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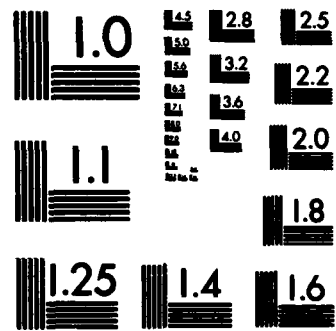
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THE BIOLOGICAL FATE AND EFFECTS

OF

ORGANOTIN COMPOUNDS

IN THE

MARINE ENVIRONMENT

John A. Strand

July 1983

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This report summarizes the readily available literature on the biological fate and effects of organotin compounds in the marine environment, with particular interest in tin compounds of potential use to the U.S. Navy in antifouling paints or hull coatings. The review distinguishes among organotin occurrence and distribution, bioaccumulation, chemical and biological degradation, biomethylation, effects of exposure, and mode of toxic action. The

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ABSTRACT

This report summarizes the readily available literature on the biological fate and effects of organotin compounds in the marine environment, with particular interest in tin compounds of potential use to the U.S. Navy in antifouling paints or hull coatings. The review distinguishes among organotin occurrence and distribution, bioaccumulation, chemical and biological degradation, biomethylation, effects of exposure, and mode of toxic action. The assembled information base is reviewed with sufficient depth to permit a reasonable determination of their adequacy for assessment and prediction of environmental impacts. A recommended program of research is also presented for purposes of generating enumerated data deficiencies.

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CONTENTS

1.0 INTRODUCTION

1.1 Purpose and Scope

The purpose of this study was to summarize the readily available literature on the biological fate and effects of organotin compounds in the marine environment, with particular interest in tin compounds of potential use by the U.S. Navy in antifouling paints or hull coatings. It was also the purpose of this study to outline a recommended program of research with the objective of generating missing data useful to the assessment and prediction of impacts associated with marine releases of organotin compounds.

1.2 Sources of Data

Among the data sources utilized were:

Technical Library at the Naval Oceans Systems Center, San Diego, California.

Technical Library at the Naval Oceanographic Office, Bay St. Louis, Mississippi, and its information retrieval services (DTIC).

Technical Library at the Naval Research Laboratory, Washington, D. C., and its informational retrieval services (DTIC, NTIS).

Technical Library at Battelle, Pacific Northwest Laboratories, Richland, Washington, and its information retrieval services.

Technical Library at the University of Washington, Seattle, Washington.

1.3 Organization of Report

Section 2.0 presents readily available data on environmental fate of organotin compounds distinguishing among occurrence and distribution, bio-accumulation, chemical degradation, biological degradation, and biomethylation. Section 3.0 discusses implications of entry or organotin compounds in aquatic food chains emphasizing effects of exposure and mode of toxic action. In Section 4.0, the assembled technical data are reviewed with sufficient depth and scope to permit a reasonable determination of their adequacy for assessment and prediction of impacts. In Section 5.0, a recommended program of research is outlined with the objective of generating those data deficiencies enumerated in Section 4.0.

2.0 ENVIRONMENTAL FATE

2.1 Occurrence and Distribution

In recent measurements, Hamagauchi et al. (1964) found tin concentrations between 0.30 and 1.22 μg/l in seawater from the northwest Pacific. Smith and Burton (1971) determined that concentrations of tin in seawater decreased from estuarine and shelf (0.02-0.04 μg/kg) to surface Atlantic waters (0.009 μg/kg) in the vicinity of the English Channel.

Zingaro (1979) reported that urban sewage sludges - representing gross "indicators" of heavy metal pollution and biocycling - contain unusually high tin concentrations (111-492 mg/kg dry weight). However, information is lacking on chemical forms, or bioavailability, of these anthropogenic sources. Zingaro (1979) also reported on the work of R. Braman which describes widespread dis-tribution of methyltins at extremely low concentrations in diverse environments. Braman found that methyltins range from 2.5-13.5 μg/l in seawater to rainwater, and comprise about half of the total tin detected.

Hodge et al. (1979) found compounds yielding dimethyltin dihydride in San Diego Bay at levels of 15 to 45 ng/l, usually exceeding concentrations of methyltin and inorganic tin. Compounds yielding n-butyltin trihydride and di-n-butyltin dihydride were found in Lake Michigan at 10 to 1600 ng/l, also higher than methyl species and inorganic tin.

