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Hazard/Risk Assessment

AQUATIC ECOLOGICAL RISKS POSED BY TRIBUTYLTIN IN UNITED STATES SURFACE WATERS: PRE-1989 TO 1996 DATA

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Abstract—Acute and chronic risks to aquatic life from exposure to tributyltin (TBT) in surface waters were assessed probabilistically using more than 9 years of monitoring data. More than 50 sites around the United States were sampled, representing six saltwater regions and one freshwater (Lake Erie). Ambient TBT concentrations were compared to acute and chronic effect thresholds to estimate risks for each year (1986–1988 and 1992–1996), site type, and region. Site types comprised commercial harbors, shipyards, marinas, and fish/shellfish habitats proximate to these commercial areas. Tributyltin concentrations in surface waters have declined in all regions and site types since passage of the Organotin Antifouling Paint Control Act in 1988. No risks of acute toxicity have been suggested since 1994. Chronic risks have remained highest in marinas compared to other site types but have declined from a risk involving 25% of the species prior to 1989 to one involving 6% of the species in 1996. Risks associated with commercial harbors and shipyards have been similar (4–6%) since 1994. Chronic risks have been ≤1% in fish and shellfish habitats sampled <1 to 2 km from TBT sources. Risks in Galveston Bay, over all years (1–19%), have been greater than in the other regions (≤9%).

Keywords—Tributyltin Risk assessment Toxicity Chronic Acute

INTRODUCTION

Tributyltin (TBT) has been used as an antifoulant in marine paints since its commercial introduction in 1965 [1,2]. Elevated surface water concentrations of TBT have been observed where vessel usage was high, specifically in marinas and harbors [3,4]. Tributyltin exerts chronic effects on aquatic organisms at concentrations down to ≤10–20 ng/L, and 10 ng/L has been proposed by the U.S. Environmental Protection Agency (U.S. EPA) as the chronic marine water quality criterion protective of at least 95% of aquatic organisms [5]. In the 1980s, concern about risks to nontarget organisms prompted passage of the Organotin Antifouling Paint Control Act (OAPCA) by the U.S. Congress in late 1988. This Act restricted use of TBT-containing paints to ships over 25 m and those with aluminum hulls. It also limited TBT use to paints with laboratory-tested release rates of ≤4 ($\mu g/cm^2$)/day. Subsequent to restrictions on TBT usage, environmental monitoring revealed declining TBT concentrations in the United States, Europe, and Japan [1,6–10]. For instance, since passage of OAPCA, median TBT concentrations in U.S. marine waters generally have averaged less than 10 ng/L, the U.S. EPA [5] chronic marine water quality criterion [11]. Concentrations in the freshwater region monitored (Lake Erie) have been less than EPA's freshwater chronic water quality criterion, 63 ng/L, since 1991 [11].

Key questions remain, however, as to whether the reductions have sufficiently diminished risks to levels adequately protecting aquatic life and, if not, whether temporal trends in risks near TBT sources suggest protective levels will be attained within a reasonable time period.

The objective of this study was to determine acute and chronic risks to aquatic life from exposure to TBT in U.S. surface waters and to evaluate regional and source-related (e.g., marinas, commercial harbors, and shipyards) differences in risks. Risks in saltwater were emphasized because fouling on boat hulls is generally higher in saltwater versus freshwater, therefore, TBT use is higher on saltwater vessels. The risk assessment approach used is probabilistic because it models variability in estimated TBT exposure levels as well as in TBT's acute and chronic toxicity to aquatic organisms. The resultant risk characterization yields a probability density function (PDF), which expresses the variability in the percentage of aquatic taxa at risk. The statistical expectation of the acute risk PDF is referred to as the expected mean acute risk, and the mean of the chronic risk PDF is referred to as the expected chronic risk. An expected chronic risk of 30%, for example, means that, on average, 30% of the taxa are predicted to be missing or replaced, based on comparison to the number and types of species expected to occur at similar uncontaminated sites.

The risk assessment methodology and terminology used in this investigation follows the Water Environment Research Foundation (WERF) [12]. The WERF probabilistic approach uses aquatic toxicity data to develop a model (the PDF or cumulative distribution function sometimes referred to as the species sensitivity curve). The distribution of expected environmental concentrations (EECs) is compared to the species sensitivity curve to determine the percentage of aquatic taxa whose effects thresholds are exceeded by the EEC. Thus, it tells us, for example, whether an EEC poses risk to 5, 10, etc. percent of the aquatic community.

PROBLEM FORMULATION

Problem formulation defines the surface water sources of the stress (TBT), what species of organisms (receptors) are affected and how, the assessment endpoint, measures of ex-

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posure, measures of effects, and the objectives and scope of the risk estimation.

Sources of tributyltin

The primary source of TBT in surface water is from vessels painted with TBT-containing antifoulant paints. In the 1970s and 1980s, use of TBT as an antifoulant in boat bottom paints elevated surface water concentrations where vessel usage was high, specifically marinas and harbors. For example, in U.S. waters, Hall [13] observed concentrations ranging from 20 to 1,800 ng/L, and Seligman et al. [8] reported concentrations up to 450 ng/L (based on TBT ion, unless noted otherwise). In the late 1980s, the United States and many other nations sought to reduce environmental concentrations of TBT by restricting the types and sizes of vessels using TBT-containing antifoulant paints and by limiting TBT leaching rates in paints [14–16].

To determine its concentrations in the United States, various programs have monitored TBT in surface waters, sediments, and biota [6,9,11]. Monitoring has been sponsored by the TBT manufacturers in a program required by U.S. EPA. The monitoring was mandated by the U.S. Congress with passage of OAPCA, which required the U.S. EPA and the U.S. Navy to monitor and report on the Act's effectiveness at reducing TBT concentrations. From 1992 through 1996, our program annually collected approximately 1,200 surface water samples, 600 sediment samples, and 140 composites of bivalve mollusks. Data are summarized by Russell et al. [1], and details can be found in Parametrix [11]. The surface water data from this program as well as those from the program conducted by the Navy are used in this assessment to define EECs over time in relation to source (e.g., marinas).

Biological resources affected

The biological resources (i.e., receptors) most directly susceptible to TBT in surface waters include organisms that ventilate and feed in the water column. These organisms comprise pelagic and demersal fish, holo- and meroplankton, epibenthos (fish and invertebrates living on the sediment's surface), and filter-feeding benthos or benthic invertebrates (e.g., clams). Mollusks are most susceptible to TBT, and this may be due to diminished cytochrome P-450 content and slower depuration rates [17].

Tributyltin appears to be assimilated by aquatic organisms mainly via the gills and other respiratory membranes. Although most of the toxicity appears to manifest via the water pathway, variable exposure occurs via the diet [18,19]. Tributyltin concentrations in surface waters can be used to index risks from both bioconcentration and dietary accumulation if one assumes TBT concentrations are in equilibrium among the receptor, its prey, and the water it ventilates [20].

Assessment endpoint

The assessment endpoint represents the social values to be protected and serves as a point of reference for the risk assessment. The assessment endpoint will be achieved if populations are not compromised, signified by inconsequential effects on growth, survival, or reproductive success of most species, including those that are ecologically keystone (influence the associated community directly or indirectly out of proportion to their abundance or biomass) or economically important [21].

