Coastal Marine Science 35(1): 129–135, 2012

Ecotoxicological risk of organotin compounds on zooplankton community

Inneke F. M. RUMENGAN[1] and Madoka OHJI[2]

[1] *Faculty of Fisheries and Marine Science, Sam Ratulangi University. Kampus Unsrat, Bahu, Manado 95115, Indonesia*
**E-mail: inneke0511@gmail.com; innekerumengan@unsrat.ac.id*
[2] *Institute of Symbiotic Science and Technology, Tokyo University of Agriculture and Technology, Fuchu, Tokyo 183–8509, Japan*

Received 29 October 2010; Accepted 30 June 2011

Abstract—Organotin compounds are bioavailable to zooplankton through direct cellular interaction or feeding mechanisms and therefore could be biomagnified to higher trophic levels. Among organotin species, tributyltin (TBT) shows the longest persistence in the lower trophic levels, and is accumulated in zooplankton up to 150,000 times that in ambient water. Higher ability of zooplankton to accumulate butyltins could be associated with their feeding behaviour as filter feeders. Some zooplankton such as rotifers can accumulate 5 and 2 folds that of alga and mysids, respectively. Generally, meroplankton such as veliger larvae of the mussel (*Mytilus edulis*) and lobster larvae (*Homarus americanus*) are more sensitive than the adults. Threshold concentration (i.e. the concentration at which toxicity starts) for acute toxicity in certain copepods is below 0.3 mg l^{-1} TBTO. Chronic toxicity levels are in the range of one-thousandth to one-tenth part per billion, and generally the No Observed Effects Concentration (NOEC) for zooplankton is 1 ng l^{-1}. The most worrisome are chronic effects on reproduction that could decrease marine-species populations. Long term monitoring programs, including those on zooplankton, for the assessment of the effectiveness of global ban is obviously necessary.

Key words: organotin compounds, zooplankton, bioaccumulation, acute and chronic effects

Introduction

Zooplankton are potentially used for ecotoxicological studies, considering their short period of life cycle and the ease and speed of making quantitative measurements of mortality and reproduction, and their sensitivity to common pollutants. Although less attention has been paid to the biomass of this lower end of the marine food chain, there are few reports on levels of organotin compounds in zooplankton and toxicity tests in laboratories that have demonstrated adverse ecotoxicological effects of organotin compounds on zooplankton population. Taylor et al. (2002) provided some information on the nonlinear responses of plankton communities to the environmental perturbations, and suggested that plankton, especially copepods, the most abundant zooplankton, are more sensitive indicators of environmental change.

Annual world consumption of organotin compounds grew from 35,000 tons in 1986 (Maguire 1987) to 50,000 tons in 1992 (Guruge et al. 1996). Among the 260 known organotin compounds, 36 are potentially very hazardous to natural resources (Eisler 1989). Among these compounds, butyltins including the mono-, di- and triibutyltins (MBT, DBT, TBT) are the most well known organotin compounds. The most concerned butyltin compounds is TBT, since it is used not only as a marine biocide in anti-fouling paint, but also in wood treatment and preservation, antifungal action in textiles and industrial water system (Extoxnet 1996). TBT is a highly tioxic biocide (USEPA 2003), and therefore there has been a great deal of public concern focused on the ecotoxicological aspects of this compound. The International Maritime Organization (IMO) has adopted a convention to ban the application of organotin compounds especially as an antifouling agent after January 2003 and forbid its usage after 2008. Restrictions on the use of TBT antifouling paints have yet to be established elsewhere in ASEAN, but recently the subject has been receiving increasing attention. In Indonesia, there still is no regulation banning the application of organotin chemicals (Rumengan et al. 2008). Widespread contamination of BTs along the coastal waters of Asian developing countries including Indonesia in 1990s has been reported by several authors (Ellis and Pattisina 1990, Evans et al. 1995, Sudaryanto et al. 2002, 2004, 2005, Midorikawa et al. 2004b, Rumengan et al. 2008).

Contamination of organotin compounds has been of global concern, due to their ubiquity in water ecosystem, and most often present at lower nanomolar concentrations in water but then accumulate in aquatic organisms (Atanasov et al. 2005, Hu et al. 2006). The persistence of organotin compounds in the environment is a function of physical (adsorption to suspended solids and sediments), chemical (chemical and photochemical degradation) and biological (uptake and biological degradation) removal mechanisms (Eghomwanre 2005). After the publication of Rumengan and Ohji (2008),

almost no new report on the toxic effects appeared on marine zooplankton. Such systematic toxicity studies of organotin compounds on the secondary producers of aquatic food chain have not been reported, after studies of Ohji et al. (2002a,b, 2003, 2004) conducted on caprellid amphipod. This review aims to contribute to the dissemination of information related to toxicity effects of organotin compounds on zooplankton.

