Risk assessment of human health from exposure to the discharged ballast water after full-scale electrolysis treatment

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Abstract: The presence of disinfection by-products (DBPs) releasing from ballast water management systems (BWMS) can cause a possible adverse effects on humans. The objectives of this study were to compute the Derived No Effect Levels (DNELs) for different exposure scenarios and to compare these levels with the exposure levels from the measured DBPs in treated ballast water. The risk assessment showed that when using animal toxicity data, all the DNELs values were approximately 10^3 - 10^{12} times higher than the exposure levels of occupational and general public exposure scenarios, indicating the level of risk was low (risk characterization ratios (RCRs) < 1). However, when using human data, the RCRs were higher than 1 for dichlorobromomethane and trichloromethane, indicating that the risk of adverse effects on human were significant. This implies that there are apparent discrepancies between risk characterization from animal and human data, which may affect the overall results. We therefore recommend that when appropriate, human data should be used in risk assessment as much as possible, although human data are very limited. Moreover, more appropriate assessment factors can be considered to be employed in estimating the DNELs for human when the animal data is selected as the dose descriptors.

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1. Introduction

The introduction of invasive marine species into new environments via ships' ballast water has been identified as one of the greatest threats to the world's oceans (Bax et al., 2003; Ricciardi, 2006; Molnar et al., 2008; Hulme, 2009; Bradie et al., 2010; Carbona et al., 2010; Bailey et al., 2011). Upon discharge during de-ballasting, nonindigenous invasive species (NIS) may develop and become established in the new receiving environment with high propensity, causing serious ecological problems (Briski et al., 2013; Pam et al., 2013). Many experts consider invasive marine species to pose a greater threat to the environment than oil pollution (North of England P&I Association, 2009). Furthermore, aquatic NIS are a significant and growing contributor to the global spread of red tide blooms (Briski et al., 2013), and cause enormous ecological and economic damage (Ruiz et al., 2000). To reduce the risk of ballast water introductions, the International Maritime Organization (IMO) has proposed discharge standards limiting the maximum concentrations of living organisms (Miller et al., 2011). As a result, developers and manufactures have been producing onboard ballast water management systems (BWMS).

The predominant technologies for ballast water treatment generally consist of filtration plus electrolysis, UV irradiation, cavitation and/or ozonation (Lloyd's Register, 2012). Both electrolysis and ozonation are highly effective for the removal of harmful organisms; however, the majority of these systems generate disinfection by-products (DBPs) in varying amounts (Banerji et al., 2012). Several DBPs have been proven to be carcinogens and mutagens to humans (Wang et al., 2013; Delacroix et al., 2013). Consequently, humans may be exposed to treated ballast water containing DBPs in the workplace (occupational exposure) or from the use of consumer products (consumer exposure) and indirectly via the environment (Banerji et al., 2012; Werschkun et al., 2012). Many research studies have paid attention to the human

health risks posed by trihalomethanes (THMs) in drinking water (Wang et al., 2007; Basu et al., 2011). The US Environmental Protection Agency (EPA) and the World Health Organization (WHO) have established regulations for DBPs (e.g., THMs, haloacetic acids (HAAs)) control in drinking water.

However, specific discharge standards limiting are unavailable for DBPs in treated ballast water. Although the exposure level of treated ballast water is much lower than drinking water, seawater generally has higher brominated DBPs formation potentials, most likely because the amounts of bromide ion and natural organic substances are higher than those in drinking water (Delacroix et al., 2013). Echardt and Kornmuller (2009) reported that the concentrations of THMs, HAAs and bromate generated by electrolytic chlorination BWMS were as high as 338.25, 69.64 and 55.25 µg/L, respectively. For ozone-based BWMS, the concentration of bromate was as high as 490 µg/L when the total residual oxidant (TRO) level was 6.4 mg/L as Cl₂ in seawater (Maritime Environment Protection Committee, 2012), which is 49 times higher than the WHO guideline value of 10 µg/L (World Health Organization, 2011).

According to Lloyd's Register (2012), 29% of the technologies are based on electrolysis; while 27% are applying UV irradiation as the main disinfection process and 44% other technologies are based on ozonation, deoxygenation, heating and nonchlorine chemical disinfection (Delacroix et al., 2013). This means that electrolysis-based BWMS are dominant technologies in this market. Biological inactivation by electrolysis-based BWMS achieved the D-2 performance standard of the Ballast Water Convention (i.e., discharging less than 10 viable organisms ≥50 µm per m³; less than 10 viable organisms ≥10 to <50 µm per mL; less than 1 colony forming unit (cfu) of toxicogenic *Vibrio cholerae* (O1 and O139) per 100 mL; less than 250 cfu of *Escherichia coli* per 100 mL; and less than 100 cfu of intestinal Enterococci per 100 mL) (International Maritime Organization, 2004) when the TRO level was approximately 7.5-9.5 mg/L as Cl₂ (Maritime Environment Protection Committee, 2009). In addition, aquatic toxicity tests for organisms of three trophic levels were conducted to confirm that there were no detectable toxic effects on algae, invertebrates, or fish, indicating that the effects of treated ballast water were acceptable when discharged (Maritime Environment

Protection Committee, 2009). According to IMO G9 procedure (International Maritime Organization, 2008a), electrolysis-based BWMS have to provide human health risk assessment, and many of these health risk assessments provided limited toxicological information on many DBPs or used only animal toxicity data required for effects assessment (Banerji et al., 2012).