Measures of exposure

The measure of exposure for this study was total surface water TBT concentration, as reported by the monitoring programs described by Russell et al. [1] and Seligman et al. [8,9]. In the program described by Russell et al. [1], a stratified random design was used in which three sites of each site type were monitored quarterly in each of four regions and from within each site type [11]. Replicate samples were taken 1 m below the surface and 1 m above the bottom at three randomly chosen locations. Each water sample represented a composite of three 2-L water samples taken side by side. The number of samples represented in this database was 115 in 1991 and 575 to 971 per year from 1992 through 1996. Typically, at least 32 samples were taken each year within any site type.

The site types met specific TBT use patterns. Accordingly, the marinas monitored possessed more than 200 boat slips for vessels less than 25 m in length, and the harbors accommodated more than 100 ocean-going vessel visits per year of ships at least 77 m in length. Shipyards had to be capable of servicing vessels >25 m in length. An effort was made to study shipyards that had serviced vessels with paints containing TBT. Fish/shellfish habitats sampled had to be adjacent (less than ~1–2 km) to shipping or boating activities. Four regions were monitored quarterly around the United States (Fig. 1).

In the Navy program [8,9], the sampling scope varied by region. In general, water column samples were collected quarterly to annually in triplicate 0.5 m below the surface and 1 m above the bottom. Each bay (San Diego Bay, California; Pearl Harbor, Hawaii; and Norfolk Harbor, Virginia) was divided into study areas based primarily on geography, circulation characteristics, and vessel use (Fig. 1). At least three stations were sampled within each study area. Four study areas were monitored in San Diego Bay based primarily on current velocities and use patterns, eight in Pearl Harbor based on geography and use patterns, and five in Norfolk Harbor based on geography and use patterns. For comparison, we classified the Navy monitoring stations into marinas, commercial harbors/drydocks, and fish/shellfish habitat. Harbor and drydock locations could not be distinguished. This database comprised 1,561 samples prior to 1989 and from 424 to 920 per year from 1989 through 1991.

Our TBT EEC data [1,11] had to meet specific quality control criteria. These criteria included use of a quantitative internal standard recovery and no more than a certain level (e.g., 10 ng/L) of TBT detected in method blank samples, etc. Because the analytical laboratory primarily responsible for TBT analyses reported consistently low levels of TBT in its artificial seawater method blanks (i.e., mean of 5.2 ng/L, standard deviation of 3.5), the data were adjusted to account for the overestimated environmental TBT concentrations. The TBT concentrations in the three method blanks associated with each analytical batch of environmental samples were averaged and subtracted from each EEC measured in the batch. Resulting values less than the method detection limit (MDL) of 5.5 ng/L were converted to one half the MDL for use here. MDLs were calculated as described in U.S. Federal Register 40 CFR 136, Appendix B. As confirmation of the approach's appropriateness, duplicate samples ($n = >950$) were analyzed in 1996 by two laboratories, our primary laboratory and one that did not report any TBT in its method blanks. The accuracy and precision of these two laboratories did not differ significantly ($p < 0.05$) when the data were blank-corrected as de-

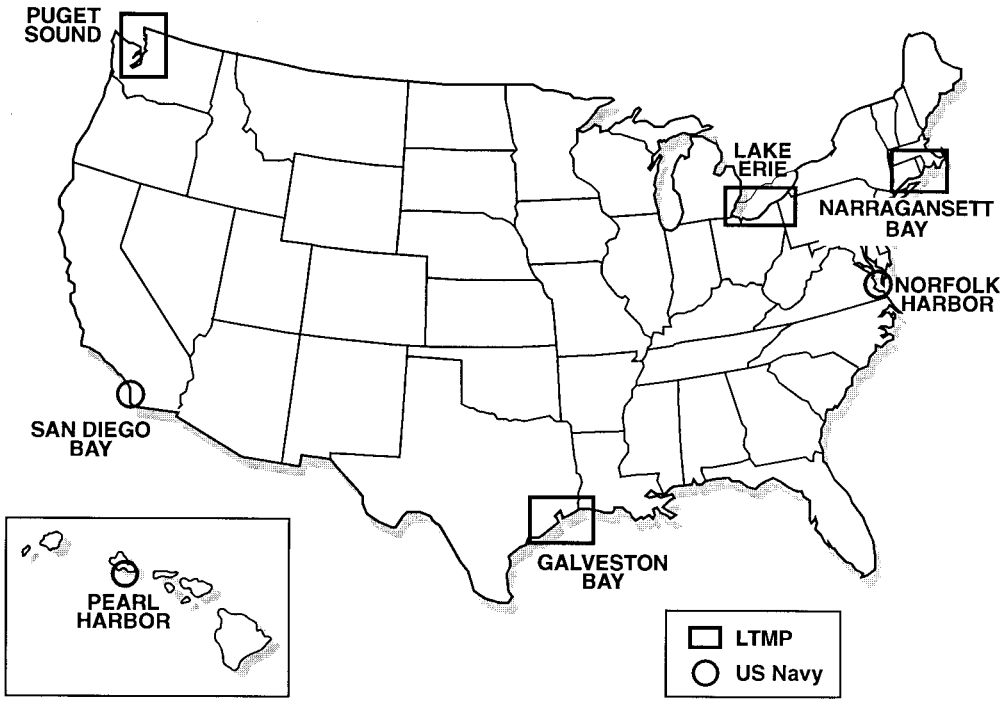

Fig. 1. Surface waters sampled.

scribed. The Navy's data were used as received from the Naval Ocean Systems Center, San Diego, California. The differences in treatment of these databases do not appear to represent a significant uncertainty as data from the two laboratories could not be distinguished statistically.

Measures of effect

Measures of effect are functionally analogous to median lethal or effective concentrations for acute toxicity, which are divided by two to estimate the lethal concentration to 1% (LC1), and the *chronic value* for chronic toxicity [21]. The latter value is equivalent to the geometric mean of the no observed effect concentration (NOEC) and the lowest observed effect concentration (LOEC) of the most sensitive endpoint measured in chronic tests involving growth *and* survival or reproductive success [21]. These endpoints are compatible with U.S. EPA guidance [21,22]. Most chronic values were predicted from acute data using a TBT acute to chronic ratio of 14.7 [5].

Objectives and scope of risk estimation

This paper's primary objective is to quantify the risks to aquatic life posed by TBT in U.S. surface waters. Other objectives include ranking risks by TBT sources (identified as marinas, commercial harbors, shipyards, and fish/shellfish habitat) and gauging temporal trends in risks. The latter were based on monitoring data collected annually prior to and since OAPCA's passage in 1988.

Probabilistic risk assessment provides a means for addressing the foregoing objectives by quantifying risks to particular organisms specifically and to the aquatic community generally [12]. The WERF methodology used is based on risk assessment paradigms inherent in the EPA's water quality-based approach [23] and water quality criteria [21]. Species are ranked in terms of sensitivity to TBT, and a specified fraction of the aquatic community is protected. Similar techniques have been proposed in Europe [24]. The Society for Environmental Toxicology and Chemistry [25] recommends a 90% level of ecological protection, whereas the U.S. EPA recommends a 95% level. Thus, both the 90 and 95 percentiles are used herein as benchmarks.