Bioaccumulation

Soluble organotin compounds in the water column are available to zooplankton through direct contact, and the compounds may be taken up either through such direct biochemical process, cellular diffusion or filtering mechanisms (Eghomwanre 2005). The filtering mechanism is a pathway associated with feeding behaviour of zooplankton as filter feeders. This lead to the potential biomagnification of organotin compounds via zooplankton (Rumengan and Ohji 2008).

As reviewed by USEPA (2003), the solubility of organotin compounds is influenced by its status, oxidation/reduction potential, pH, temperature, ionic strength, and concentration and composition of the dissolved organic matter. The solubility of tributyltin oxide (TBTO) in different pH was reported by Maguire et al. (1984) to be 750 $\mu g\,l^{-1}$ at pH of 6.6, 31,000 $\mu g\,l^{-1}$ at pH of 8.1, and 30,000 $\mu g\,l^{-1}$ at pH 2.6. Harino et al. (1998) found that the partition coefficients of TBT to sediment, plankton, and mussels from the Port of Osaka were higher than those of DBT and MBT, and those of TBT to plankton and mussels were higher than those in sediment. Accumulation of organotin compounds in zooplankton could be 70,000 times the concentration in ambient seawater (Takahashi et al. 1999), and even up to 150,000 times the concentration in the seawater of the Port of Osaka (Harino et al. 1999).

Evidence exists which implies that organotin compounds can be transferred through the marine food chain, notably that based on filter feeders and predatory species (Cooney 1988). Field and laboratory studies on trophic magnification factors of organotin compounds in a marine food web are very limited. Lee et al. (2006), who conducted a field study on accumulation of organotin compounds in different taxa, suggested that TBT and TPT accumulations in fish might arise principally from their feed, and indicated, circumstantially, that biomagnification has occurred along the aquatic food chain.

Verslycke et al. (2005) reported their studies on toxicant-induced biomarker responses and population effects of TBT on resident mysids. High concentrations of organotin compounds in these benthic zooplankton indicated that potential point sources are their feed such as detritus, algae and other zooplankton. TBT concentrations of $>50\,ng\,l^{-1}$ in water would be high enough to result in effects on low trophic level organisms.

As discussed in Rumengan and Ohji (2008), there are two applicable factors, a bioaccumulation factor (BAF) which is the ratio of the concentration of the organotin compounds in an organism derived from both water and food to the concentration of the compounds in ambient water, and bioconcentration factor (BCF) which is the ratio of the concentration of organotin compounds in organisms which is derived from water only to the concentration of the organotin compounds in ambient water. The food chain transfer number f (unitless) can be calculated from the difference between BAF and BCF divided by the ratio of the concentration of contaminant in food and the concentration of contaminant in ambient water. If values of f are >1.0 biomagnification is inferred. Hu et al. (2006) calculated a trophic magnification factor of 3.70 for TPT compared to TBT which is only 0.59. Sun et al. (2001) have assessed the biomagnification of butyltins for three species at relatively low trophic levels, e.g. algae (*Platymonas* sp.), rotifers (*B. plicatilis*), and mysids (*Neomysis awatschensis*). The food chain transfer number f was 1.44 between the algae and rotifers , but only 0.59 between the rotifers and mysids.

Toxicity effects

The general order of toxicity of organotin compounds increases with the number and chain length of organic groups bonded to the tin atom from one to three, and decrease with the incorporation of a fourth, making triorganotin more toxic than other forms. Within triorganotin, toxicity increases as the number of carbon atom in the organic moiety increases from one to four, then decrease (USEPA 2003). Thus the most toxic to aquatic life is TBT. At cellular level, toxic effects of organotin compounds take place primarily through interactions with membrane lipids (Eng et al. 1988, Avery et al. 1993), because of their lipophilicity. White et al. (1999) proved that the site of action of organotin compounds is both at the cytoplasmic membrane and intracellular level of microorganisms including plankton. TBT may cross biomembranes more easily than the hydrophilic cations and interact with cytosolic enzymes (Fent and Looser 1995, Veltman et al. 2006), resulting in higher accumulation levels. TBT acts as an enzymatic inhibitor and affects the transport of ions across cell membrane (USEPA 2003).