To further verify the health acceptability of electrolysis-based BWMS, this study was conducted to evaluate the risk to human health associated with occupational and general public exposures due to operation of the electrolysis-based BWMS. This study followed a risk assessment procedure that consisted of four basic steps, hazard identification, dose-response assessment, exposure assessment, and risk characterization. Briefly, the first step was to identify the potential DBPs in treated ballast water at discharge. The Derived No Effect Levels (DNELs) of the measured DBPs were then derived on the basis of available dose descriptors from human epidemiology and/or animal toxicity studies such as the No Observed Adverse Effect Level (NOAEL). We next linked the measured DBPs levels in different exposure scenarios with risk. In the interim, MAMPEC-BW (Marine antifouling model to predict environmental concentrations-ballast water) model version 3.0 was used to calculate the predicted environmental concentrations (PECs) of the measured DBPs. Finally, the acceptable levels of risk for human health were determined by comparing the exposure levels to various DNELs.

2. Materials and methods

2.1. Test water

According to the IMO Guidelines, brackish water was tested to verify BWMS efficiency in a wide salinity range (International Maritime Organization, 2008b). During the experiment, brackish water (salinity 3-32 PSU) was achieved by mixing natural seawater (Qingdao, China) with municipal tap water. The initial salinity, temperature and pH of the influent brackish water

were 21.3 PSU, 12.6 °C and 8.1, respectively. To meet the required water quality indexes of the IMO Guidelines ([International Maritime Organization, 2008b](#)), commercial humic acid, globigerina and kieselguhr were added to make the concentrations of dissolved organic carbon (DOC), particulate organic carbon (POC) and total suspended solid (TSS) of 6.14, 5.41 and 73.3 mg/L, respectively. Compared to natural seawater (DOC = 3.82 mg/L; POC = 2.29 mg/L; TSS = 2.15 mg/L), water quality conditions of brackish water was considered to be relatively bad due to high DOC, POC and TSS contents. Plankton densities were adjusted by adding concentrated natural populations from the harbor into the influent water; the target densities for organisms $\geq 50 \mu\text{m}$ and ≥ 10 to $< 50 \mu\text{m}$ were at least 10^5 individuals (ind.)/ m^3 and 10^3 cells/mL, respectively ([International Maritime Organization, 2008b](#)).

2.2. Identification/description of unit operations in the BWMS and experimental procedure

To perform human health risk assessment for occupational exposure, a description of the ballast water treatment process associated with the system as a set of unit operations is a basic step to identify which individual system components are likely to lead to human exposure to the BWMS related DBPs ([International Maritime Organization, 2012](#)). The system regarded as a set of unit operations based on the treatment process is shown in [Figure 1](#). Filtration unit (1) and Sampling unit (5) were set in the engine room; Electrolysis unit (2) and Neutralization unit (4) were installed in the equipment room; and the BWMS were operated automatically in cargo control station. During ballasting, the influent brackish water with a flow rate of $250 \text{ m}^3/\text{h}$ was filtered using an automatic self-cleaning filter to remove particles and organisms larger than $50 \mu\text{m}$, after which a side stream of filtered seawater (approximately $5.5 \text{ m}^3/\text{h}$ of $250 \text{ m}^3/\text{h}$) was subjected to an inactivation step employing an electrolytic cell where active substances (mainly sodium hypochlorite and sodium hypobromite) were produced, and then was injected back into the main ballast stream after degas. The range of TRO concentration measured by TRO analyzer 1 (CLX-Xt, HF Scientific, USA) was 8.0-9.5 mg/L as Cl_2 ([Figure 1](#)). During de-ballasting, the filter and the electrolytic cell were by-passed.

Treated ballast water was neutralized using sodium thiosulphate ($\text{Na}_2\text{S}_2\text{O}_3$) upon discharge if the TRO content measured by TRO analyzer 2 (CLX-X, HF Scientific, USA) was larger than 0.2 mg/L as Cl_2 (Figure 1). The present study was conducted to measure 25 types of DBPs (Table S1, Supporting Information). According to Guideline G8 of the IMO (International Maritime Organization, 2008b), treated ballast water was stored in the tank for at least 5 days. Chemical analysis for DBPs in treated ballast water were carried out immediately after treatment (day 0), as well as each day over the 5-day storage period. Two sampling points were arranged as shown in Figure 1. Treated water samples were collected from SP1 immediately after treatment, while samples of discharged water were collected from SP2. Three replicate samples of influent and/or treated water at discharge were collected over the period of uptake and/or discharge (e.g., beginning, middle, end). All samples were poured into 1-L sample containers, preserved with $\text{Na}_2\text{S}_2\text{O}_3$ to quench the TRO, placed in a cooler, returned to the laboratory, and stored at 4 °C in the dark until analysis.