EXPOSURE CHARACTERIZATION METHODS

The exposure characterization predicts the TBT concentrations to which the receptors are exposed. The combined surface water TBT concentration data from the U.S. EPA-required and Navy programs were used (Figs. 2 and 3). Expected environmental concentrations of TBT in surface waters were estimated by fitting PDFs to the TBT concentrations, grouped by monitoring year, site type, and region.

Probability density functions were fit to only the individual data points (not means or medians) greater than the MDL using the commercial software package BestFit (Palisade, Newfield, NY, USA) [26]. Then, the same proportion of the simulated data set was replaced with one half the MDLs as had existed in the original data set. Method detection limits were sufficiently low such that one half of the value was expected to result in zero risk, so the uncertainty in quantifying nondetects contributed negligibly to the risk estimates.

BestFit uses an optimization routine to compute the parameters (e.g., mean and standard deviation) of 26 continuous PDFs according to whether they best fit gamma, lognormal, Weibull, or other distributions. Three statistical tests (chi square, Kolmogorov-Smirnov, and Anderson-Darling) are used to describe each optimized PDF's goodness-of-fit to the data. We used all available nonnegative PDFs to empirically fit EEC models to the TBT data. Because the EECs cannot be <0, we restricted our analysis to nonnegative distributions. Any PDF outperformed by another PDF on all three goodness-of-fit tests was classified as ''dominated'' and rejected as an EEC model. For example, the best fitting Weibull distribution would be rejected if the best-fitting lognormal distribution gave

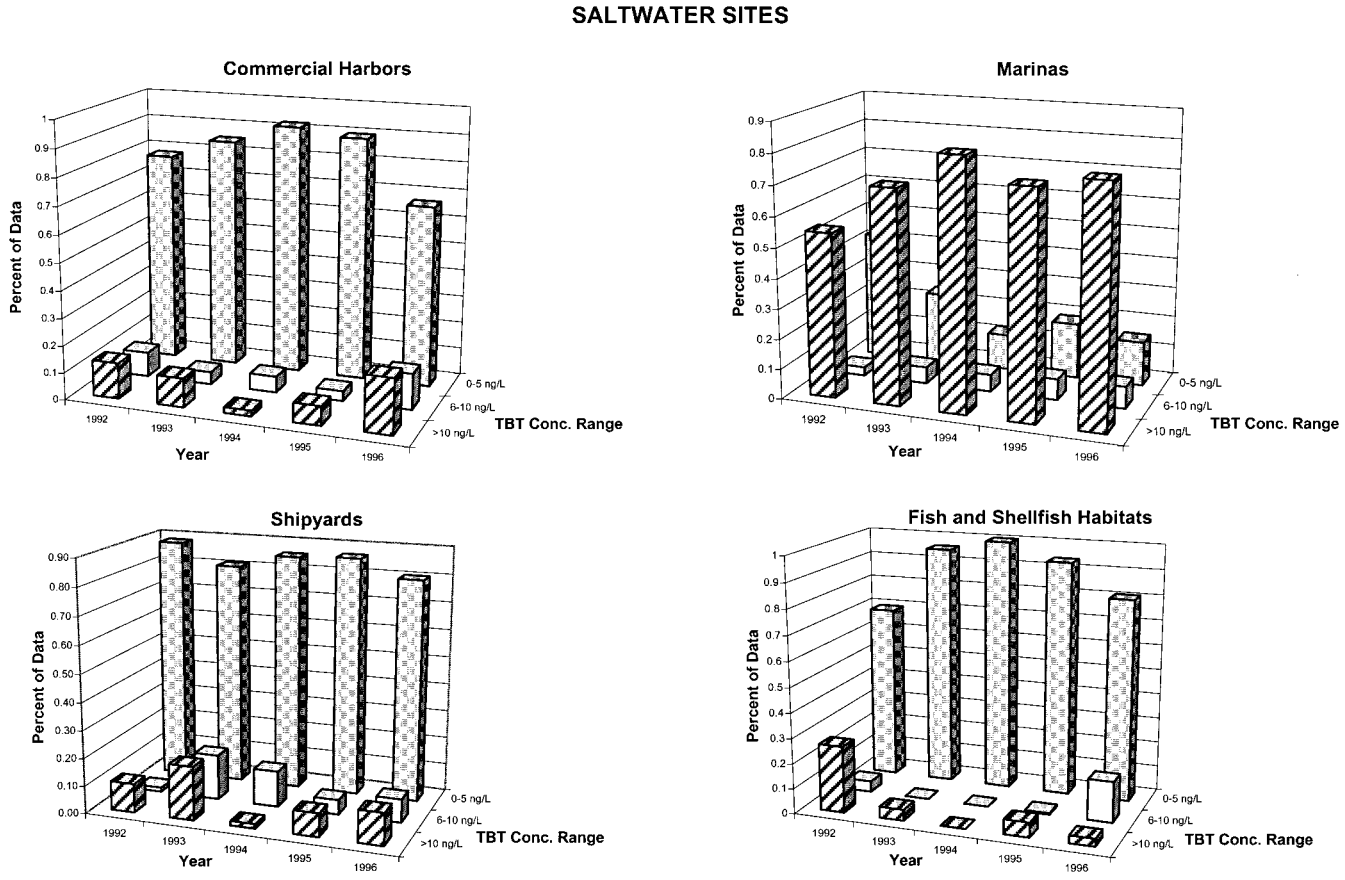

Fig. 2. Tributyltin concentrations in saltwater areas sampled.

