[b] No recommended values documented, values were calculated using equations documented in USEPA (1992).

Dose-response Assessment

1.29 This stage of HHRA involved determination of the relationship between the contaminant doses from exposure and corresponding response in humans (risk of cancer development, in terms of cancer slope factor and/or non-cancer health impact, in terms of reference dose). This relationship for various contaminants is documented in database/publications in authorities such as US Environmental Protection Agency (USEPA) and World Health Organization (WHO).

1.30 The Cancer Slope Factor (CSF) and reference dose of the COCs adopted in World Health

Organization (WHO) and USEPA[12] were presented in **Table 8**. More stringent value was typed in bold adopted. For the identified COCs, adjustment of oral toxicity data (cancer slope factor and/or reference dose) for calculation of the risk/hazard due to absorbed doses was not needed according to USEPA (2001b). Therefore, the oral cancer slope factor and reference dose selected for oral exposure were used for the risk calculation in dermal exposure pathway.

Table 8 Cancer Slope Factor and Reference Dose of COCs

COC	Cancer Slope Factor (oral, $(mg/kg/d)^{-1}$)		Reference Dose (µg/kg/d)	
	WHO	USEPA	WHO	USEPA
Bromoform	N/A	**7.9E-3**[a]	25[b]	**20**[a]
Bromodichloromethane	5.0E-3[c]	**6.2E-2**[a]	N/A	**20**[a]
Chloroform	N/A	Note d	**10**[b]	**10**[a]
Dibromochloromethane	N/A	**8.4E-2**[a]	30[b]	**20**[a]
Chloroacetic acid	N/A	N/A	N/A	**2**[f]
Dibromoacetic acid	N/A	N/A	**20**[b]	N/A
Dichloroacetic acid	N/A	**5E-2**[a]	40[b]	**4**[a]
Trichloroacetic acid	N/A	N/A	**40**[b]	N/A
Total residual chlorine	N/A	N/A	150[b]	**100**[a]
Tetrachloroethylene	N/A	N/A	14[c]	**10**[a]
Trichloroethylene	N/A	N/A	**23.8**[c]	N/A
Pentachlorophenol	N/A	**1.2E-1**[a]	N/A	**30**[a]
2,4,6-trichlorophenol	N/A	**1.1E-2**[a]	N/A	N/A
Alpha-BHC	N/A	**6.3**[a]	N/A	N/A
Beta-BHC	N/A	**1.8**[a]	N/A	N/A
Gamma-BHC	N/A	N/A	5[c]	**0.3**[a]

Note: N/A: Not Available
[a] Source: USEPA IRIS Database
[b] Source: WHO (2000)
[c] Source: WHO (2004b)
[d] According to Integrated Risk Information System (IRIS) database, a dose of 0.01mg/kg/d can be considered protective against cancer risk.
[e] According to WHO (2004a), the available data are inadequate to establish guideline values for the chemical.
[f] Health Effects Assessment Summary Tables (HEAST) as reported in The Risk Assessment Information System.

Risk/Hazard Characterization

1.31 There were 2 types of risk/hazard to be characterized in HHRA, as follows:

- Cancer risk, from exposure to identified carcinogenic COCs
  - The lifetime individual excess cancer risk can be calculated by the following equation:

$$\text{Cancer Risk}_i = \text{LADD}_i \times \text{CSF}_{oral(i)} \qquad \text{Equation 9}$$

Where
Cancer Risk_i = incremental probability that an individual will develop cancer over a lifetime as a result of a specific exposure to carcinogenic COC *i*
$\text{CSF}_{oral(i)}$ = oral cancer slope factor for COC *i*

$$\text{Cancer Risk}_T = \Sigma \text{ Cancer Risk}_i \qquad \text{Equation 10}$$

Where
Cancer Risk_T = total cancer risk for exposure to all identified carcinogenic COCs via a specific exposure pathway

[12] In SSDS/EIAS DRA (1998), values adopted from National Health and Medical Research Council and Agricultural and Resource Management Council of Australia and New Zealand (NHMRC) were also compared. However, cancer slope factor and reference dose for the COCs were not identified in NHMRC (2004).

$$\text{Total Cancer Risk} = \Sigma \text{ Cancer Risk}_T \qquad \text{Equation 11}$$

Where
Total Cancer Risk = total cancer risk for exposure to all identified carcinogenic COCs via all identified exposure pathways

From Equations 9 to 11, the lifetime incremental cancer risk due to exposure of all identified carcinogenic COCs via the pathways "ingestion of seawater", "dermal contact of water" and "consumption of contaminated seafood" can be calculated.

- Non-cancer effect health hazard, from exposure to identified COCs imposing non-carcinogenic health effects
  - The Hazard Quotient (HQ) can be calculated by the following equation:

$$HQ_i = ADI_i / RfD_i \qquad \text{Equation 12}$$

Where
HQ_i = hazard quotient for COC *i*
ADI_i = average daily COC *i* intake
RfD_i = reference dose for COC *i*

$$HI_i = \Sigma HQ_i \qquad \text{Equation 13}$$

Where
HI_i = Hazard Index, total hazard attributable to exposure to all identified COCs through a single exposure pathway

$$\text{Total HI} = \Sigma HI_i \qquad \text{Equation 14}$$

Where
Total HI = Total hazard index from multiple pathways

From Equations 12 to 14, the total hazard index for both children and adult human receptor due to exposure of all identified COCs imposing non-carcinogenic effect via the pathways "ingestion of seawater", "dermal contact of water" and "consumption of contaminated seafood" can be calculated.

Output of Risk Assessment

1.32 The output of the HHRA are listed as follows:

- Lifetime incremental cancer risk due to exposure of identified carcinogenic COCs (contributed by both HATS effluent and "background" COC concentrations existing in ambient seawater) by incidental exposure to seawater at edge of ZID and consumption of contaminated seafood
- Lifetime incremental cancer risk due to exposure of identified carcinogenic COCs (contributed by both HATS effluent and "background" COC concentrations existing in ambient seawater) by swimming activity at edge of mixing zone and consumption of contaminated seafood
- Lifetime incremental cancer risk due to exposure of identified carcinogenic COCs (contributed by both HATS effluent and "background" COC concentrations existing in ambient seawater) by swimming activity at the nearest beach from SCISTW outfall and consumption of contaminated seafood
- Total health hazard index due to exposure of identified COCs (contributed by both HATS effluent and "background" COC concentrations existing in ambient seawater) by incidental exposure to seawater at edge of ZID and consumption of contaminated seafood
- Total health hazard index due to exposure of identified COCs (contributed by both HATS effluent and "background" COC concentrations existing in ambient seawater) by swimming activity at edge of mixing zone and consumption of contaminated seafood
- Total health hazard index due to exposure of identified COCs (contributed by both HATS effluent and "background" COC concentrations existing in ambient seawater) by swimming activity at the nearest beach from SCISTW outfall and consumption of contaminated seafood

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