As summarized in Table 1, it is clear that acute toxicity data for zooplankton are limited to a few species, and very limited species have been used for both acute and chronic toxicity tests. The lethal concentrations of TBT for copepods are below 2 $\mu g\,l^{-1}$, much lower than those for rotifers and meroplankton. USEPA (2003) has calculated or interpolated graphically from original acute toxicity data the following order of sensitivity to tributyltin oxide: calanoid copepod *Acartia tonsa* (0.24 $\mu g\,l^{-1}$), mysid *Acanthomysis sculpta* (0.42–1.68 $\mu g\,l^{-1}$), harpacticoid copepod *Nitocra spinipes* (1.9 $\mu g\,l^{-1}$), mysid *Metamysidopsis elongata* ($<$0.97 to 6.8 $\mu g\,l^{-1}$), and mysid *Mysidopsis bahia* (1.1 to 2.2 $\mu g\,l^{-1}$).

U'ren (1983) suggested that the threshold concentration of acute toxicity (i.e. the concentration at which toxicity starts) for *A. tonsa* is below 0.3 $\mu g\,l^{-1}$ TBTO.

Generally, meroplankton stages of any tested species are more sensitive to organotin compounds than the adults. Beaumont and Budd (1984) exposed veliger larvae of the mussel *Mytilus edulis* to TBTO for 15 days and found that no larvae survived longer than 5 days in 10 $\mu g\,l^{-1}$ TBTO, or longer than 10 days in 1 $\mu g\,l^{-1}$ TBTO. Larvae of the lobster *Homarus americanus* did not survive more than 6 days when exposed to 5 $\mu g\,l^{-1}$ TBTO (Laughlin and French 1980, cited in U'ren 1983).

In our preliminary experiment using milkfish fries with average body size of 26.9 mg in weight and 1.8 cm in length, collected from a coastal water in North Sulawesi, the TBT concentration that start to have an acute effects is 0.01 $\mu g\,l^{-1}$, and the 48 h LC50 is 10 $\mu g\,l^{-1}$ (Table 1). This meroplankton is much more resistant than the holozooplankton, copepods and rotifers.

Toxicity effects on population level are field assessed on the imposex phenomenon and morphological changes and population related parameters, such as growth and reproduction. Organotin compounds are suspected endocrine-disrupting chemicals because they cause irreversible sexual abnormality (masculinization) in female mollusks, called "imposex" (Filho et al. 2010) and linked to immuno-supression in molluscs (USEPA 2003). Laboratory investigation for this assessment is based on chromic toxicity experiments.

Determination of the the lowest observed effect concentration (LOEC) is a prerequisite for any chronic toxicity test. Most antifouling compounds have LOEC values at $\mu g\,l^{-1}$ levels, which represent the initial toxicity threshold of a chemical (Fernandez-Alba et al. 2002, USEPA 2003). Because plankton are generally more sensitive than the organisms at higher trophic levels, most chronic toxicity tests for plankton are conducted at 0.001–0.1 $\mu g\,l^{-1}$ levels. Generally, the no observed effect concentration (NOEC) for phytoplankton and zooplankton is $\sim$1 $ng\,l^{-1}$ (Extoxnet 1993, Linley-Adam 1999).

As shown in Table 1, only a few data is available on chronic effects of organotin compounds on zooplankton. Chronic effects of TBT on plankton are clearly highly varied amongst plankton species. Nevertheless, from the early 1980s to the present day, laboratory studies have continued to show that, at low levels, the chronic effects of organotin compounds may have considerable impacts on plankton dynam-