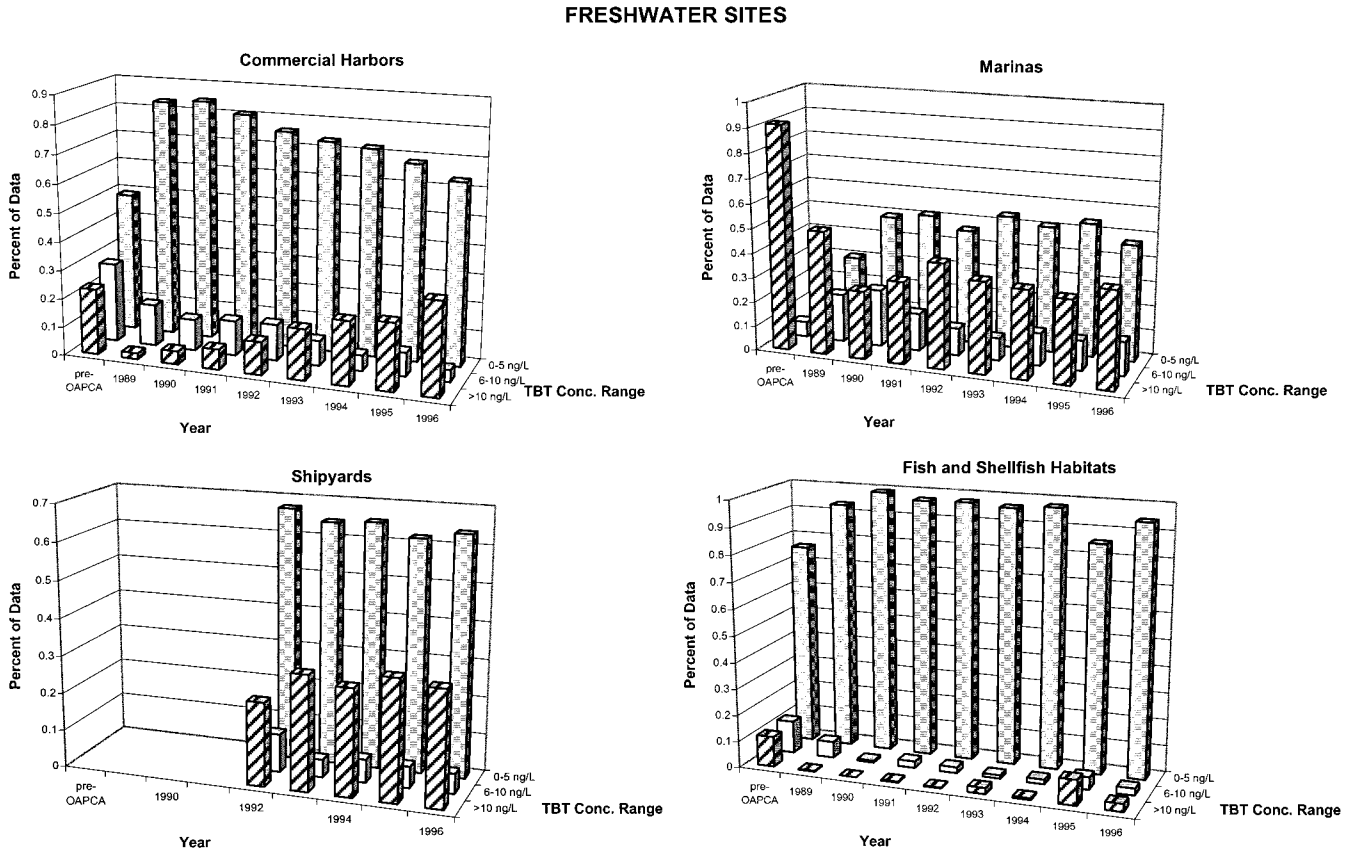

Fig. 3. Tributyltin concentrations in freshwater areas sampled.

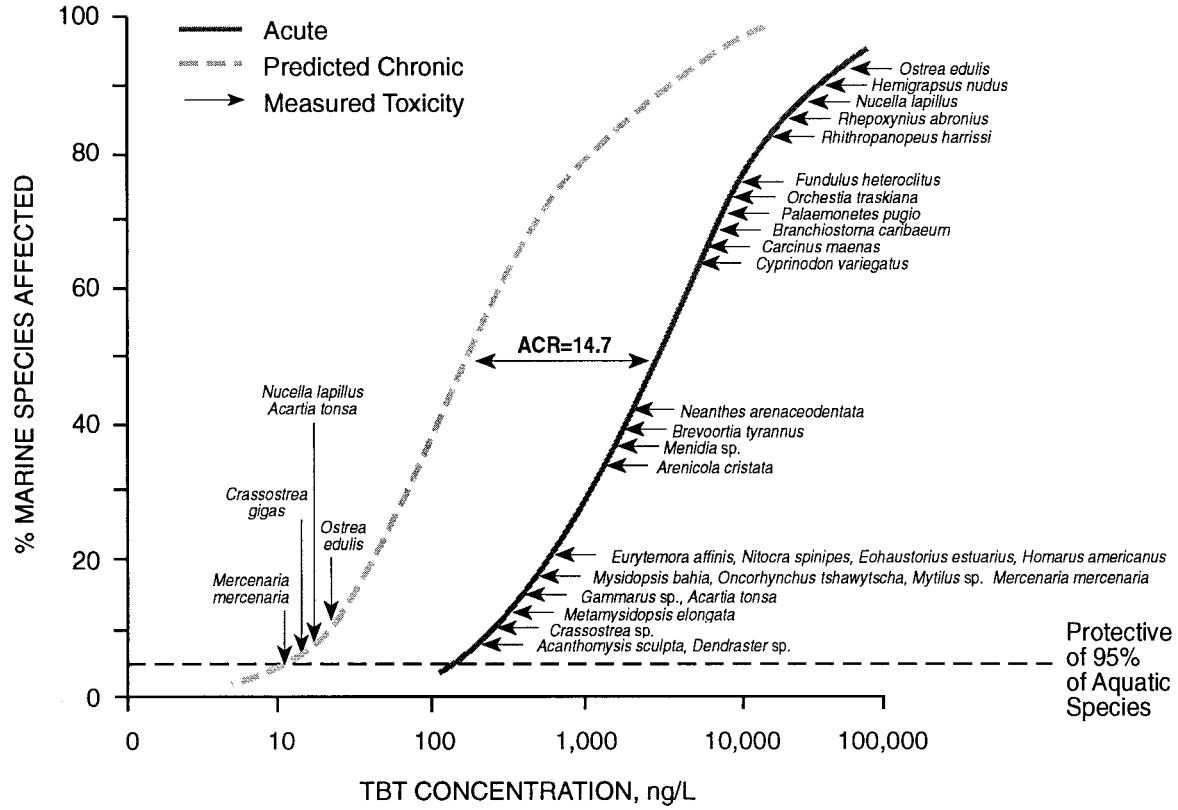

Fig. 4. Acute and chronic toxicity of tributyltin to saltwater species.

a better fit according to all three goodness-of-fit tests. We considered each PDF that was not dominated to be a valid EEC model and used it to calculate risks. Data sets corresponding to each PDF were simulated using @Risk [27].

We conducted sensitivity analyses using 1996 data to evaluate the uncertainty in the EEC model used by comparing our model results to those obtained from other valid EEC models. Similar expected risk predictions were obtained from all valid EEC models, so we did not pursue further the issue of how best to combine or select from among the valid EEC models. We used the best fitting model for data analyzed from each year and site type.

A variety of assumptions was applied to the exposure estimates, and these assumptions influenced the risk estimates. First, we assumed that the TBT measured in the environment was as bioavailable as the TBT in the laboratory experiments used to estimate toxic effects thresholds. Both the field (in situ) and laboratory data are total TBT concentrations. If dissolved ionic TBT is the bioavailable phase, as suggested by work with TBT [18] and metals [28], then the question arises as to whether the ratio of dissolved ionic to total TBT concentrations is the same in the field and the laboratory samples. The ratio of dissolved ionic to total TBT should be only slightly lower in the environment than in the laboratory, based on TBT's K_d [29] and suspended solids characteristic of study area surface waters (e.g., <100 mg/L). Therefore, total concentrations should only slightly overestimate risks.

Second, we assumed the EEC databases were sufficient to estimate not only the magnitude of exposure but also exposure frequency and duration. Given the number of samples (>70) measured quarterly at each site type, frequency appears reliably measured. By assuming that measured TBT concentrations will persist for the duration necessary for acute and chronic effects to manifest, our quarterly sampling may have overestimated the variability in acute and chronic EECs because variation increases with time between sampling. This would overestimate the probabilities in the upper tails of the risk distributions and overestimate expected total risks (ETRs).