Hallas and Cooney (1981) examined sediments and water samples from nine stations in Chesapeake Bay for tin content and determined that tin concentrations were higher (3.0 to 7.9 mg/kg) at sites impacted by human activity than at open water sites (0.8 to 0.9 mg/kg). Highest levels (239.6 mg/kg) were recorded in Baltimore Harbor which is impacted by both shipping and heavy industry. There was also significantly more tin in sediments than in water samples.

2.2 Bioaccumulation

Seawater from the Bay of Naples was found to contain concentrations of 0.020 to 0.024 µg of Sn/kg (Smith, 1970). In _Ascidia mentula_, the concentration in the internal organs was relatively high at 15 mg/kg, while the test (integument) was depleted of tin. The concentration of tin in the internal organs representated a concentration factor of 10^6 when compared with seawater. In cephalopods (_Sepia officinalis, Octopus vulgaris_), the concentration of tin was significantly higher in the liver than in muscle. Algae contained tin in concentrations similar to or greater than most animal forms.

Stroganov et al. (1973) studied the distribution of ^{117}Sn labelled triethyltin chloride in _Cyprinus carpio_ as a function of water concentration and time. They concluded that disbributions in organs and tissues were not uniform. Maximum quantities were found in the bile, blood, and liver; lesser quantities were found in bone and muscle. Triethyltin chloride was concentrated in the gall bladder. Its' content was 10-100 times greater than in soft tissues and 100-1000 times greater than in the surrounding medium.

Brown et al. (1977) injected labelled bis (tri-n-butyltin) oxide into rats and obtained data which suggested that this compound was rapidly turned over by these organisms. He concluded that there was no evidence to suggest bioaccumulation of the compound in a manner similar to methyl mercury.

Young and Alexander (1977) reported abnormally high levels of a number of potentially toxic trace metals including tin in tissues of bay mussel, _Mytilus edulis_, collected from areas of either vessel activities or municipal wastewater discharges in the Southern California

Bight. Analyses of tin in mussels collected from Newport Harbor and Newport Beach revealed concentrations of 3.6 mg/kg in digestive gland, 5.4 mg/kg in gonad, and < 0.5 mg/kg in muscle.

More recently, Blair et al. (1982) studied uptake and possible metabolic transformation of tri-n-butyltin cation by tin-resistant estuarine bacteria (Pseudomonas) isolated from sediments in Baltimore Harbor. The bacterial isolates were found to accumulate tributyltin from 3.7-7.7 mg tin per g dry weight of cells by a non-energy requiring process, probably by adsorption to the cell envelope. However, liquid chromatography-atomic absorption spectrophotometry and tin-selective purge and trap flame photometric gas chromatography failed to reveal transformation products (di- and monobutyltin) suggesting that the issolates accumulated but did not metabolize tributyltin.

2.3 Chemical Degradation

Zuckerman et al. (1978) generalized that progressive cleavage of organic groups from tin is dependent upon the type of organic compound, the number of organic constituents, and the solvolytic conditions. Rate of removal of aliphatic groups decreases with increasing size of the group, and unsaturated and aromatic groups are cleaved more rapidly. Solvolytic reactions, however, represent extreme pH conditions (pH < 1 or > 14), and half-lives range from one minute to 115 days, depending upon these conditions and specific organotin compounds studied. The inorganic anionic groups also react with water and air to cleave from tin in a hydrolysis-oxidation to yield stannols and oxides. Thus, successive reaction of both parts of the molecule leads ultimately to completely inorganic hydrated tin oxides.

Zuckerman et al. (1978) also reported on the work of Engelhart and Sheldon which describes the degradation of the antifouling agent tributyltin fluoride in seawater. The compound was found to hydrolyze very rapidly in low concentrations in seawater to yield the chloride and oxide. Carbon dioxide is available to react with the oxide to form carbonate. And, ultimately through sunlight and oxygen, hydrated tin oxide is formed.