2.3. Analytical methods

The viability of the plankton was qualified by automatic microscope (Olympus BX61, Japan) according to the specification for marine monitoring Part 7 of National Standards of Peoples Republic of China: ecological survey for offshore pollution and biological monitoring (GB 17378.7, 2007). Heterotrophic bacteria were enumerated using the agar spread plate method with autoclaved marine broth 2216E. The culture dishes were then closed and placed upside down in a $37 \pm 0.5^\circ\text{C}$ incubator for 48 h (GB 17378.7, 2007; GB 5750.12, 2006). *Escherichia coli* were cultivated in an incubator at $44 \pm 0.5^\circ\text{C}$ for 24 h according to GB 17378.7 (2007) and GB 5750.12 (2006). Intestinal enterococci were cultivated at temperature of $35 \pm 2^\circ\text{C}$ and incubated for 24 h based on SN/T 1933.1 (2007). According to GB 17378.7 (2007) the total number of *Vibrio* sp. was determined by placing the filter on TCBS cholera medium agar plates which were incubated at 37°C for 18 h. Physiological and biochemical test kits (API 20E) were used for further identification.

Ion chromatographic method performed on an ion chromatograph system (DIONEX ICS-1500, Thermo Fisher Scientific Inc., United States) was applied to determine bromate based on US EPA method 317.0 (US EPA, 2001). According to US EPA methods 524.2 (US EPA, 1995a), 551.1 (US EPA, 1995b), and 552.3 (US EPA, 2003), halogenated hydrocarbon, HANs, and HAAs were analyzed using a gas chromatograph (Agilent 7890A, Agilent Technologies Inc., United States) with mass spectrometer (Agilent 5975C, Agilent Technologies Inc., United States) and electron capture detection. TRO was determined by a colorimetric DPD (N,N-diethyl-p-phenylene diamine) method based on US EPA method 330.5 (US EPA, 1978). An iodometric method was used to determine $\text{Na}_2\text{S}_2\text{O}_3$ as a neutralizer in seawater.

2.4. Exposure assessment

For worker exposure in the ballast water tank, the concentration of the measured chemicals that might be transferred from the aquatic environment into the atmosphere was determined according to Equation 1:

$$C_{\text{air}} = H \cdot C_{\text{water}} / (R \cdot T) \quad (1)$$

where C_{air} = the concentration in air (mg/m^3), H = Henry's law constant ($\text{Pa} \cdot \text{m}^3/\text{mol}$), C_{water} = the concentration in the treated water ($\mu\text{g}/\text{L}$), R = the gas constant ($8.314 \text{ Pa} \cdot \text{m}^3/(\text{mol} \cdot \text{K})$), and T = the absolute temperature (K).

For exposure situations regarding the general public, the concentrations of the measured chemicals after discharging into the harbor were calculated using the MAMPEC-BW model. These values were used to compute C_{air} for general public inhalation exposure based upon Equation 1. The potential uptake of chemicals by humans through the various routes of exposure was determined according to Equation 2:

$$C_{\text{fish}} = \text{BCF} \cdot C_{\text{MAMPEC-BW}} \quad (2)$$

where C_{fish} = the concentration in fish ($\mu\text{g}/\text{kg}$), BCF = the bioconcentration factor (L/kg), and $C_{\text{MAMPEC-BW}}$ = the concentration of chemicals in the water calculated from the MAMPEC-BW

model ($\mu\text{g/L}$).

To quantify exposure level, the European Chemicals Agency (ECHA) provided guidance documents, from which six main equations were used to quantify the exposure to a chemical in treated ballast water via three possible exposure routes (inhalation, dermal and oral) (European Chemicals Agency, 2010a). Table 1 shows the six main equations and the exposure routes associated with occupational and general public exposures along with assumed values of each parameter used to estimate exposure levels.

Table 1. Quantitative formula of the exposure levels and the Derived No Effect Levels (DNELs)

