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An ecological risk assessment (ERA) and a human health risk assessment (HHRA) of butyltins were performed, based on the estimated tissue residues in the food web of the Jincheng Bay mariculture area using the fugacity-based bioaccumulation model. The fugacity-based model was readily applicable to refine the ERA and HHRA of pollutants in marine areas to provide a basis for the protection of marine ecology and the security of fishery products, which could also help determine the feasibility of aquaculture before being initiated.

Risk Assessment of Butyltins Based on a Fugacity-based Food Web

Bioaccumulation Model in the Jincheng Bay Mariculture Area: II. Risk

Assessment

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Abstract

A fugacity-based food web bioaccumulation model was constructed, and the biotic concentrations of butyltins in the food web of the Jincheng Bay Mariculture Area were estimated accordingly, using the water and sediment concentrations, which was described in the accompanying paper (Part I). This paper presents an ecological risk assessment (ERA) and a human health risk assessment (HHRA) of the butyltins, based on the estimated tissue residues in this area. The results showed that the ecological risk probability was greater than 0.05 as the critical level for management control and sensitive marine species would be profoundly penalized by butyltin contamination, whereas little or no detrimental effects would be generated for humans from exposure to butyltins through seafood consumption. The fugacity-based model was readily applicable to refine the ERA and HHRA of pollutants in marine areas to provide a basis for the protection of marine ecology and the security of fishery products, which could also help determine the feasibility of aquaculture before being initiated.

Keywords

Bioaccumulation; Food web; Fugacity; Mariculture area; Risk assessment; Organotin

1. Introduction

Butyltins, including tributyltin (TBT), dibutyltin (DBT), and monobutyltin (MBT), are types of extensively distributed persistent organic chemicals that could adversely affect marine organisms and human beings at trace levels¹⁻⁶. Similarly to other chemicals, an aquatic ecological risk assessment (ERA) of butyltins is usually based on the external aqueous exposure of target pollutants or environmental quantity guidelines^{7,8}, which is termed an

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external ecological risk assessment (EERA). For example, Díez and Bayona⁹ performed an ERA of TBT in the sediment of the Iberian Peninsula by comparing its sediment concentrations with the corresponding sediment quality guidelines. Hall et al.¹⁰ characterized the ecological risk of TBT in the Chesapeake Bay with the overlap of the probability distributions of the water concentrations and those of the toxic values. However, an EERA appears to underestimate the true risk for highly hydrophobic and poorly depurated chemicals^{11, 12}, an internal ecological risk assessment (IERA) was thus addressed to achieve a refinement.

In this paper with respect of the butyltin pollution in the Jincheng Bay Mariculture Area (JBMA), an ERA for the biota and a human health risk assessment (HHRA) for the consumers *via* seafood were implemented, based upon the biotic residues of the butyltins that was predicted employing the fugacity-based food web model. This work would provide a basis for the protection of the marine ecology and the security of fishery products and to simultaneously provide a refinement for the HHRA of aquatic products.

2. Materials and methods

2.1 Ecological risk assessment

The joint probability curve (JPC) approach^{7, 8} was employed to perform an ERA of the butyltins for the marine biota by integrating the distributions of biotic tissue residues based on the bioaccumulation model with the species sensitivity distributions (SSDs) based on internal toxicity data. The JPC describes the probabilities that a certain proportion of species are expected to be adversely affected, with the cumulative probability of the exposure data as the independent variable and the reverse cumulative probability of exposure data, named the exceedance probability (*EXP*), as the dependent variable. The distance between a JPC and its relative axes could indicate the risk level. The farther the distance, the higher the risk.^{7, 8} More specifically, the area under the curve profiles the overall risk probability (*ORP*), with the adverse effects expected to occur in the following formula:

$$ORP = \int_0^1 EXP(x)dx \tag{1}$$

where *EXP*(*x*) is the exceedance probability of the exposure data, associated with 100*x*% of the species expected to be adversely affected.