In similar degradation studies of triphenyltin and tributyltin in distilled water and artifical seawater, Monaghan et al. (1979) found that $(n-C_4 H_9)_3 Sn -$ was the only organotin structural unit found in the chloroform extracts of tributyltin suspensions, but several different units were found in

extracts of triphenyltin suspensions. Structures such as R_3SnOH and $R_3SnOSnR_3$ were eliminated as possible soluble species since O-H and Sn-O-Sn stretching vibrations were absent in the infrared spectra of the residues of the chloroform extracts.

2.4 Biological Degradation

Cremer (1958) showed both with a rat liver microsomal enzyme system and with rats _in vivo_ that tetraethyltin underwent dealkylation to yield a triethyltin derivative. Casida et al. (1971) determined that the triethyltin derivatives are biologically oxidized to diethyltin derivatives. Subsequently, Bridges et al (1967) established that diethyltin derivatives are degraded _in vivo_ to monoethyltin derivatives.

In related studies, Kimmel et al. (1977) determined that microsomal monooxygenase metabolism of tributyltin acetate yielded α-, β-, γ-, and δ- hydroxybutyldibutyltin derivatives. The major metabolites, the α- and β- hydroxy compounds underwent dealkylation reactions under acidic conditions to form dibutyltin derivatives and 1-butanol and 1-butene, respectively. The γ- hydroxy compound was further oxidized to the corresponding ketone. Tetrabutyltin yielded tributyltin derivatives via β- and possibly the α- hydroxybutyltributyltins. Dibutyltin diacetate underwent monooxygenase or monenzymatic cleavage to butyltin derivates. Fish et al, (1978) working with tributyltin acetate concluded that the primary biological oxidation reaction is one of hydroxylation of carbon-hydrogen bonds that are α, β, γ, and δ to the tin atom. Also, a free radical process accounts for the predominance of β and the significant quantity of α-carbon-hydroxylation in n-alkyltin compounds. Data was presented to suggest that triphenyltin derivatives are not hydroxylated or dealkylated by a monooxygenase system although _in vivo_ studies with rats revealed dealkylation products.

More recently, Arakawa et al. (1981) studied dealkylation and distribution in rabbits and determined that upon intravenous injection, tetraethyltin was quickly distributed to the liver, whereas both tetrapropyltin and tetrabutyltin were slowly distributed. Tetraethyltin was more readily converted into the corresponding trialkyltin than tetrapropyltin; which, however, was more readily converted into trialkyltin than tetrabutyltin. About 20% of the tetraethyltin, 4% of the tetrapropyltin, and 1% of the tetrabutyltin were converted to the

5

corresponding trialkyltins, respectively, within three hours after treatment. Thus, the extent of formations of trialkyltins decreased as the size and stability of the alkyl groups increased.

Brown et al. (1976) compared the biological half-life of organotin with inorganic tin in the mouse using ^{113}Sn labelled bio-tributyltin oxide and metallic ^{113}Sn respectively. The effective biological half-life of ^{113}Sn was estimated to be 29 days and was not changed by injection of an excess of unlabeled tin. During the first several days, tributyltin was eliminated rapidly from the mouse. At later times, the turnover rate of tributyltin approached that of organic tin.

2.5 Biomethylation

Microbial methylation of tin has been reported for Pseudomonas isolated from Chesapeake Bay (Huey et al., 1974). In the laboratory, tin (II) is methylated at 20°C under nitrogen at pH 1 in 1.0 $\underline{M}$ aqueous salt solution in the presence of methylcobalamin at $\underline{ca}$ 5×10^{-4}M with a single electron oxidizing agent such as iron (III) chloride. Methylation occurred at a rate of $1.4M^{-1}s^{-1}$ in 10 - to 100 - fold excess of tin (II). Huey et al. (1974) also indicated that methylation of tin by Pseudomonas was not reported past the monomethyl stage. To the contrary, materials from anaerobic sediments removed from San Francisco Bay converted $(CH_3)_3SnOH$ to $(CH_3)_4Sn$ over a period of 80 days. The conversion demonstrated both biotic and abiotic components (Guard et al., 1981). Similarly, Blair et al. (1981) have indicated that the volatile methylated species produced by Pseudomonas from Sn (IV) and to a lesser extent from Sn (II), included tetramethyltin $(CH_3)_4Sn$ and a number of hydritic methylstannes $(CH_3)_n S_n H_{4-n}$, n=2, 3). More recently, Hallas et al. (1982) provided evidence of production of both dimethyl and trimethyltin species from inorganic Sn (IV) by sediment microflora.