Third, we assumed TBT's surface water concentrations accurately estimated risks, although they did not directly address toxicity via uptake of food or suspended particulates. As discussed earlier, most TBT uptake appears to occur via the water [18], considering the volume of water passed over the gills, bronchial uptake efficiency, TBT's relatively low octanol-water partition coefficients (median reported K_{ow} = 1,550 [30]), and relatively low bioconcentration factors (median value = 4,000 [30]). In addition, if one assumes equilibrium partitioning is at steady state, because compartmental fluxes occur much faster than the biological responses, risks based on water EECs should integrate those from the diet [20]. In the aggregate, these assumptions appear realistic. Last, we assumed that the Navy and U.S. EPA sites were representative of U.S. waters.

ECOLOGICAL EFFECTS CHARACTERIZATION METHODS

Tributyltin's acute and chronic toxicities to saltwater and freshwater organisms were characterized using an approach developed by the U.S. EPA for its water quality criteria [21]. Figure 4 (saltwater species) exemplifies how toxicity PDFs were represented for both freshwater and saltwater species tested for TBT sensitivity. The only difference is we used test data for all species rather than criteria concentrations protecting 95% of the species. We assumed the species tested represented the spectrum of species found in aquatic communities. To achieve enough phylogenetic diversity and spe-

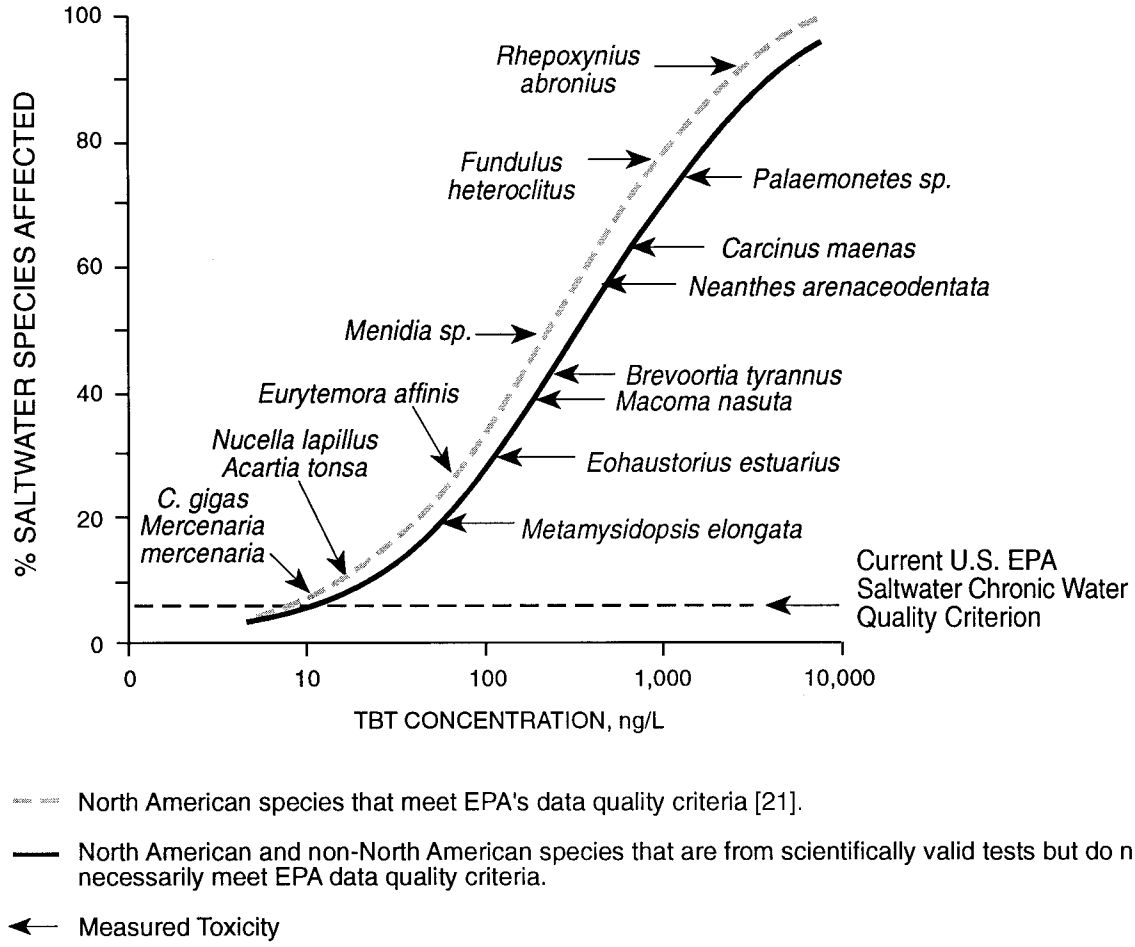

Fig. 5. Estimated chronic toxicity of tributyltin: United States versus cosmopolitan saltwater species.

cies sensitive enough to represent a generic aquatic community, the U.S. EPA [21] requires testing at least one species in at least eight different families. In freshwater, this comprises (1) Salmonidae, (2) bony fish (Osteichthys), (3) Phylum Chordata, (4) planktonic crustacea, (5) benthic crustacea, (6) insects, (7) a phylum other than Arthropoda or Chordata, and (8) any order of insect or other phylum not already represented. In salt water, required families include two chordates, a phylum other than Arthropoda or Chordata, either Mysidae or Penaeidae, three other nonchordate families, and any other family. The freshwater and saltwater TBT data sets used here met these criteria.

The toxicity data used (e.g., Fig. 4) comprised tests reported in the U.S. EPA water quality criterion document for TBT [5] and tests identified from a computerized literature search through June 1996. All data were expressed as TBT ion and were comparable in terms of endpoints (e.g., threshold effect concentrations) and quality control. Chronic toxicity data reflected actual tests and those estimated using an acute to chronic ratio of 14.7 [5], which was based on the geometric mean of ratios from tests of *Daphnia magna, Eurytemora affinis, Pimephales promelas,* and *Acanthomysis sculpta.* [5]. Actual chronic test results were used for five species ranking within the lower 10 percentile in sensitivity: *Nucella lapillus, Acartia tonsa, Crassostrea gigas, Ostrea edulis,* and *Mercenaria mercenaria.* The literature data were subjected to the data quality criteria in Stephan et al. [21] and rejected for the following reasons. Toxicity tests did not contain a control treatment, control mortality, or stress rates unacceptably high (e.g., >10% mortality) based on criteria stated in standard test protocols [31]. Species does not reproduce naturally in North America. Organism was previously exposed to substantial concentrations of the test material or other contaminants. Raw data were unreported. Endpoints were clearly not translatable to population-level effects, i.e., effects manifesting as mortality in acute tests and population-level effects in chronic tests, as originally proposed by Mount and Stephan [32].

Test data using biomarkers as endpoints (e.g., imposex, shell thickening, and phototaxis) were not used because they did not translate to population level effects, as discussed by Gentile and Slimak [33]. The U.S. EPA uses the foregoing approach in its water quality criteria documents, categorizing biomarker data as questionable and hence useful as auxiliary and discretionary information. Only population-level effects on survival, growth, and reproductive success were included, and growth effects were only included if a chronic reproductive effect or lethality was also reported. If several endpoints were measured in a chronic test, the most sensitive was used.