Exposure routes	Equations	Exposure routes	Equations
Occupational exposure		Oral	$\text{DNEL}_{\text{hum-oral}} =$
Inhalation	$\text{EL}_{\text{inh}} = C_{\text{air}} \cdot t_{\text{exp}} \cdot \text{IR} / \text{BW}$ (3)		$[\text{dose descriptor}] \cdot \frac{\text{CF}_{\text{dr}}}{\text{ASF} \cdot \text{OSF} \cdot \text{ISF} \cdot \text{DSF}}$ (10)
Dermal	$\text{EL}_{\text{der}} = C_{\text{water}} \cdot A_{\text{hands/body}} \cdot \text{TH}_{\text{der}} \cdot \text{BIO}_{\text{der}} / \text{BW}$ (4)	Dermal	$\text{DNEL}_{\text{hum-der}} = \text{DNEL}_{\text{hum-oral}}$ (11)
General public exposure		Inhalation	
Inhalation	$\text{EL}_{\text{inh}} = C_{\text{air}} \cdot \text{IR} \cdot n \cdot t_{\text{exp}} \cdot \text{BIO}_{\text{inh}} / \text{BW}$ (5)	Worker	$\text{DNEL}_{\text{hum-inh}} =$
Dermal	$\text{EL}_{\text{der}} = C_{\text{MAMPEC-BW}} \cdot A_{\text{body}} \cdot \text{TH}_{\text{der}} \cdot n \cdot \text{BIO}_{\text{der}} / \text{BW}$ (6)		$\frac{[\text{dose descriptor}]}{\text{sRV}_{\text{ani}} \cdot \text{OSF} \cdot \text{ISF} \cdot \text{DSF}} \cdot \frac{24}{t_{\text{exp}}} \cdot \frac{\text{sRV}_{\text{hum-ba}}}{\text{wRV}_{\text{hum}}}$ (12)
Oral	$\text{EL}_{\text{oral}} = C_{\text{MAMPEC-BW}} \cdot \text{IR}_{\text{swim}} \cdot n \cdot t_{\text{exp}} \cdot \text{BIO}_{\text{oral}} / \text{BW}$ (7)	General public	$\text{DNEL}_{\text{hum-inh}} =$
Eating fish	$\text{EL}_{\text{fish}} = \text{QFC} \cdot C_{\text{fish}} \cdot \text{BIO}_{\text{oral}} / \text{BW}$ (8)		$\frac{[\text{dose descriptor}]}{\text{sRV}_{\text{ani}} \cdot \text{OSF} \cdot \text{ISF} \cdot \text{DSF}} \cdot \frac{24}{t_{\text{exp}}} \cdot \frac{\text{sRV}_{\text{hum-ba}}}{\text{sRV}_{\text{hum-li}}}$ (13)

Notes:

(1) Based on the ECHA guidance documents (European Chemicals Agency, 2010a), the value of each parameter was used as follows: EL = exposure level; IR = 1.25 m³/h (inhalation rate); t_{exp} = case by case (exposure duration); BW = 60 kg (body weight); A_{hands} = 0.168 m² (surface area of two hands); A_{body} = 1.94 m² (surface area of whole body being exposed to water); TH_{der} = 0.0001 m (thickness of the product area on the skin); BIO_{der} = 1 (dermal bioavailability); BIO_{inh} = 1 (fraction of chemical absorbed through the lungs); BIO_{oral} = 1 (oral bioavailability); n = 5 (number of swims per day); IR_{swim} = 0.025 L/h (ingestion rate of water while swimming); QFC = 0.188 kg/d (quantity of fish consumed per day).

(2) Based on the ECHA guidance documents (European Chemicals Agency, 2010b), the value of each parameter was used as follows: ASF = 4 (rat), 7 (mouse), and 1.4 (dog); OSF = 2.5; ISF = 5 (worker) and 10 (general public); DSF = 2 (sub-chronic to chronic exposure); CF_{dr} = 5/7 (acute exposure), 225/365 (chronic exposure), 14/365 (swimming), and 1 (eating fish); t_{exp} = case by case (exposure duration); sRV_{rat} = 1.15 m³/kg (standard 24 h respiratory volume of a rat), $\text{sRV}_{\text{hum-ba}}$ = 20 m³/60 kg (standard 24 h respiratory volume of a human doing basic activity), wRV_{hum} = 30 m³/60 kg (standard 24 h respiratory volume of a worker), and $\text{sRV}_{\text{hum-li}}$ = 30 m³/60 kg (standard 24 h respiratory volume of a human doing light activity).

2.5. Dose-response assessment

Dose-descriptors of the DBPs used in the present study were obtained from previously conducted human epidemiology and/or animal toxicity studies (e.g., rat, mouse and dog). The observed adverse health effects were generally grouped as short-term and repeated-dose toxicities. When using animal toxicity data, the dose descriptors for the threshold effect (e.g., NOAEL_{ani}) were converted into the correct starting point (e.g., corrected NOAEL_{hum}) to evaluate the risk level for humans. Route-to-route extrapolation as an alternative method was used to obtain the appropriate dose descriptor. Additionally, assessment factors (AFs) were used to address the differences between experimental conditions and actual field conditions, taking into account the uncertainties associated with the extrapolation procedure and in the available data set (European Chemicals Agency, 2010b). Specifically, five AFs were taken into account during calculation of the DNELs: the allometric scaling factor (ASF) to reflect the differences between the body weight of humans and animals; other interspecies scaling factors (OSF) to reflect the perceived toxicokinetic differences between species; intraspecies scaling factor (ISF) to reflect the differences between groups of humans due to the sensitivity to toxic insult; duration scaling factor (DSF) to reflect the difference in the exposure duration; and the dosing regimen correction factor (CF_{dr}) to correct the duration of dosing regimens (European Chemicals Agency, 2010b).