Zingaro (1979) reported on separate studies conducted by Huey and Brinkman at the National Bureau of Standards Laboratories which demonstrated that hydrated trimethyltin cation was capable of abiotically transferring CH_3 to aqueous Hg^{2+} at a rate competitive with biomethylation of tin. These studies led to other experiments in which it was learned that Pseudomonas, metabolizing under dual stress of Sn^{4+} and Hg^{2+} yielded not only Hg, but also CH_3Hg+. As

6

well, the reaction required the presence of the biogenic CH_3-Sn metabolite. Guard et al. (1981) and Chau and Wong (1981) also reported that methylated tin species may result from abiotic as well as biotic processes.

3.0 TOXICOLOGY

3.1 Effects of Exposure

The effects of inorganic tin on frustular ultra structure of the marine diatom, Nitzschia liebethrutti were studied by Saboski (1977). At 1.5 mu.g atom/l, abnormalities included reduction in length/width ratios, fused cardinal dots, and reduction in number of cardinal dots per frustule. Curved raphes and cardinal dots alligned parallel to the raphe were also observed.

Frick and DeJimenez (1964) investigated the molluscidal properties of tri-n-phenyltin oxide, tri-n-butyltin acetate, and tri-n-phenyltin acetate against representative life stages and sizes of the snail, Australorbis grabratus. Tri-n-butyltin acetate and tri-n-propyltin oxide gave very similar results, that is, in 24 hr exposures, the LC_{90} values for either chemical did not exceed 0.115 mg/l for hatched snails. Against 4 day eggs, however, tri-n-butyltin oxide showed an LC_{90} of 0.45 mg/l as compared with only 0.17 mg/l for tri-n-propyltin. Under the conditions of the tests, tri-n-phenyltin acetate was significantly less effective than either of the other two compounds.

Employing the snail, Biomphalaria glabrata, Hopf et al. (1967) found the LD_{50} for triphenyltin acetate and tri-n-propyltin oxide to be 0.05 mg/l. Similar tests with tri-n-butyl acetate and triphenyltin fluoride gave LD_{50} values of 0.1 - 0.3 mg/l and 0.01 - 0.55 mg/l respectively.

Polster (1970) studied the toxicity of tributyltin oxide, acetate, chloride, oleate, laurate, and benzoate employing bacteria, fungi, phytoplankton and mammals. LD_{50} values for lower life forms (bacteria, fungi, plankton) ranged between 0.082 - 0.1 mg/l; while values for rats and mice fell in the range of 100 - 250 mg/kg.

Stroganov et al. (1970) investigated the relationship of toxicity to chemical composition of organometallic compounds of the type $RnMX_{4-n}$ (R = Me, Et, Pr; X = AcO_2, OH_2, Cl; M = Si, Ge, Sn, Pb) employing Daphnia magna, Chlorella vulgaris, and Scenedesmus quadricauda. They

7

determined that the most toxic compounds contained chloro, methyl, ethyl groups. The organotin and lead compounds were toxic for Daphnia at 0.02 - 0.5 mg/l, and organosilicon compounds were toxic to Scenedesmus at concentrations of 0.01 mg/l after 15 days.

Weisfield (1970) determined that the common guppy, Lebistes reticulatus, was sensitive to bis (tri-n-phenyltin) oxide at concentrations less than 1 mg/l. Other tests on Lebistes with triphenyltin hydroxide showed that 0.1 mg/l was lethal to 43 percent of the test organisms after 19 hr exposure, while 100 percent died in 48 hr (Hopf et al. 1967).

The effects of low concentrations of tributyltin chloride on growth and reproduction of the snail, Lymnaea stagnalis, were studied by Stroganov et al. (1977). The minimum lethal concentration was found to by 10^{-1} mg/l. In the range of 10^{-6} - 10^{-2}, effective food assimilation was diminished, the soft body either hydrated or dehydrated, the shell became thin, and reproduction was suppressed.