Internal aquatic toxicity data, including the no observed effect levels (NOELs) and lowest observable effect levels (LOELs), that involved multiple types of effects, such as survival, growth, and development, were retrieved from the ERED database (<http://www.wes.army.mil/el/ered>) to develop the internal species sensitivity distributions (ISSDs) (Table 1), of which the LOELs were converted into the corresponding NOELs referring Kalf et al.¹³ and Sijm et al.¹⁴, while no NOEL was available for a certain species. The conversion is detailed in Box S1 of the Supplementary Information. If multiple toxicity values were available for a single species, the geometric

mean was taken as a surrogate.¹⁵ Because insufficient toxicity data of MBT were available due to its low toxicity potency, its risk to marine species was not quantified herein.

Table 1 Statistical summary and log-logistic fitting parameters of NOEL for butyltins in aquatic species

Butyltin	N	Statistic				log-logistic fitting [†]			
		Min	Max	GM	GSD	α	β	D-value	P
Internal toxicity data (ng-Sn/g, wet weight)									
TBT	25	1	2.09E+4	65.9	11.4	1.78 (1.37-2.19)	0.60 (0.40-0.79)	0.11	0.92
DBT	9	2.53	9.95E+3	209	14.8	2.38 (1.63-3.13)	0.65 (0.30-1.00)	0.14	0.99
External toxicity data (ng-Sn/L)									
TBT	53	0.36	2.04E+7	601	30.5	2.67 (2.30-3.40)	0.80 (0.61-0.98)	0.10	0.69
DBT	5	195	1.25E+8	9.63E+4	293	4.87 (2.68-7.06)	1.38 (0.42-2.33)	0.22	0.94

[†] The log-logistic model was $\Phi_{(x)} = 1/(1 + \exp(-(x - \alpha)/\beta))$, where $\Phi_{(x)}$ was defined as the empirical cumulative density function of NOEL modified in the form of $\Phi_{(x)} = i/(n + 1)$, with n representing the total number of NOELs and i representing the ascent order of x.

2.2 Human health assessment

Considering no evidence for human carcinogenicity^{16, 17}, the HHRA of the butyltins from seafood were performed by computing the meal consumption limits¹⁸, as follows:

$$CRlim = \frac{RfD \times BW}{C} \quad (2)$$

$$CRmm = \frac{CRlim \times Tap}{MS} \quad (3)$$

where *CRlim* and *CRmm* are the maximum allowable consumption rate in kg/d and in meals/month, respectively; *RfD* is the reference dose, which is 0.1 µg-Sn/kg/d for both TBT and DBT;¹⁹ *BW* is the body weight of consumers, which is 60 kg for an average Chinese adult;²⁰ *Tap* represents the average period (30 d/month); *C* represents the concentrations of butyltins in the seafood; and *MS* is the meal size, which averages 197 g/meal for a Chinese adult, converted according to the EPA¹⁸.

The risk of MBT was not assessed as it failed to show any evidence of teratogenic activity in Wistar pregnant rats at any doses tested (50-400 mg/kg/d), whereas low levels of TBT and DBT (e.g., 1.7-15 mg/kg/d) were both observed to cause dose-dependent thymic atrophy and increases in fetus death and resorption.²¹ In addition, the immunotoxicity and neurotoxicity potencies of MBT were much weaker than those of both TBT and DBT.^{17, 22}

Because of analogous molecular structures and a similar toxic mode of action (MOA), the joint risk of a TBT and DBT mixture was also recommended to be assessed by a concentration addition.¹⁷ Therefore, the seafood consumption limit for the exposure of a mixture could be calculated as follows:¹⁸

$$CRlim = \frac{BW}{\sum Ci/RfDi} \quad (4)$$

where C_i and $RfDi$ represent the concentrations of the butyltins compound i in seafood and its respective reference dose.