Table 1. Toxicity effects of TBT on zooplankton

Zooplankton taxon	Lethality category	TBT ($\mu g\,l^{-1}$)	Chronic effect	Chronic value ($\mu g\,l^{-1}$)	Reference
Calanoida					
Acartia tonsa	48 h-LC50	1.100			Bushong et al. (1987)
A. tonsa	96 h-LC50	0.948			U'ren (1983)
Eurytemora affinis	72 h-LC50	0.600	Reproduction ability reduced	<0.088	Bushong et al. 1987; Hall et al. (1987, 1988)
Harpacticoida					
Nitocra spinipes	96 h-LC50	1.895			Linden et al. 1979
Mysida					
Acanthomysis sculpta	96h-LC50	0.420	Reduction in reproduction	0.1308	Davidson et al. (1986a,b)
Mysidopsis bahla	96 h-LC50	1.100			Goodman et al. 1988
Rotifera					
Brachionus plicatilis	24 h-LC50	300			Snell et al. 1991a,b
Caprellidae					
Caprella danilevskii	48 h-LC50	5.900	Survival reduction, morphological alterations, inhibition of maturation	0.01	Ohji et al. 2002a, 2003a
Milkfish fry					
(Chanos chanos)	48 h-LC50	10			Unpublished data
Larval Pacific oyster					
Crassostrea gigas	96 h-LC50	1.557			Thain 1983 in USEPA 2003
Larval blue *mussel*					
Mytilus edulis	96 h-LC50	2.238			Thain 1983 in USEPA 2003
Postlarval hard					
clam M. mercenaria	96 h-LC50	0.015			Becerra-Huencho, 1984 in USEPA 2003

ics and morphology in culture, although data are available only for some species, mainly copepods and meroplankton, mostly from subtropical waters. The available data on chronic values for TBT, including those for some copepod species, are all below 0.01 $\mu g\,l^{-1}$ (USEPA 2003). *A. tonsa* showed inhibition of development at 0.003 $\mu g\,l^{-1}$. Many reports demonstrate that reductions in growth occur in commercially or ecologically important marine species at concentrations of TBT less than the final chronic value of 0.0658 $\mu g\,l^{-1}$. Hall et al. (1987) performed chronic tests on *E. affinis* and found that TBT had a significant effect at 0.2 $\mu g\,l^{-1}$ TBT, whilst the threshold was apparently 0.1 $\mu g\,l^{-1}$ TBT—much higher than that in *A. tonsa*. U'ren (1983) found that some copepods exposed to 0.3 $\mu g\,l^{-1}$ TBTO (equivalent to 0.285 $\mu g\,l^{-1}$ TBT) became moribund at 96 h and effective survival was only 63% after 6 days. Organotin compounds may also inhibit the developmental rate of larvae of marine copepods at 1 $ng\,l^{-1}$, whereas the survival of larvae was affected at 15 to 20 $ng\,l^{-1}$. *A. tonsa* is particularly sensitive to TBT (Extoxnet 1993). Two partial life-cycle toxicity tests have been conducted using the copepod *E. affinis* (Hall et al. 1987). The chronic value was $<0.088\ \mu g\,l^{-1}$ in this test. The percentage survival of neonates was significantly reduced (73% lower than control survival) at 0.22 $\mu g\,l^{-1}$. The chronic value in this test was 0.14 $\mu g\,l^{-1}$, while the geometric means of the NOEC and LOEC were 0.094 $\mu g\,l^{-1}$ and 0.224 $\mu g\,l^{-1}$, respectively. Compared to copepods, the saltwater mysid, *Acanthomysis sculpta* is more resistant to TBT. The effects of TBT on survival, growth, and reproduction of *A. sculpta* were determined by Davidson et al. (1986a, b) in five separate tests lasting from 28 to 63 days. The number of juveniles released per female at a TBT concentration of 0.19 $\mu g\,l^{-1}$ was 50% of the number released in the control treatment. Numbers of females releasing viable juveniles was reduced in 0.19 and 0.33 $\mu g\,l^{-1}$ TBT. At concentrations of 0.38 $\mu g\,l^{-1}$ and above, survival and weight of female mysids were reduced; all mysids in 0.48 $\mu g\,l^{-1}$ died. Based on reproductive effects, the geometric mean of chronic values was 0.13 $\mu g\,l^{-1}$ (USEPA 2003).

Chronic toxicity effects on meroplankton are comparable to those for holozooplankton. Beaumont and Budd (1984) have reported ~50% mortality in the larvae of mussels *M. edulis* exposed to 0.1 $\mu g\,l^{-1}$ TBTO for 15 d (i.e., 15-d LC50 approximately 0.1 $\mu g\,l^{-1}$ tributyltin oxide), and most surviving larvae were moribund and had grown significantly more slowly than controls. It is clear that TBT causes adverse reproductive and developmental effects in aquatic organisms at very low concentrations (USEPA 2003). Comparable level of chronic effect for marine gastropods, even at very low concentrations (a few $ng\,l^{-1}$), TBT triggers the development of male sex characteristics in female (BUA Report 2003).