Chliamovitch and Kuhn (1977) studied both acute and sublethal effects of bis (tri-n-butyltin) oxide on the fish species Salmo gairdneri and Tilapia rendalli. Based on the loss of rheotaxis, the 24 hr EC_{50} for trout was 30.8 mu.g/l, while a value of 53.8 mu.g/l was reported for Tilapia. A histo-pathological study on trout showed that concentrations from 5.85 - 0.0117 mg/l resulted in damage to gill epithelium. At 0.023 - 1.17 mg/l, packed cell volume, Hb concentration, and erythrocyte count increased. Swelling was observed in cell volume at 1.17 mg/l, and shrinkage at 0.053 mg/l. It was thought that bis (tri-n-butyltin) oxide interfered with the process of respiration. A safe level of 0.1 mu.g/l was suggested for these species.

Responses of other non-target species to trialkyltin oxides were studied by Laughlin et al. (1980). Tributyltin oxide concentrations of 1 ppb were lethal to more than 90 percent of developing lobster, Homarus americanus, while control survival was about 50 percent during the same period (22 days). Similarly, tributyltin oxide produced a 50 percent mortality in crab larvae, Hemigrapsus nudus, at 25 ppb in 6 days, while control mortality for the same period was only 5 percent.

Zingaro (1979) indicated that for a given R group, mammalian toxicity increased in the order $RSn^{3+} < R_2Sn^{2+} < R_3Sn \sim R_3Sn^+$, with tetraorganotin

toxicity dependent upon the rate of oxidation to R_3Sn^+. In contrast, for a given structure such as R_3Sn^+, mammals show decreasing toxicity to R = ethyl > methyl > propyl > butyl > phenyl, although doses are found to be species dependent. For bacteria and fungi, however, this order appears to be reversed. Also, iso-alkyl derivatives are generally more lethal than their normal-alkyl isomers.

The relationship of structure to toxicity of tin compounds was also studied by Wong et al. (1982). Organotin compounds were found to be more inhibitory to primary production and reproduction of green and blue-green algae than inorganic tin compounds. Toxicity varied with the number and nature of the organic group. Trialkyltin compounds were the most toxic, followed by dialkyl and monalkyl tin compounds. Within a given alkyl series, the longer the carbon chain, the greater the toxicity; that is, phenyl- and butyl- tin compounds were more toxic than propyl-, ethyl-, and methyltin compounds. Wong et al. (1982) also observed a direct relationship between toxicity and partition coefficients of trialkyltin compounds. Triphenyl-, tributyl-, and tripropyltin compounds that were soluble in octanol were significantly more toxic than the relatively insoluble triethyl- and trimethyl compounds.

3.2 Toxic Mode of Action

Barnes and Magos (1968), Aldridge and Street (1971), and Rose and Aldridge (1972) conducted extensive studies on mammals and established that trialkyltins, including tributyltin, inhibit the vital process of oxidative phosphorylation. Similarly, Kahn (1968) reported that n-butyltin chloride inhibited photophosphorylation in isolated chloroplasts of Euglena gracilis. Stoner (1966) determined that triphenyltin compounds produced similar effects.

Boulton et al. (1971) studied the effects of triethyltin sulfate on the metabolism of ^{14}C labeled glucose and ^{14}C labeled acetate by exposing barnacles, Elminius modestus, to concentrations of this antifouling compound which were lethal after 30-36 hr. The changes in metabolism observed suggested that pyruvate utilization was restricted via pyruvate dehydrogenase.

From more recent studies with mammals, Aldridge (1976) showed that triorganotins derange mitochondrial function in three different ways: 1) by secondary responses caused by discharge of a hydroxyl-chloride gradient across

9

mitochondrial membranes; 2) by interaction with the basic energy conservation system involved in the synthesis of ATP; and,-3) by an interaction with mitochondrial membranes to cause swelling and disruption.

Seinen et al. (1977) determined that both di-n-butyltin dichloride and di-n-octyltin dichloride in rats decreased the delayed hypersensitivity reaction, allograft rejection, humoral immune response against sheep red blood cells, hemagglutination, and hemolysin response. These results were attributed to a selective inhibition of T-lymphocyte activity. It was also determined that immune suppression was most pronounced in animals exposed to the chemicals during the developmental phase of the lymphoid system.

Zuckerman et al. (1973) suggested that monomethyltins were not potent neurotoxins such as methyl mercury derivatives because the lipid solubility of monomethyltins was too low.