3. Results and discussion

3.1 Ecological risk of butyltins

JPCs were constructed by integrating the distributions of the estimated tissue residues of the butyltins with the corresponding ISSDs as non-conservative estimations. Regarding the uncertainties of the extrapolation from laboratory data to the field, two conservative approaches were also chosen to create JPCs for the security of more-sensitivity species not covered in the data assemblage. With respect to the first approach, JPCs were created based on the upper CIs of ISSDs, and with respect to the second, the toxicity data were divided by an assessment factor (AF) of 5. As shown in Fig. 1, under the non-conservative estimations, the $ORPs$ of TBT and DBT were 0.06 and 0.07, respectively, and the corresponding exceedance probabilities of 5% of the potentially affected fraction (EXP5) were 0.48 and 0.59, respectively. Under the first conservative estimations, the $ORPs$ of TBT and DBT were 0.11 and 0.18, respectively, with the EXP5 being 0.78 and 0.96, respectively. The results of the second conservative estimation were close to those of the first (Fig. 1). The species protection level was usually chosen to be 95%, which meant that an ORP of less than 0.05 was acceptable and vice versa.²³ In this respect, the risk values of the butyltins were higher than the critical level of 0.05 for risk management even under the non-conservative estimations, and sensitive species would be seriously affected by the butyltin pollution in this area.

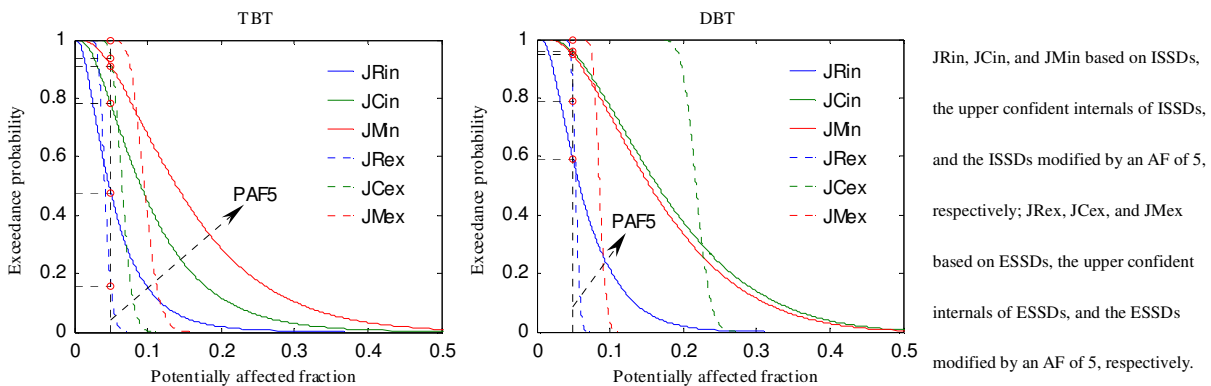


Fig. 1 JPCs of butyltins in the JBMA based on internal toxicity data

The main fishery products in the JBMA were mollusca, including *A. irradians*, *C. ariakensis*, and *N. didyma*. As illustrated in Fig. S1, the mollusca were relatively sensitive to both TBT and DBT. WHO²⁴ and Axiak et al.² reported that the exposure of 20-200 and 10-1000 ng/L TBT induced significant shell thickening and digestive cell volume reduction in *C. gigas* and *Ostrea edulis*, respectively. Fisher et al.²⁵ reported that the long-term exposure to 30 and 80 ng/L TBT significantly exacerbated the infectious disease process of *C. virginica* to a protozoan

pathogen (*Perkinsus marinus*). Leung et al.²⁶ reported that the exposure of 1-2 ng/L TBT could induce imposex and body weight descent in various gastropods. Roepke et al.²⁷ reported that the 96 h exposure to TBT delayed the development of the *Lytechinus anamesus* larvae and generated abnormal individuals exhibiting features such as misshapen spicules, missing arms, and incomplete guts, even at a level as low as 0.1 ng/L. The water levels of the butyltins in the JBMA were 23.9-44.8 ng-Sn/L with TBT being 0.60-2.90 ng-Sn/L, which exceeded the adverse levels to various mollusca such as *N. lapillus* and *L. littorea*. The comparison suggested a considerable risk to sensitive species from butyltins in the studied area. Consequently, the risk values estimated by the fugacity-based model were reliable.