Tocixity effects of organotin compounds at the community level are lacking, except a citation of USEPA (2003) from a study in San Diego Bay by Lenihan et al. (1990) who hypothesized that changes in faunal composition in hard bottom communities were related to boat mooring and TBT. In an ecotoxicological risk assessment for aquatic ecosystems, the lowest effective value can be based either on the LOEC of 0.002 $\mu g\,l^{-1}$ (imposex in the dogwhelk *Nucella lapillus*: Davies et al. 1987) or the EC10 of 0.0007 $\mu g\,l^{-1}$ (development in *A. tonsa*, copepods: Kusk and Petersen 1997). USEPA (2003) develops for TBT the criterion to protect saltwater aquatic life from chronic toxic effect is 0.0074 $\mu g\,l^{-1}$ (lowered to protect growth of commercially important molluscs, survival of the ecologically important copepod *A. tonsa,* and survival of the ecologically important gastropod *N. lapillus*).

Results from long-term studies with organisms from at least three trophic levels indicated that the predicted no-effect concentration (PNEC) for aquatic ecosystems, based on the value for *A. tonsa*, is 0.07 $ng\,l^{-1}$ (BUA Report 2000). Organotin coumpounds alters development and sexual parameters of reproductive system (Filho et al. 2010). The accumulation of a organotin compound in a zooplankton could result in inhibition of reproduction ability, which will then reduce the population.

Even though toxicity data with zooplankton are limited to several species, some zooplankton are good indicator for assessment of organotin compounds, particularly tributyltin (TBT) contamination in marine environment. Snell and Janssen (1995) consider that rotifers are useful in ecotoxicological studies due to some reasons including the ease and speed of making quantitative measurements of mortality and reproduction, their sensitivity to common pollutants, and the commercial availability of cysts. As an example, the rotifers *Brachionus* have proved to be a better indicator organism for assessing environmental gradients than the entire zooplankton assemblage. Hence, this taxon can be considered a target taxon for more intensive monitoring and conservation planning (Austoni et al. 2006).

Chronic toxicity levels are in range of one-thousandth to one-tenth part per billion, and generally the (NOEC for zooplankton is 1 $ng\,l^{-1}$ (Extoxnet 1996). The term 'no effect' is usually directed to no reduction or inhibition in a biological process, while a toxic compound even could increase or stimulate a biological process (hormesis). Although hormesis is associated to an acute toxicity effect, but it probably has long-term effects afterwards. Hormesis could be interpreted as indicative of a particular response of a physiological system to a toxin.

Future recommendation

Assessment of any ecotoxicological risk of organotin compounds on plankton could be laboratory-based works on the potential transfer pathway of organotin compounds from seawater to zooplankton through feeding mechanism. This

could lead to further assess the threat of biomagnification to public health, especially from fish and other seafood of that might consume contaminated plankton. Acute and chronic bioassays are possible approaches for that achievement. Zooplankton used in bioassays should be of a single genotype as a world-wide standard for ecosystem testing.

The toxicity and potential hazard of organotin compounds to zooplankton community remain areas for future studies. Field works on monitoring on zooplankton community as a compartment in ecosystem are obviously necessary to evaluate any disturbance including the impacts of organotin compounds input. Contamination simplifies the assemblage of certain taxa and prompts the numerical dominance of a handful of species. It is obvious that the number of species decreased in polluted circumstances and that the more severe the level of contamination the greater the reduction, and therefore the field surveys should cover both spatial and temporal variation over the hot spot and uncontaminated areas. There is none a single factor in nature could be charged as the cause of disturbance in zooplankton, either in population or community levels. Therefore, monitoring on plankton should be based on the integrated multi tasks approaches. The data of butyltin in zooplankton from long-term monitoring are only provided by Harino et al. (1999) for the Port of Osaka. However, they have just observed the total zooplankton, not the community assemblages as reported for some zooplankton species that appear typical of eutrophic waters (Gulati 1983, Austoni et al. 2006, and Javed 2006). Long-term monitoring program on the organotin compound contamination in marine ecosystem compartments including zooplankton is obviously necessary for assessment of the effectiveness of global ban.

Acknowledgments

We thank to the sponsorship of Japan Society for Promotion of Science (Multilateral Core University Program: Coastal Marine Science) for enabling us having more perspectives on the marine pollution through many international events and for making this review be disseminated.