Based on the dose-descriptor values, the DNELs for further risk assessment were determined by applying AFs as follows:

$$\text{DNELs} = [\text{dose descriptor}]/\text{AFs} \quad (9)$$

where DNELs are in mg/kg for short-term oral and dermal exposure, mg/m³ for short-term inhalation exposure, mg/kg/d for long-term oral and dermal exposure, and mg/m³/d for long-term inhalation exposure. Table 1 provides the assumed value of each parameter used to calculate the DNELs.

2.6. Risk characterization

The risk to human health, which was defined as risk characterization ratios (RCRs) in this

study, was evaluated by a comparison of the exposure scenario for the proposed patterns of use and the DNELs of the measured DBPs. The equation used to accomplish this was as follows:

$$\text{RCRs} = [\text{Exposure level}]/\text{DNELs} \quad (14)$$

Comparison of this measured or estimated exposure level with an exposure limit was then used to judge whether the situation was satisfactory or if risk management measures were required. If the RCR was < 1 , the risk of exposure was considered acceptable, while an RCR > 1 indicated that there was a need for risk control.

3. Results and discussion

3.1. Plankton and bacteria inactivation

A summary of inactivation of plankton and bacteria is shown in [Table S2 \(Supporting Information\)](#). Results indicated that sufficient inactivation of plankton and bacteria in the treated water were achieved with an initial TRO level of around 9.5 mg/L as Cl_2 . The mean densities of viable organisms $\geq 50 \mu\text{m}$ and ≥ 10 to $< 50 \mu\text{m}$ were approximately 6 ind./ m^3 and 5 cells/mL immediately after treatment (day 0), respectively, which satisfied the acceptable standards prescribed in D-2 performance standard of the Ballast Water Convention. The treated water was kept for five days to ensure that biotic factors, such as biological re-growth, DNA repair, and resting stages would not develop during testing. After 5 days of storage, the densities of viable organisms $\geq 50 \mu\text{m}$ and ≥ 10 to $< 50 \mu\text{m}$ were approximately 2 ind./ m^3 and 0.021 cells/mL (day 5), respectively, whereas the untreated tanks contained approximately 100540 ind./ m^3 and 1088 cells/mL ([Table S2, Supporting Information](#)). For bacteria, TRO had a significant effect on inactivation with a 4-log reduction in the viable count immediately after treatment from an initial heterotrophic bacterial concentration of around 10^4 cfu/100 mL ([Table S2, Supporting Information](#)). No obvious re-growth was found over the period of five days.

3.2. DBPs concentrations in treated ballast water

Chemical analysis was carried out on each day over the 5-day storage period, and test results indicated that four THMs (i.e., dibromochloromethane (DBCM), dichlorobromomethane (DCBM), tribromomethane (TBM) and trichloromethane (TCM)), four HAAs (i.e., monochloroacetic acid (MCAA), monobromoacetic acid (MBAA), dibromoacetic acid (DBAA), and tribromoacetic acid (TBAA)), dibromoacetonitrile (DBAN) and bromate were detected when the TRO level was 9.5 mg/L as Cl_2 . Based on the worst case principle, the maximum concentrations of the measured DBPs in treated ballast water (Table S3, Supporting Information) were used to conduct the human health risk assessment. The brominated THMs, especially TBM, are dominant, most likely due to the formation of bromine (i.e., HOBr and OBr^-) from the reaction between highly active species and bromide ions (Zhang et al., 2013). The concentrations of four THMs followed an increasing then decreasing pattern with storage time extension (data not shown). The time-effect trends of the observed DBPs were dependent on their formation, stability, as well as TRO decay (Fang et al., 2010). THMs were relatively stable, and it was the final products in the presence of TRO over 5-day period of storage. Therefore, the concentrations of THMs increased and reached a peak with increasing storage time when the TRO was available at the beginning, after which they decreased with TRO decay over time (data not shown). The measured maximum concentrations of total THMs (74.51 $\mu\text{g/L}$) and HAAs (22.88 $\mu\text{g/L}$) in the treated ballast water meet the drinking water standards of US EPA (80 and 60 $\mu\text{g/L}$ for total THMs and HAAs, respectively) and the WHO (560 and 270 $\mu\text{g/L}$ for total THMs and HAAs, respectively) (World Health Organization, 2011; US EPA, 2006). Low concentrations of HAAs may be due to high pH level of seawater ($\text{pH} \approx 8.1$) (Zhang et al., 2013). In addition, the maximum dosage of $\text{Na}_2\text{S}_2\text{O}_3$ was measured as 0.88 mg/L when the TRO concentration was highest immediately after treatment. On the other hand, emission modeling software for ballast water (i.e., MAMPEC-BW) was used to calculate PECs in the discharged ballast water. The maximum calculated PECs were also selected to compute these parameters (i.e., C_{air} and C_{fish}). Several

other calculated parameters of the measured DBPs are shown in [Table S3 \(Supporting Information\)](#). These parameters were used in the subsequent risk assessment.