To make a comparison with EERA, JPCs were also constructed by integrating the distributions of the butyltins in the seawater (Table 1 of the accompanying paper), with the SSDs based on aqueous toxicity data (Table 1 & S2). The results were delineated in the identical figure (Fig. 1). Accordingly, on the non-conservative and conservative bases, the *ORPs* of TBT were 0.04, 0.07, and 0.10, whereas those of DBT were 0.05, 0.22, and 0.09. The *ORPs* of TBT (0.06, 0.11, and 0.16) and DBT (0.07, 0.18, 0.17) from the IERA were slightly higher than or comparable with the values from the EERA. These discrepancies could be attributable to several reasons: (1) the EERA characterized risk by integrating aqueous exposure concentrations with external toxicity data, whereas the IERA integrated biotic tissue residues with internal toxicity values; (2) the EERA based on the external environmental exposure did not take the interspecies bioaccumulation heterogeneity into account, whereas the IERA, based on internal tissue levels, incorporated multiple exposure pathways; and (3) different species usually exhibit diverse sensitivities to butyltins. However, the species involved in the toxicity dataset for the IERA (Table S1) were different from those for the EERA (Table S2). Taking TBT as an example, only five species such as *N. lapillus*, *Nassarius reticulatus*, and *Mytilus edulis* were shared by the two assemblages. Moreover, because the available toxicity data of butyltins were limited, the critical toxicity values compiled to implement the IERA and EERA involved all types of effects in the targeted species at multiple life stages *via* different exposure routes, with various organism body parts being detected, as shown in Table S1 and S2. Therefore, the disunity of the limited toxicity data decreased the comparability of the IERA with the EERA, and contributed much to the discrepancies. (4) The tissue residues of the butyltins were estimated with the fugacity-based model, the inherent uncertainties of which would necessarily be introduced to the IERA.

As discussed above, the exposure *via* diet and the toxicity kinetics of the target toxicants were not taken into consideration in the traditional approach (EERA), which would underestimate the total effect of multiple exposures.²⁸ As reported by Huang and Wang²⁹, the BCFs of TBT in *M. edulis* and *A. irradians* were determined to be 7700-11000 and 2000-10000, respectively, which increased exponentially with the exposure intensities (0.02,

0.064, 0.10, and 0.50 $\mu\text{g/L}$) under a chronic continuous flow-through exposure system. These results revealed that the concentrations in a specific environmental medium were not adequate to characterize the true biotic exposure *via* multiple pathways. In the previous studies, the sediment quality guidelines (SQGs) of Cd were usually derived based on aqueous and sediment toxicity data, with the threshold effect level (TEL) and probabilistic effect level (PEL) determined to be 0.2-1.5 and 1.2-12 mg/kg, respectively.³⁰ However, based on a long-term extensive investigation (2200 benthos were collected in 4200 sampling stations for 6 years), the TEL and PEL were determined to be 0.058 and 0.129 mg/kg, respectively, by constructing field-based species sensitivity distributions (f-SSD) with population abundances³⁰, which were evidently lower than the above-mentioned reported values based upon the traditional approach. Therefore, internal tissue residues were more appropriate to characterize the risk of a pollutant than external exposure. However, tremendous resources are required to conduct a long-term, many-station, multiple-species investigation to obtain the internal tissue residues. This study revealed the feasibility of an ERA with the internal tissue residues estimated by constructing a fugacity-based food web bioaccumulation model. However, most reported aquatic toxicity data were based on external aqueous exposure, whereas the internal toxicity data were quite scarce, which decreased the accuracy of the IERA. Thus, the toxicity test for internal data, with the use of biotic internal concentrations to prepare toxicity curves, should draw more attention, particularly for highly hydrophobic persistent contaminants.