A second set of aquatic toxicological thresholds was also evaluated (Fig. 5). It included tests considered scientifically valid by U.S. EPA but not meeting all of their acceptance criteria. This data set included non-North American species and tests encompassing shorter or longer exposure durations than those specified in standard test protocols [31]. Otherwise, the data met U.S. EPA requirements [21].

Following WERF guidance, a logistic regression model was

Table 1. Example tributyltin (TBT) expected risk calculation: saltwater marinas in 1996

TBT concn. (ng/L)	% EECs[a]	% Taxa affected[b]	% Taxa at risk
0–5	47	0.041	0.019
6–10	14	5.7	0.80
11–20	23	9.3	2.1
21–30	9.0	13.9	1.3
31–40	3.4	18.2	0.62
41–50	1.3	21.9	0.28
51–60	0.96	25.2	0.24
61–70	0.35	28.5	0.10
71–80	0.31	31.0	0.096
81–90	0.12	33.1	0.04
91–100	0.16	35.6	0.057
101–161	0.15	42.7	0.064
Total expected risk			6

[a] EEC = expected environmental concentration.
[b] Percent taxa with chronic effects thresholds occurring within the range of TBT concentrations in the first table column.

applied to the acute and chronic toxicity data. In this model, the independent variable is the log of the genus mean acute value for acute toxicity or the log of the genus mean acute value divided by the TBT acute to chronic ratio for chronic toxicity. For the five most sensitive species where actual chronic tests were available, we used the threshold effect values. The dependent variable is each species' sensitivity ranking (r) relative to the total number of species tested (n), transformed to its logit, i.e.,

$$\text{Ranking in terms of probability } (p) = r/n$$

$$\text{Logit } (p) = \ln(p/1 - p)$$

This transformation converts a nonlinear regression into a linear regression. This model estimates the percentage of taxa expected to be acutely and chronically affected given exposure to a given TBT EEC.

RISK CHARACTERIZATION METHODS

Acute and chronic risks were estimated by comparing distributions for TBT EECs and effects thresholds. The estimated risk for any given EEC equals the percentage of taxa affected at that EEC. The probability associated with this level of risk is the probability that the stated EEC will occur. For the entire distribution of TBT concentrations [R(EEC)], the percentage of taxa expected to be at risk is plotted against [f(EEC)], the probability associated with the EEC. This creates the risk PDF. Expected total risk is found by summing (or integrating) the product R(EEC)·f(EEC). Expected total risk can also be considered a weighted average where risks associated with a given EEC are weighted by the probability of occurrence. Table 1 exemplifies the probabilistic risk estimation method. For example, a TBT concentration range of 0 to 5 ng/L encompasses 47% of the EECs, and within the 0- to 5-ng/L range, only 0.041% of the taxa will be affected. The percent taxa at risk is 47% × 0.041% = 0.019%. The statistical expectation of the risk is simply the sum (or integral in the continuous case) of the possible risk levels, each weighted by its probability of occurrence.

EXPOSURE CHARACTERIZATION RESULTS

The results of the exposure characterization are illustrated in Figure 6. Although only saltwater sites are illustrated, the exposure characterization was also conducted with data from the one freshwater area monitored, Lake Erie.

ECOLOGICAL EFFECTS CHARACTERIZATION RESULTS

The results of the ecological effects characterization are shown for saltwater species in Figures 4 and 5. The distributions of acute and chronic toxicity are shown in Figure 4; sensitivity rankings for some of the species are identified with arrows, including the five species actually tested for chronic toxicity. Figure 5 compares two TBT chronic toxicity distributions for 29 saltwater species. One of the distributions, duplicated from Figure 4, meets all U.S. EPA data quality criteria, including all North American species. The other distribution also meets these criteria, but includes tests of non-North American species and nonstandard exposure durations, for a total of 38 species. The distribution shown in Figure 5 probably is the most representative for cosmopolitan species, including related species, because it encompasses more taxa and responses than those embodied in Figure 4. However, we have based our risk estimates on Figure 4 because our assessment is limited to U.S. waters. Because Figure 4 suggests species are more sensitive to TBT than Figure 5, risks calculated from it will be higher.

RISK CHARACTERIZATION RESULTS

The risk characterization presents the results of the probabilistic risk analysis with the percentages of taxa expected to be at acute or chronic risk of exposure to TBT concentrations in the water column. First, we discuss the risks of acute and chronic toxicity on a regional basis. This is followed by a discussion of the risks associated with the different sources of TBT, a discussion of the results that would be obtained from a hazard quotient approach versus the approach applied herein, and, last, the ecological significance of the risks and uncertainties.

Risks posed to aquatic life by TBT concentrations in U.S. surface waters have varied significantly between regions of the United States and the sites monitored (Tables 2a–e). At most of the sites and regions monitored, risks were low, affecting ≤6% of the species even prior to OAPCA's passage in 1988. These risks seem lower than those based on comparing a specific TBT effect criterion (e.g., 10 ng/L) to a specific EEC value or frequency (e.g., 36% of EECs exceeded the criterion). By taking a community-based approach and considering the relative sensitivity of species within that community, ETR turns out to be less. Because TBT water column concentrations continued to decline over time, the majority of the characterization focuses on the ETRs calculated for 1996 because they represent the most recent data collected.

Risks of acute toxicity

Risks of acute toxicity have been low (≤3%) since prior to OAPCA because TBT's acute toxicity occurs at ≥110 ng/L using the logistic distribution in Figure 4. The U.S. EPA [5] has estimated the acute toxicity threshold at ≥356 ng/L. By 1993, there was no risk of acute toxicity in Puget Sound, Lake Erie, or Narragansett Bay. Acute risks occurred only within Galveston Bay's commercial sites, and by 1996, ≤1% of the aquatic taxa were at risk to acute toxicity at these sites.

Regional differences in chronic risks

Tributyltin concentrations typifying each of the site types differed between regions (see Figs. 2 and 6 for saltwater data),