3.3. Risk characterization of occupational exposure scenarios

Occupational exposure assessment was carried out by evaluation of different exposure scenarios in which the workers could be exposed based on analysis of the operational process of the BWMS to divide the process into unit operations. There were six routine operations identified as likely exposure scenarios for workers: carrying out ballast water treatment activities (i.e., ballasting and de-ballasting), maintenance work on the BWMS, periodic cleaning and inspections of ballast tanks, ballast water sampling, and normal work on deck ([International Maritime Organization, 2012](#)). As shown in [Figure 1](#), work activities were assigned to five unit operations in the BWMS, and the exposure potential for each work activity was determined in detail as follows:

(1) Filtration Unit: Filtration unit operation does not produce any chemical substances; therefore, it does not pose any occupational exposure.

(2) Electrolysis Unit: This unit is composed of an electrolytic cell, degas tank, connecting pipe and valves, etc. In theory, hydrogen gas (H_2) is produced when this unit is operating; however, the workers are not likely exposed to high concentrations of this gas because H_2 is fully separated and diluted in a degas tank, and then discharged to atmosphere outside ships. Since the electrolysis process is performed in an enclosed system, crew will not be exposed to the electrolyzed water.

(3) Ballast Tank: There is a potential risk for dermal uptake of chemicals and/or inhalation of chemicals that may evaporate into the air phase when performing periodic cleaning and/or inspections of ballast tanks. There is also a potential risk for inhalation of chemicals that may be released through vent-heads located on the edge of the deck when carrying out normal work on deck. The concentrations of chemicals in the air calculated using Equation 1 are shown in [Table S3 \(Supporting Information\)](#). Different exposure times (t_{exp}) were assumed for

different working activities. Specifically, t_{exp} was 8 h/d when cleaning the ballast tanks, 3 h/d when inspecting ballast tanks, and 1 h/d when working on deck. The exposure times in such scenarios were regarded as acute exposure (up to 24 h); consequently short-term toxicity effects from the animal studies were used to calculate the DNELs due to the lack of acute human toxicity data. Although TRO consumed with time, risk assessment of TRO was evaluated using maximum level of 9.5 mg/L as Cl_2 based on the worst case principle. For the maximum TRO and DBPs levels measured in this study (Table S3, Supporting Information), the exposure levels of inhalation and dermal routes were calculated based on Equations 3 and 4 (Table 1). Oral acute effects from rat studies ($\text{LC}_{50, \text{rat-oral}}$) were used as the starting point to calculate the DNELs for dermal and inhalation routes based on Equations 10, 11 and 12 (Table 1).

All of these DBPs and TRO caused adverse acute oral effects at doses of $10\text{-}10^3$ mg/kg (Table 2). Comparisons of the exposure levels with the DNELs indicated that the exposure levels were lower than the DNELs, suggesting that the RCRs are orders of magnitude lower than 1, and that there is no risk associated with the ballast tank. The RCRs for dermal exposure ranged from 10^{-7} to 10^{-4} , and the computed range of RCRs was from 10^{-12} to 10^{-3} for inhalation exposure through different working activities. The RCRs for inhalation exposure through cleaning of ballast tanks were approximately 10 times and 10^3 times larger than those for inspections of ballast tanks and working on deck, respectively. These differences reflected differences in the assumed exposure time for different working activities.

Table 2. Risk characterization ratios (RCRs) of occupational exposure through cleaning and inspections of ballast tanks as well as working on deck using animal**toxicity data**

DBPs	Exposure levels ^a				DNELs ^b					RCRs ^c				References
	Dermal (mg/kg)	Inhalation (mg/m ³)			LC _{50, rat-oral} (mg/kg)	Dermal (mg/kg)	Inhalation (mg/m ³)			Dermal	Inhalation			
		Cleaning	Cleaning	Inspecti-on			On deck	Cleaning	Inspection		On deck	Cleaning	Cleaning	
DCBM	6.76E-6	6.12E-2	2.30E-2	7.65E-4	916	13.1	127	340	1020	5.16E-7	4.80E-4	6.75E-5	7.50E-7	Lewis, 2004
DBCM	1.50E-5	6.35E-2	2.38E-2	7.94E-4	370	5.29	51.5	137	412	2.84E-6	1.23E-3	1.73E-4	1.93E-6	Lewis, 1996
TBM	2.16E-4	4.94E-1	1.85E-1	6.17E-3	1147	16.4	160	426	1280	1.32E-5	3.09E-3	4.35E-4	4.84E-6	US EPA, 2005
TCM	3.46E-6	5.37E-2	2.02E-2	6.72E-4	908	13.0	126	337	1010	2.67E-7	4.25E-4	5.98E-5	6.65E-7	WHO, 2000
MBAA	9.54E-6	2.66E-7	9.99E-8	3.33E-9	177	2.53	24.6	65.7	197	3.77E-6	1.08E-8	1.52E-9	1.69E-11	WHO, 2000
MCAA	1.23E-6	4.88E-8	1.83E-8	6.10E-10	55.0	0.786	7.65	20.4	61.2	1.56E-6	6.37E-9	8.96E-10	9.96E-12	European Communities, 2005
DBAA	5.20E-5	9.86E-7	3.70E-7	1.23E-8	1737	24.8	242	644	1930	2.10E-6	4.08E-9	5.74E-10	6.37E-12	WHO, 2000
TBAA	1.12E-5	1.60E-7	6.02E-8	2.01E-9	400	5.71	55.7	148	445	1.96E-6	2.88E-9	4.06E-10	4.51E-12	Worthing and Walker, 1987
DBAN	2.88E-5	1.23E-5	4.63E-6	1.54E-7	245	3.50	34.1	90.9	273	8.24E-6	3.62E-7	5.09E-8	5.65E-10	Hayes et al., 1986
Bromate	2.21E-5	9.43E-6	3.54E-6	1.18E-7	160	2.29	22.3	59.4	178	9.65E-6	4.24E-7	5.96E-8	6.62E-10	Kurokawa, 1990
TRO	3.07E-2	2.00E-4	7.48E-5	5.99E-5	5800	47.3	807	2150	6460	6.49E-4	2.47E-7	3.48E-8	3.86E-10	Lewis, 1996