References

Atanasov, A. G., Nashev, L. G., Tam, S., Baker, M. E. and Odermatt, A. 2005. Organotins disrupt the 11(beta)-hydroxysteroid dehydrogenase type 2-dependent local inactivation of glucocorticoids. Environ. Health Perspect. 113: 1600–1606.

Austoni, M., Cardoso, A. C., Deviller, G., Kamburska, L., Marinov, D., Somma, F. and Zaldívar, J. M. 2006. Indicators for the assessment of thresholds and points of non-return. A Thresholds Project Report. Dir. General Joint Research Centre, EUR 22130EN, European Community, Ispra, Italy.

Avery, S. V., Codd, G. A. and Gadd, G. M. 1993. Biosorption of tributyltin and other organotin compounds compounds by cyanobacteria and microalgae. Appl. Microbiol. Biotechnol. 39: 812–817.

Beaumont, A. R. and Budd, M. D. 1984. High mortality of the larvae of the common mussel at low concentrations of triutyltin. Mar. Pollut. Bull. 15: 402–405.

BUA 2003. Tributyltin oxide (Bis-[tri-n-butyltin]oxide) (No. 36). BUA Report 238 (Supplementary Reports IX), Wissenschaftliche Verlagsgesellschaft, S. Hirzel Verlag, Stuttgart.

Bushong, S. J., Hall, S., Johnson, W. E. and Hall, L. W. 1987. Toxicity of tributyltin to selected Chhesapeake Bay biota. *In* Proceedings of the Organotin Symposium, Oceans ‘87 Conference. pp. 1499–1503, Halifax, Nova Scotia.

Cooney, J. J. 1988. Microbial transformations of tin and tin compounds. J. Indust. Microbiol. Biotech. 3: 195–204.

Davidson, B. M., Valkirs, A. O. and Seligman, P. F. 1986a. Acute and chronic effects of tributyltin on the mysid *Acanthomysis sculpta* (Crustacea, Mysidacea). NOSC-TR-1116 or AD-A175-294-8. National Technical Information Service, Springfield, VA.

Davidson, B. M., Valkirs, A. O. and Seligman, P. F. 1986b. Acute and chronic effects of tributyltin on the mysid *Acanthomysis sculpta* (Crustacea, Mysidacea). *In* Proceedings of the International Organotin Symposium, Oceans ’86. pp. 1219–1225, Washington, D.C.

Davies, I. M., Bailey S. K. and Moore, D. C. 1987. Tributyltin in Scottish sealocks, as indicated by degree of imposex in the dog-whelk, *Nucella lapillus* (L). Mar. Pollut. Bull. 18: 400–404.

Eghomwanre, A. A. 2005. Anthropogenic influence with respect to heavy metals and organotin pollution in Göta Älv River in Sweden. Master’s Thesis in Applied Environmental Measurement Techniques. Chalmers University of Technology Göteborg, Sweden. pp. 69.

Eisler, R. 1989. Tin hazards to fish, wildlife, and invertebrates: A synoptic review. Contaminant Hazard Reviews. Report No. 15. Biological Report 85 (1.15).

Ellis, D. V. and Pattisina, L. A. 1990. Widespread neogastropod imposex: A biological indicator of global TBT contamination? Mar. Pollut. Bull. 21: 248–253.

Eng, G., Tierney, E. J. and Bellama, J. M. 1988. Correlation of molecular total surface area with organotin toxicity for biological and physicochemical applications. Appl. Organomet. Chem. 2: 171–175.

Evans, S. M., Dawson, M., Day, J., Frid, C. L. J., Gill, M. E., Pattisina, L. A. and Porter, J. 1995. Domestic waste and TBT pollution in coastal area of Ambon Island (Eastern Indonesia). Mar. Pollut. Bull. 30: 109–115.

Extoxnet (Extention Toxicology Network) 1996. Tributyltin. Oregon State University. www.extoxnet.orst.edu/pips/ghindex.html. assessed October 1, 2007.

Fent, K. and Looser, P. W. 1995. Bioaccumulation and bioavailability of tributyltin chloride: influence of pH and humic acid. Water Res. 29: 1631–1637.

Fernandez-Alba, A. R., Hernando, M. D. and Piedra, L. 2002. Toxicity evaluation of single and mixed antifouling biocides measured with acute toxicity bioassays. Anal. Chim. Acta 456: 303–312.