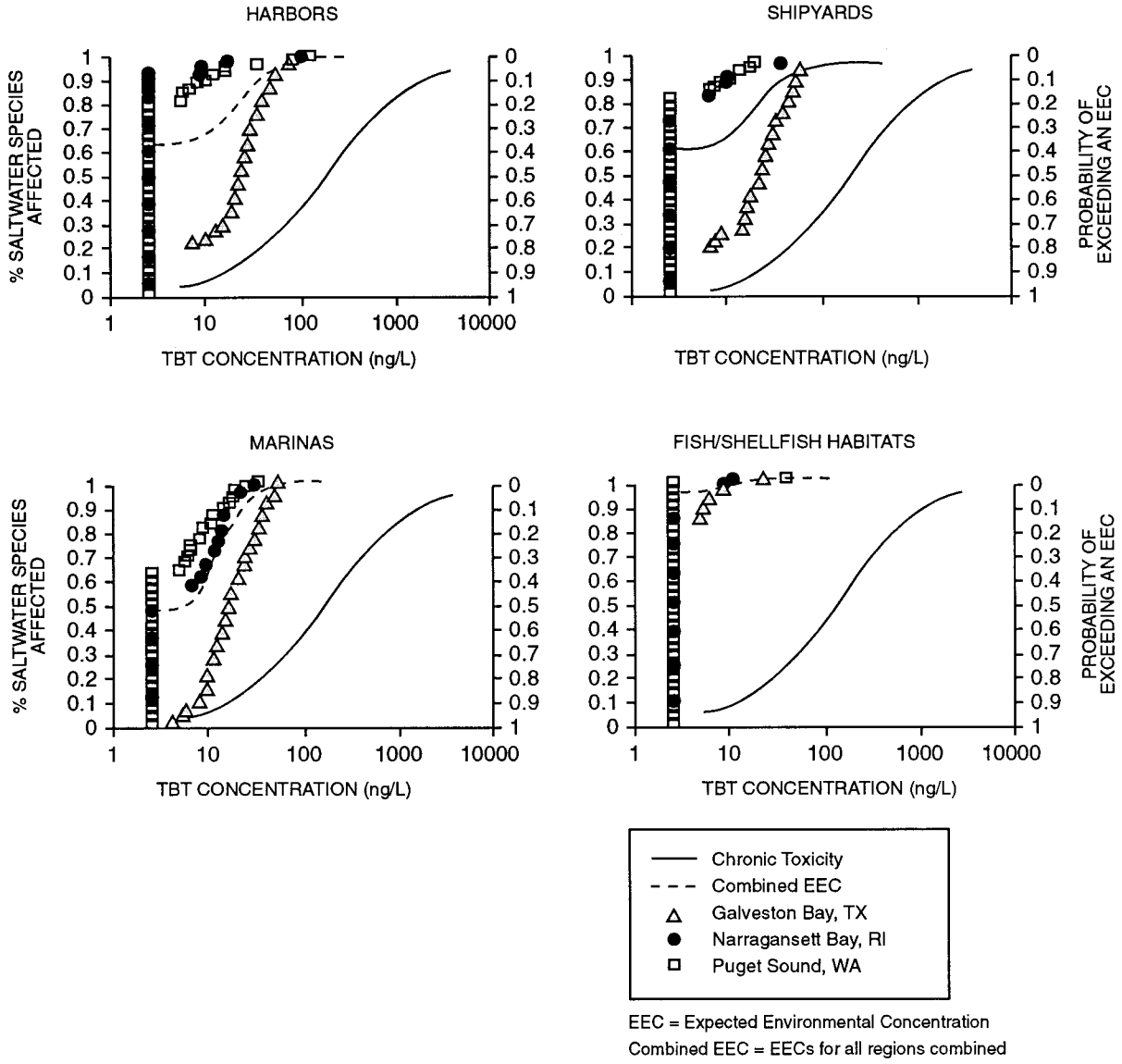

Fig. 6. Tributyltin risk characterization.

with chronic risks being higher (12—14% in 1996) in Galveston Bay and negligible (≤4%) in the other regions (Tables 2a–d). The ETR for Puget Sound and Narragansett Bay in 1996 was ≤3% at all site types. In Lake Erie, risks were <1% except at marinas (≤2%). In Galveston Bay, ETRs were 12 to 14% at all site types except fish/shellfish habitats (≤1%). The regional differences in risks reflect the higher EECs seen in Galveston Bay (Fig. 5 and Tables 2a–d). Leading explanations concerning Galveston Bay's elevated TBT concentrations include greater shipping activity and naturally elevated suspended solids (8–155 mg/L), which offers a substrate for TBT sorption [34].

Source differences in chronic risks

Expected chronic risk also varied greatly by site type (Table 2a). Over the 9+ years of monitoring (the Navy and U.S. EPA programs), ETRs have been highest in marinas (5–25%), followed by shipyards (5–6%), commercial harbors (<1–6%), and fish and shellfish habitats proximate to these sites (<1–2%). Although this reflects differences in exposure (i.e., differences in EEC distributions), different organisms also inhabit

Table 2a. Expected chronic risks for all saltwater areas monitored

	Expected risks (%)			
Year	Marinas	Shipyards[a]	Harbors	Fish/shellfish habitats
Pre-1989	25	—	5	2
1989	12	—	1	1
1990	7	—	0	0
1991	7	—	1	0
1992	8	6	2	0
1993	6	6	3	0
1994	6	5	4	0
1995	5	6	4	2
1996	6	5	6	1

[a] The Navy monitoring stations were classified into the site types used on the U.S. Environmental Protection Agency (U.S. EPA) mandated program. Because shipyards and commercial harbors were indistinguishable, all of the Navy data meeting the harbor designation were considered as such, whether or not they contained a drydock. The U.S. EPA-mandated program began in 1992, and shipyards per se were only monitored after this time.

Table 2b. Expected chronic risks for Galveston Bay waters

Year	Expected risks (%)			
	Marinas	Shipyards	Harbors	Fish/shellfish habitats
1992	18	15	9	1
1993	19	13	8	1
1994	13	12	10	1
1995	13	13	9	4
1996	13	12	14	1

Table 2d. Expected chronic risks for Puget Sound waters

Year	Expected risks (%)			
	Marinas	Shipyards	Harbors	Fish/shellfish habitats
1992	9	0	1	0
1993	4	2	3	1
1994	4	1	1	0
1995	3	1	1	1
1996	3	1	2	0

the various site types. The latter is not a factor in the risk estimates, which assume a diverse saltwater community is attainable in commercial sites.

The site type differences were also reviewed at the regional level using 1992 to 1996 data (Tables 2b–e). The pattern was similar for Puget Sound, Narragansett Bay, and Lake Erie, where ETRs were highest in marinas (2–9%), low in commercial harbors and shipyards (<1–5%), and even lower in fish and shellfish habitats (<1–2%). Although a similar pattern was seen in Galveston Bay, ETRs were higher (Table 2b). In Galveston Bay, ETRs in marinas were highest (13–19%), followed by shipyards (12–15%), commercial harbors (8–14%), and fish and shellfish habitats (1–4%).

Trends in chronic risks

Annual monitoring of each TBT site type from pre-OAPCA to 1996 reveals ETRs decreasing over time in saltwater marinas, the only site type where risks were high enough to track temporal trends (Table 2a). Prior to 1989, ETRs were as high as 25% in saltwater marinas, but by 1996, they had declined to ≤14% in Galveston Bay at all four site types and to ≤2% in all Puget Sound and Narragansett Bay site types except marinas (3–4%) (Tables 2a–e). Risks at the Lake Erie sites have remained low (0–2%) since monitoring commenced in 1992. To place these risks into context, the U.S. EPA water quality criteria seek to protect all but 5% of the species, and the Society of Environmental Toxicology and Chemistry [25] recommends protecting all but 10% of the species. Economically important species and those that influence the community out of proportion to their abundance or biomass (keystone species) are also protected.