Dibutyltin dichloride was investigated for its ability to induce hepatic fibrosis in rats (Yermakoff et al. 1979). Hepatic fibrosis is recognized as one of the sequelae of inflammation and chemically induced liver damage. Dibutyltin dichloride administered by oral intubation (10 and 20 mg/kg) every other day for 12 days produced extensive inflammation in portal tracts, biliary damage, fibrosis, necrosis, infarcted areas, and granulomatous tissues. In higher dose groups, increases in hydroxyproline, prolyl hydroxylase, and relative collagen synthesis in vitro were observed at the end of the 12 day period. These results suggested that dibutyltin induced fibrogenesis was the result of biliary damage and/or inflammatory processes rather than direct stimulation of fibroblasts.

4.0 SUMMARY AND CONCLUSIONS

Tin released to the marine environment from antifouling paints may be in the form of a number of compound types varying in solubility and affinity for biological organisms. Sediments act as a "sink" for organotin compounds and both plant and animals concentrate tin by a factor of at least 10^6. No evidence was presented to suggest that organotins are bioaccumulated in a manner similar to methyl mercury. Most organotins in seawater are thought to rapidly hydrolyze to the chloride or oxide. Biologically, they are oxidized to

monoalkyltin derivatives by either dealkylation or hydroxylation. Microbial methylation of tin has been reported. Also, microbiologically produced methyltin species were shown to methylate Hg (II) ion. However, again it was suggested that methyltin compounds did not pose the hazard experienced with methyl mercury compounds.

Organotin toxicity was demonstrated in a variety of organisms including bacteria, fungi, algae, molluscs, crustaceans, fish, and mammals. Generally, toxicity was found to peak with ethyl substituted tins, and decreased with increasing side chain length. Molluscs, crustaceans, and fish were found to be significantly more sensitive than mammals.

Organotins (trialkyltins) in general can be classified as nonspecific metabolic inhibitors. Inhibition of specific enzymes or enzyme systems (pyruvate dehydrogenase) have been attributed to triethyltin sulfate. However, most effects of trialkyltins including tributyltin have been on oxidative phosphorylation in animals, and similarly, photophosphorylation in plants. Trialkyltins also likely bind to mitochondrial proteins and derange mitochondrial function.

However from the literature review, it is evident that the existing data base is incomplete, often contradictory, and of questionable value for the prediction and assessment of impact associated with marine releases of organotin compounds. The lack of information on speciation of organotin compounds of potential use as antifouling agents, in particular, complicate this problem. Very little data are available on the fate of transformation products in sediments or the potential for bioaccumulation in food chains leading to man. Present research also does not include studies over longer periods and at lower levels of exposure to detect potential chronic or sublethal levels of stress. Toxicity is highly dependent on compound type and species of test organism making it difficult to predict safe levels in ecosystems. And, tests under site specific conditions and utilizing indigenous species have yet to be conducted.

5.0 RECOMMENDED RESEARCH PLAN

5.1 Objectives and Scope

This recommended research plan includes laboratory and field studies to

determine the fate of organotin compounds and their transformation products in the marine environment, with particular reference to food chains leading to man, and the effects of organotin compounds and their transformation products on marine biological systems. The research is also designed to provide a means of biologically screening organotin compounds of potential use in antifouling paints or coatings, and to provide a reference data base likely to be required for a generic environmental impact statement and various federal and state permits.

5.2 Biological Fate

The purpose of this research is to determine the potential for transfer of organotin compounds or their transformation products in food chains leading to man. This work includes studies on environmental fate, biological transport, and environmental pathways modeling. Research on environmental fate emphasizes investigations on the physical, chemical, and biological processes governing dispersal of released organotin residues into available ecological compartments (air, water, and sediments). Biological transfer studies are directed to evaluate the potential for organotin residues to enter aquatic food chains and accumulate, or to concentrate through food chains, thereby increasing the risk to man. Environmental pathways modeling is useful to assess the relative importance of major routes by which man or other organisms may be affected by organotin residues, and ultimately, to predict levels of exposure to man and his resource organisms.