Filho, V. S. D., Mancin, C. N., Silva, I. V., Pedrosa, D. F., Destefani, A. C., Samoto V. Y., Takiya, C. M. and Graceli, J. B. 2010. En-

docrine disruption induced by triorganotin (IV) compounds: Impacts in the reproductive and genetic function. J. Med. Genet. Genomics 2: 29–37.

Goodman, L. R., Cripe G. M., Moody, P. H. and Halsell, D. G. 1988. Acute toxicity of malathion, tetrabromobisphenol-A and tributyltin chloride to mysids (*Mysidopsis bahia*) of three ages. Bull. Environ. Contam. Toxicol. 41: 746–753.

Gulati, R. D. 1983. Zooplankton and its grazing as indicators of trophic status in Dutch lakes. Environ. Monitor. Assess. 3: 343–354.

Guruge, K. S., Tanabe, S. and Iwata, H. 1996. Distribution, biomagnification and elimination of butyltin compound residues in common cormorants (*Phalacrocorax carbo*) from Lake Biwa, Japan. Arch. Environ. Contam. Toxicol. 31: 210–217.

Hall, L. W., Bushong, S. J. and Ziegenfuss, M. C. 1987. Chronic toxicity of tributyltin to Chesapeake Bay Biota. Water Air Soil Pollut. 39: 365–376.

Harino, H., Fukushima, M. and Kawai, S. 1999. Temporal trends of organotin compounds in the aquatic environment of the Port of Osaka, Japan. Environ. Pollut. 105: 1–7.

Harino, H., Fukushima, M., Yamamoto, Y., Kawai, S. and Miyazaki, N. 1998. Organotin compuonds in water, sediment, and biological sample from the Port of Osaka, Japan. Arch. Environ. Contam. Toxicol. 35: 558–564.

Hu, J., Zhen, H. and Wan, Y. 2006. Trophic magnification of triphenyltin in a marine food web of Bohai Bay, North China: comparison to tributyltin. Environ. Sci. Technol. 40: 3142–3147.

Javed, M. 2006. Studies on metal contamination levels in plankton and their role as biological indicator of water pollution in the River Ravi. Pakistan J. Biol. Sci. 9: 313–317.

Jeon, J. K., Lee, M. H. and Lee, J. S. 2003. Toxicity of TBT and TPT compounds on the survival of rotifer (*Brachionus plicatilis*). Korean J. Environ. Bull. 21: 158–163.

Kusk, K. O. and Petersen, S. 1997. Acute and chronic toxicity of tributyltin and linear alkylbenzene sulfonate to the marine copepod *Acartia tonsa*. Environ. Toxicol. Chem. 16: 1629–163.

Lee, C. C., Hsieh, C. Y. and Tien, C. J. 2006. Factors influencing organotin distribution in different marine environmental compartments, and their potential health risk. Chemosphere 65: 547–559.

Lenihan, H. S., Oliver, J. S. and Stephenson, M. A. 1990. Changes in hard bottom communities related to boat mooring and tributyltin in San Diego Bay: a natural experiment. Mar. Ecol. Prog. Ser. 60: 147–159.

Linden, E., Bengtsson, B. and Svanberg, O. 1979. The acute toxicity of 78 chemicals and pesticide formulations against two brackish water organisms, the bleak (*Alburnus alburnus*) and the harpacticoid *Nitocra spinipes*. Chemosphere 8: 843–851.

Linley-Adam, G. 1999. Harmful effects of the use of anti-fouling paints for ships. The accumulation and impact of organotin compounds on the marine biota within the Mediterranean Region. International Maritime Organization Report. MEPC43/INF14.WPD. pp. 15.

Maguire, R. J., Wong, P. T. S. and Rhamey, J. S. 1984. Accumulation and metabolism of tri-*n*-butyltin cation by a green alga, *Ankistrodesmus falcatus*. Can. J. Fish. Aquat. Sci. 41: 537–540.

Maguire, R. J. 1987. Environmental aspects of tributyltin. Appl. Organometal. Chem. 1: 475 –498.

Midorikawa, S., Arai, T., Harino, H., Ohji, M., Cu, N. D., Duc, P. A. and Miyazaki, N. 2004b. Organotin levels in bivalves in Southeast. Coast. Mar. Sci. 29: 57–62.

Ohji, M., Takeuchi, I. and Takahashi, S. 2002a. Differences in the acute toxicities of tributyltin between the Caprellidea and the Gammaridea (Crustacea: Amphipoda). Mar. Pollu. Bull. 44: 16–24.