^a Exposure levels for dermal and inhalation routes were based on Equations 3 and 4. ^b DNELs for dermal and inhalation routes were based on Equations 10, 11 and 12. ^c RCRs were based on Equation 14.

Notes: The abbreviation listed in Table 2 represent as follows: DCBM = dichlorobromomethane, DBCM = dibromochloromethane, TBM = tribromomethane, TCM = trichloromethane, MBAA = monobromoacetic acid, MCAA = monochloroacetic acid, DBAA = dibromoacetic acid, TBAA = tribromoacetic acid, DBAN = dibromoacetoneitrile.

(4) Neutralization Unit: This unit works during de-ballasting. $\text{Na}_2\text{S}_2\text{O}_3$ is the only substance handled and mixed by the crew on board. Although there is the potential for exposure to $\text{Na}_2\text{S}_2\text{O}_3$ during transfer, such risks are considered to be dealt with through the use of appropriate protective measures, such as wearing protective clothing.

(5) Sampling Unit: Similar to the ballast tank, there are potential risks for dermal uptake of chemicals and inhalation of chemicals that evaporate into the air phase while collecting samples. In such scenarios, the exposure times were considered long-term exposure. The highest exposure levels for inhalation and dermal routes were based on Equations 3 and 4 (Table 1). There are two sources of data on for DNELs calculation: those derived from animal toxicity data and those from human epidemiology data. Oral repeated-dose effects from animal studies ($\text{NOAEL}_{\text{ani-oral}}$) were used as the starting point to calculate the DNELs for dermal and inhalation routes, at doses 0.0881-0.462 mg/kg/d and 2.78-36.2 mg/m³/d, respectively (Table S4, Supporting Information). Owing to the lack of sub-chronic dose-response information pertaining to MBAA and TBAA, the animal toxicity data for MCAA and TCAA were used as the starting point to calculate the DNELs. Table S4 (Supporting Information) indicates that the dermal and inhalation DNELs of the measured DBPs using animal toxicity data were approximately 10²-10⁷ and 10³-10⁹ times higher than the exposure levels, respectively. As for human data, most epidemiology studies have focused on DCBM, TCM, DCAA and TCAA (Hamidin et al., 2008). Oral effects from human toxicity data of DCBM, TCM, DCAA and TCAA were observed at the level of 0.001, 0.0007, 0.02 and 0.02 mg/kg/d (King et al., 2000; King et al., 1996; Wright et al., 2004; Hinckley et al., 2005), respectively, and as a result the DNELs for dermal and inhalation routes ranged between 4.32-123 × 10⁻⁵ mg/kg/d and 1.12-32 × 10⁻³ mg/m³/d, respectively (Table S4, Supporting Information). Since the toxicity data of DBAA and TBAA was limited, the data from the closest resembling chlorinated compound (i.e., DCAA and TCAA) was used instead.

For THMs, the RCRs for dermal exposure were approximately 10² times lower than those for inhalation exposure, whereas the RCRs of HAAs, HANs and bromate for inhalation exposure were approximately 10³ and 10² times lower than those for dermal exposure (Table

S4, Supporting Information). These findings indicate that the risk level of inhalation exposure to THMs is higher than that of dermal exposure, but that the opposite is true for HAAs, HANs and bromate.

For occupational exposure, there were five unit operations in the BWMS, and all exposure routes were thoroughly evaluated based on very conservative assumptions. The calculated RCRs using animal data were all less than 1, implying that there is no unacceptable risk to human health without taking into account wearing personal protective equipment (PPE). However, to minimize the potential risk, PPE is highly recommended when entering empty ballast tanks and/or carrying out ballast water sampling.

On the other hand, the risk characterization derived from animal and human data are different, as seen in Figure 2. The inhalation DNELs of DCBM and TCM using human epidemiology data were approximately 10 times lower than the respective exposure levels, indicating that the RCRs of DCBM (9.56) and TCM (12) were larger than 1 (Figure 2a) and the risk of adverse effects on human health were significant. Although the RCRs of DBAA and TBAA using human data were lower than 1, the values were still $10\text{-}10^2$ times higher than those using animal toxicity data (Figure 2a). Similar results were also found when calculating dermal RCRs (Figure 2b). This implies that there are apparent discrepancies between risk characterization from animal and human data, which may affect the overall risk assessment results. These discrepancies may be related to a number of factors such as different endpoints, response time and exposure concentrations (Hamidin et al., 2008).