Comparison of risk characterization approaches

Risk assessments often are conducted in tiers (phases), with a screening phase based on quotients usually conducted in tier 1 and a detailed phase based on probabilistic techniques conducted in tier 2. Risks suggested by the two approaches may appear quite different, reflecting dissimilar assumptions and calculation methods. With the quotient method, risks appear higher because the probabilities of exposure and effect are not considered; a quotient only compares the magnitude of the ratio between the EEC and the toxicological effect threshold. In 1996, for example, 40% of the EECs measured in U.S. saltwater marinas and 3% of those in proximate fish and shellfish habitats exceeded the U.S. EPA chronic criterion of 10 ng/L, the former suggesting substantial risk (Fig. 2). However, the WERF methodology [12] indicates the ETR to be ≤6% of all saltwater species in marinas and (1% in ecological sites, that is, on average, only <1 to 6% of the taxa are predicted to be at risk of chronic toxicity in the sites and regions sampled (Table 2a).

The measure of risk used in the WERF methodology is the percentage of aquatic taxa whose effects thresholds are exceeded by the EECs to which they are exposed. Using Figure 6 (marinas) as an example, 24% of all EECs measured in saltwater marinas in 1996 exceeded 16 ng/L. At the latter EEC, approximately 10% of the saltwater taxa are expected to be at risk of chronic toxicity, i.e., to incur adverse effects on some proportion of their population given the assumptions made concerning duration and bioavailability of TBT exposure. Consequently, the ETR is 2.4% (24% × 10%).

The most sensitive 10% of taxa tested include larvae of certain bivalves (e.g., *Mercenaria, Crassostrea*), gastropods (e.g., the snail *Nucella*), and crustacea (e.g., the copepod *Acartia*) [5] (Fig. 4). For example, a percentage of the quahog clam's (*M. mercenaria*) larvae are at risk when chronically exposed to 10 ng/L TBT at the veliger stage [5]. Because EECs in the range of (10 ng/L occurred in 1996 40% of the time in marinas, it can be assumed they occurred over sufficiently long time periods to encompass bivalve larval development (~21 d). In 1996, risks to quahog clam larvae occurred in Galveston Bay's commercial sites; risks in the other regions were lower, as will be discussed.

Ecological significance of the chronic risks

Concentrations >10 ng/L may have occurred long enough to pose chronic risk to species with short life cycles or sensitive life stages, such as developing bivalve larvae and reproduction of copepods such as *Acartia tonsa* [35]. Despite the ETRs to certain species sensitive to TBT, it is not clear how chronic

Table 2c. Expected chronic risks for Narragansett Bay waters

Year	Expected risks (%)			
	Marinas	Shipyards	Harbors	Fish/shellfish habitats
1992	2	0	0	0
1993	2	2	0	0
1994	2	1	0	0
1995	3	5	1	1
1996	4	1	1	0

Table 2e. Expected chronic risks for freshwaters (Lake Erie)

Year	Expected risks (%)			
	Marinas	Shipyards	Harbors	Fish/shellfish habitats
1992	2	0	0	2
1993	2	0	0	0
1994	2	0	0	0
1995	2	0	0	0
1996	2	0	0	0

effects at early life history stages would affect populations as a whole. For example, a variety of indigenous bivalve species whose larvae are expected to be as sensitive to TBT as those tested in the laboratory have been collected in all of the saltwater regions monitored. The primary bivalves collected include *Mytilus trossulus* in Puget Sound, *M. edulis* and *Arcuatula demissus* in Narragansett Bay, *Crassostrea virginica* in Galveston Bay, and *Dreissena polymorpha* in Lake Erie. In Galveston Bay, where TBT concentrations were higher, bivalve populations were not as large or as frequent at some of the monitoring sites, yet it is clear that exposure is either less than predicted or that compensatory mechanisms at later life history stages may offset reductions at earlier stages.

In addition, the quoted risks are exaggerated for marinas, commercial harbors, and shipyards because they presume a balanced, diverse, and productive marine community within these sites is an attainable use. Because marinas, for instance, are managed for small boats and may be significant sources of certain pollutants in addition to TBT, it is questionable whether mollusk production, for example, would be a resource management goal and an attainable use. Generally, it is not. In addition, poor hydraulic exchange within marinas affects food supply for filter feeders, and their depositional environment limits habitat, limiting the species that can live in them [36]. Similar constraints on biodiversity occur in shipyards and in the innermost reaches of commercial harbors where we sampled.

Uncertainties in risk estimates

There are two categories of uncertainties in our risk estimates: data variability and assumptions. The former includes TBT detection limits. Although our data include absolute TBT quantitative limits as low as 0.5 ng/L, the MDL was 5.5 ng/L, calculated following Federal Register 40 CFR 136, Appendix B. The practical quantification limit for TBT exceeds 10 ng/L using the analytical method of Uhler and Steinhauer [37], which was approved as a U.S. EPA method in February 1989. As mentioned earlier, EECs less than the MDL were set equal to one half the MDL. This was inconsequential to the risk estimates because any value less than the detection limit was associated with zero risk. Because risks resulted from the majority of EECs being at or near one half the MDL, however, this creates some uncertainty in calculated means. If detection limits were lower, the risks would decline further. This would also be the case if TBT EECs were inflated by noise around the detection limit. Expected improvements in TBT detection limits should reduce this uncertainty.

Assumptions concerning EEC duration and effect thresholds represent important uncertainties. We assumed every EEC occurred long enough to elicit responses in all the species tested, but if they did not, risks were overestimated. We also assumed exceedance of a species' effect threshold denoted risk, knowing that most thresholds represent estimates of NOECs and do not equate with effects. To distinguish effects on individuals and those on populations, further assessment is necessary [38].

SUMMARY AND CONCLUSIONS

Tributyltin monitoring from pre-1989 through 1996 at saltwater and freshwater sites around the United States has revealed declines in chronic risks from a high of 25%, prior to late 1988 legislation restricting TBT usage, to <1 to 14% in 1996, depending on site type and region. Most of the decline has been associated with marinas, which reflects limitations on use of TBT antifoulant paints on pleasure crafts. Since 1994, all risks have been associated with chronic rather than acute toxicity in all regions monitored. Most of the risks appear to have been localized to marinas (small boat basins). Low risks ($\leq$5%) have been observed in commercial harbors and shipyards in three of the four regions monitored for the past 5 years. Negligible risks ($<$2%) have been encountered since prior to 1989 in the fish and shellfish habitats adjacent to marinas, shipyards, and commercial harbors.

Risks were not uniform among regions of the United States but were inflated by TBT concentrations measured at a variety of stations within Galveston Bay, which have ranged from 1 to 19%. By 1996, risks were low ($\leq$4%) to negligible in the Pacific Northwest (Puget Sound, WA), Atlantic Northeast (Narragansett Bay, RI and MA), and the Great Lakes (Lake Erie, Ohio, Michigan, and Pennsylvania). Most (86–100%) of the aquatic species are not expected to be at risk from TBT surface water concentrations observed in 1996. The species at risk occupy the lower tenth percentile in terms of TBT sensitivity. They include the economically important larvae of bivalves, (*Mercenaria, Crassostrea,* and *Ostrea*) and the ecologically important zooplankter *Acartia.* However, these species generally were at risk only in marinas, sites managed for commercial uses rather than shellfish production. Galveston Bay was the exception; the most sensitive species were at risk in marinas, shipyards, and commercial harbors but not in proximate habitats considered suitable for fish and shellfish production.

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