The specific research tasks are:

1. Uptake and retention of organotin compounds should be measured in flow-through systems employing test organisms representing several trophic levels (herbivore, detritivore, carnivore). Water and sediments may be treated in separate experiments. Rate constants for uptake and depuration for organs and whole body should be measured. Bioconcentration factors and biological half-lives (T 1/2) should be calculated.

2. Longer-term uptake and retention (60-days) may be determined

using a simple food chain consisting of a food organism and
consumer organism (fish). Distinction is made between fish
receiving loading from the water column, and fish receiving
loading from their forage. Particular attention is focused
on the potential for increased residue accumulation in fish
compared to the forage species. Rate constants for uptake
and depuration for organs and whole body should be measured.
Bioconcentration factors and biological half-lives (T 1/2)
should be calculated.

3. Analysis of the water column and sediments in 1 and 2 above
may provide information on the availability of organotin
compounds and their transformation products to test organisms.
This approach provides data on the rates at which component
concentrations are altered, by chemcial and or microbial action,
physical processes such as adsorption, photo-oxidation, and
solubilization.

4. Longer-term (60-days) uptake and retention should also be
determined in larger, flowing seawater (transition) systems.
As before, test organisms representing several trophic levels
are employed, and rate constants for uptake and depuration
may be calculated. Chemical fate of organotin residues in
the water column and in sediments are determined concomitantly.
Physical and microbiological factors mediating uptake and
depuration may also be studied.

5. Seasonal and annual variations in uptake and retention of
organotin residues in natural ecosystems should be determined
in proximity to an operating facility or remotely.

6. Environmental pathways modeling is conducted to determine
the relative importance of major routes by which man or other
organisms may be exposed to organotin compounds and will serve

to coordinate the efforts of both biology and environmental chemistry needed to evaluate these routes. Initial models will help prioritize and evaluate the adequacy of research. Ultimately, this effort will provide a tool for predicting levels of exposure to man and his resource species from organotin residues delivered through various environmental routes.

5.3 Biological Effects

The purpose of this research is to determine the possible direct or secondary effects of organotin compounds or their transformation products on representative aquatic organisms. This work includes studies on chemistry, acute effects, chronic or sublethal effects, behavior (avoidance or attraction), and system responses. Chemical analyses are necessary to estimate the concentration of organotin test organisms encounter. Acute and chronic studies are directed to determine short (96-hour) and long-term (30-day) effects of organotin exposure respectively. Behavior studies will aid interpretation of acute and chronic test results. And, systems level studies are designed to detect and measure the potential for population, community, and ecosystem damage.

The specific research tasks are:

1. Potential acute effects of organotin compounds or their transformation products should be determined by employing several distinct aquatic species and life stages. In general these tests are designed to measure mortality (LC_{50}) after short-term (96-hr) exposure to test materials under selected water quality conditions. Flow-through systems should be employed to avoid low D.O. levels resulting from test organism metabolism, bacterial oxygen demand, and waste breakdown. Test organisms should represent several trophic levels (herbivore, detritivore, carnivore) and have considerable experimental history.

14

2. Potential chronic and sublethal effects should also be
    determined with several distinct aquatic species and life
    stages. These tests are designed to measure long-term
    survival, growth rate and reproductive potential for
    organisms exposed to test concentrations that are not acutely
    lethal. Depending upon the species, test organisms should be
    exposed for up to 60 days in flow-through systems. Test
    organisms are selected as determined in (2) above.

3. Behavioral responses (avoidance or attraction) to organotin
    compounds or their transformation products are determined
    using fish and mobile crustaceans or molluscs.

4. Chemical analyses are performed in association with aquatic
    toxicity testing in (1), (2), and (3) above and should consist
    of monitoring of organotin concentrations as well as more
    detailed procedures for determining exposure concentrations of
    transformation products.

5. Flowing seawater (transition) systems should be employed to
    study the interactions between organotin compounds and water,
    sediments, and organisms. Organisms should include a primary
    producer, herbivore, detritivore, and fish. This system will
    allow evaluation of partitioning and transport of released
    material into water, sediments, and organisms, and measurement
    of effects on long-term survival, growth, feeding, and
    population responses under more nature conditions than in the
    laboratory.

6. Results of studies outlined in (5) above may be used to
    develop ecosystem effects studies in proximity to an
    operating facility and remotely.

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