3.2 Human health risk of butyltins

Meal consumption limits were computed to characterize the health risk of butyltins in seafood, referring the EPA¹⁸. With respect to the intra/interspecies exposure heterogeneity, the 5th percentiles were taken as the conservative risk values³¹, as shown in Table 2. In the light of the individual exposure of TBT, the maximum allowable consumption rate of the main fishery species (mollusca) from the JBMA was 0.75 kg/d or 114 meals/month, following the average seafood meal size (197 g/meal) for Chinese adults, whereas that for all categories of seafood in total was 0.99 kg/d or 150 meals/month. With respect to the individual exposure of DBT, the consumption rates were 0.35 kg/d or 53 meals/month for mollusca and 0.26 kg/d or 39 meals/month for seafood in total. As discussed above, the risk levels of DBT to human health were higher than those of TBT. Finally, in terms of the combined exposure of the TBT and DBT mixture, the consumption rate of mollusca was 0.27 kg/d or 40 meals/month, and that of seafood in total was 0.23 kg/d or 35 meals/month. These values were obviously higher than the average levels for Chinese adults both nationwide (31.9 kg/a, equivalent to 0.087 kg/d) in 2009³² and in the typical coastal city of Zhoushan (0.105 ± 0.182 kg/d) in 2003³³. The EPA¹⁸ demonstrated that seafood with a consumption rate greater than 16 meals/month would not affect human health and thereby would not be subject to consumption restriction. From this point of view, the consumption of the fishery products from

the JBMA would not affect human health for butyltin pollution, and the consumption needed no restriction.

Table 2 Consumption limits of seafood for the adverse effects of butyltins

	Mollusca			Seafood in total [†]		
	TBT	DBT	ΣBT	TBT	DBT	ΣBT
<i>CRlim</i> (kg/d)						
GM [‡]	3.91 (3.84-3.99)	1.85 (1.81-1.89)	1.09 (1.07-1.11)	3.23 (3.18-3.27)	0.87 (0.87-0.90)	0.64 (0.63-0.65)
Q5 [§]	0.75 (0.73-0.76)	0.35 (0.34-0.36)	0.27 (0.26-0.27)	0.99 (0.98-1.00)	0.26 (0.25-0.26)	0.23 (0.23-0.23)
<i>CRmm</i> (meals/month)						
GM [‡]	594 (583-606)	281 (276-287)	165 (162-168)	490 (483-497)	135 (133-137)	97 (96-99)
Q5 [§]	114 (111-116)	53 (52-54)	40 (40-41)	150 (148-153)	39 (39-40)	35 (35-36)

[†] Including all FGs except for detritus and planktons, given the consumption fraction proportional to the relative biomass^{34, 35}.

[‡] geometric mean.

[§] 5th percentile.

The HHRA for the consumption of contaminated seafood is usually based on measured biological tissue residues of the target pollutants. For example, Lee et al.¹⁶ detected the tissue levels of TBT and TPT in 31 fish species in 19 harbors in Taiwan and calculated the hazard index to human health following the local consumption rate of 67 g/d, which was 0.15-8.6, suggesting a potential health risk by the consumption of contaminated seafood from this area. Hites et al.³⁶ detected the contents of 14 persistent organic pollutants in farmed salmon in Europe and North America and determined that the consumption rates were 1-14 meals/month, which suggested a potential risk to human health for salmon consumption. In this paper, a fugacity-based model was developed to simulate the bioaccumulation of butyltins in the food web of the JBMA, which was used to perform an HHRA for the consumption of contaminated seafood for the first time. Using this model, the tissue levels of target pollutants could be predicted in a complex food web using measured concentrations in the water column and sediment, and based on the estimations, the risk levels to the aquaculture species could be qualified together with the risks to human health *via* seafood consumption, which could be applied to determine whether an area was available for aquaculture prior to actual aquaculture performance.

4. Conclusions

Based on the estimated tissue residues of the butyltins in the food web of the JBMA using the fugacity-based bioaccumulation model, an ERA for the marine biota and an HHRA for human health were performed, which showed that the ecological values of butyltins exceeded 0.05 as the critical level for management control and sensitive species would be seriously affected. Regarding the combined exposure of TBT and DBT, the consumption rates of the seafood from the JBMA were higher than average Chinese consumption levels, suggesting no evident impact of butyltins on human health *via* seafood from this area. The fugacity-based model could be readily applied to conduct an ERA in marine areas to determine whether they are appropriate for aquaculture prior to initiation, which could provide a refinement for the aquatic ERA and HHRA together with a

basis for the protection of the marine ecology and the security of fishery products.

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