Ohji, M., Arai, T. and Miyazaki, N. 2002b. Effects of tributyltin exposure in the embryonic stage on sex ratio and survival rate in the caprellid amphipod *Caprella danilevskii.* Mar. Ecol. Prog. Ser. 235: 171–176.

Ohji, M., Arai, T. and Miyazaki, N. 2003a. Chronic effects of tributyltin on the caprellid amphipod *Caprella danilevskii*. Mar. Pollut. Bull. 46: 1263–1272.

Ohji, M., Arai, T. and Miyazaki, N. 2003. Chronic effects of tribultyltin on the caprellid amphipod *Caprella danilevskii*. Mar. Pollut. Bull. 46: 1263–1272.

Ohji, M., Arai, T. and Miyazaki, N. 2004. Effects of tribultyltin on the caprellid amphipod *Caprella danilevskii*. J. Mar. Biol. Ass. UK 84: 345–359.

Rumengan, I. F. M., Ohji, M., Arai, T., Harino, H., Arifin, Z. and Miyazaki, N. 2008. Contamination status of butyltin compounds in Indonesian coastal waters: A Review. Coast. Mar. Sci. 32: 116–126.

Snell, T. W., Moffat, B. D. and Janssen, C. 1991a. Acute toxicity tests using rotifers IV. Effects of cyst age, temperature and salinity on the sensitivity of *Brachionus calyciflorus*. Ecotoxicol. Environ. Saf. 21: 308–317.

Snell, T. W., Moffat, B. D. and Janssen, C. 1991b. Acute toxicity tests using rotifers III. Effects of temperature, strain and exposure time on the sensitivity of *Brachionus plicatilis*. Environ. Toxicol. Water. Qual. 6: 63–75.

Snell, T. W. and Janssen, C. R. 1995. Rotifers in ecotoxicology: a review. Hydrobiologia 313/314: 231–247.

Sudaryanto, A., Takahashi, S., Monirith, I., Ismail, A., Muchtar, M., Zheng, J., Richardson, B. J., Subramanian, A., Prudente, M. Hue, H. D. and Tanabe, S. 2002. Asia–Pacific Mussel Watch: Monitoring of butyltin contamination in coastal waters of Asian developing countries. Environ. Toxicol. Chem. 21: 2119–2130.

Sudaryanto, A., Takahashi, S., Iwata, H., Tanabe S. and Ismail, A. 2004. Contamination of butyltin compounds in Malaysian marine environments. Environ. Pollut. 130: 347–358.

Sudaryanto, A., Takahashi, S., Iwata, H., Tanabe S., Muchtar, M. and Razak, H. 2005. Organotin residues and the role of anthropogenic tin sources in the coastal marine environment of Indonesia. Mar. Pollut. Bull. 50: 208–236.

Sun, H. W., Dai, S. G. and Huang, G. L. 2001. Bioaccumulation of butyltins via an estuarine food chain. Water Air Soil Pollut. 125: 55–68.

Takahashi, S., Tanabe, S. and Takeuchi, S. 1999. Distribution and specific bioaccumulation of butyltin compounds in a marine ecosystem. Arch. Environ. Contam. Toxicol. 37: 50–61.

Taylor, A. H., Allen, J. I. and Clark, P. A. 2002. Extraction of a weak climatic signal by an ecosystem. Nature 416: 629–632.

U'ren, S. C. 1983. Acute toxicity of bis (tributyltin) oxide to a marine copepod. Mar. Pollut. Bull. 14: 303–306.

USEPA (U.S. Environmental Protection Agency) 2003. Ambient aquatic life water quality criteria for tributyltin. USEPA Office of Research and Development, Environmental. Office of Water 4304T. US EPA 822-R-03-031.

Verslycke, T. A., Vethaak, A. D., Arijs, K. and Janssen, C. R. 2005. Flame retardants, surfactants and organotins in sediment and mysid shrimp of the Scheldt estuary (The Netherlands). Environ. Pollut. 136: 19–31.

Veltman, K., Huijbregts, M. A. J. and Van den Heuvel-Greve, M. J. 2006. Organotin accumulation in an estuarine food chain: Comparing field measurements with model estimations. Mar. Environ. Res. 61: 511–530.

White, J. S., Tobin, J. M. and Cooney, J. J. 1999. Organotin compounds and their interactions with microorganisms. Can. J. Microbiol. 45: 541–554.