3.4. Risk characterization of general public exposure scenarios

Although maximum TRO level was 9.5 mg/L as Cl_2 , human health risk assessment of TRO for general public exposure scenarios was not evaluated in this study because treated ballast water was neutralized using $\text{Na}_2\text{S}_2\text{O}_3$ upon discharge if the TRO content was larger than 0.2 mg/L as Cl_2 . Indirect exposure of humans via the environment into which treated ballast water is discharged may occur by swimming in or eating fish from the surrounding area. The

exposure of the general public is normally assessed as a chronic/lifetime risk to protect the most vulnerable population groups. Therefore, the NOAELs from the human and/or animal studies were used to calculate the DNELs. For DNELs, the computed range using animal data was 10^{-3} to $10 \text{ mg/kg(m}^3\text{)/d}$ (Table S5, Supporting Information), which were approximately $0.286\text{--}161 \times 10^2$ times higher than those using human data.

For the exposure levels that occurred via inhalation, dermal and oral routes while swimming as well as through consumption of fish, the PECs of DBPs that were used in the calculation of exposure levels based on Equations 5, 6, 7 and 8 (Table 1) differed depending on the emission simulation model and parameters of the environment (i.e., harbor dimensions, hydrodynamics, and properties). In this study, we selected the MAMPEC-BW model and the GESAMP-BWWG (Group of Experts on Scientific Aspects of Marine Environmental Protection–Ballast Water Working Group) Model Harbor to calculate the PECs of DBPs. The exposure levels for swimming ranged from 10^{-10} to $10^{-3} \text{ mg/kg(m}^3\text{)/d}$, while they ranged from 10^{-7} to 10^{-5} mg/kg/d for eating fish (Table S5, Supporting Information). Although the calculation of DNELs used NOAELs as a starting point, the values were still higher than the exposure levels. As shown in Table S5 (Supporting Information), when using animal toxicity data, the RCRs of inhalation, dermal and oral routes through swimming were $10^{-10}\text{--}10^{-4}$, $10^{-6}\text{--}10^{-3}$ and $10^{-7}\text{--}10^{-4}$, respectively, while those for fish consumption were 10^{-7} to 10^{-4} for a normal person. The differences between the RCRs computed for different exposure routes were not only dependent on the concentrations of the DBPs, but also on properties that determined their fate (e.g., volatility, bioaccumulation, biomagnification, degradation). On the other hand, the RCRs using human data were also observed for DCBM, TCM, DBAA and TBAA. The RCRs of these compounds were lower than 1, however, the values were approximately $10\text{--}10^5$ times higher than those using animal toxicity data (Figure 3).

4. Conclusions

In this study, the animal toxicity data was converted into the corrected human data by the

adjustment of five AFs as described in Section 2.4; however, the risk assessment results indicates that even higher AFs may be needed when laboratory animal toxicity data are applied in risk assessment. Based on our risk assessment results, we suggested that human data should be used in risk assessment as much as possible since it is common to have discrepancies between animal and human data in several orders of magnitudes. And the higher AFs (e.g, the difference in experimentally observed effect between human and animals) should be considered to be applied in risk assessment for human. The recommended values for AFs in this study are at least 10^3 for THMs and 10 for HAAs when the animal data is selected as the dose descriptors.

In the end, it should be noted that this study was limited in that route-to-route extrapolation was used when there was no adequate experimental data available for the relevant route of exposure. The different absorption rates of each chemical through the exposure routes were not considered because adsorption correction factors were not available for this study. Default values of absorption correction factors usually assume that 100% of the chemical is absorbed via the intestines, skin and lungs. However, the results of this study suggest that this factor may be considered in risk evaluations when the rates of absorption of chemicals into the body via different routes are known.

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Supporting Information

Auxiliary information on list of the measured DBPs (Table S1); summary of plankton and bacteria inactivation test results in the untreated and treated water on days 0 and 5, respectively (Table S2); summary of the maximum concentrations of the measured DBPs and several other calculated parameters (Table S3); risk characterization ratios (RCRs) of occupational exposure through ballast water sampling (Table S4); and risk characterization ratios (RCRs) of general public exposure through swimming and eating fish (Table S5).

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Figure captions

Figure 1. Identification and/or description of unit operations in the electrolysis-based ballast water management system.

Figure 2. Risk characterization ratios (RCRs) of DCBM, TCM, DBAA and TBAA representing the differences between animal and human toxicity data through (a) inhalation and (b) dermal exposure routes during ballast water sampling.

Figure 3. Risk characterization ratios (RCRs) of DCBM, TCM, DBAA and TBAA representing the differences between animal and human toxicity data through (a) inhalation, (b) dermal and (c) oral exposure routes during swimming, and (d) eating fishes.

Figure 1

Figure 2

Figure 3