303(d) Comment Letter Files

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FISH & MUSSEL DATA, LETTER APPENDICES

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APPENDIX 1

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DDT ANALYSIS FOR THE NEWPORT BAY WATERSHED

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DDT ANALYSIS FOR THE NEWPORT BAY WATERSHED

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EXECUTIVE SUMMARY

The Santa Ana Regional Water Quality Control Board (Regional Board) is currently working to revise EPA's 2002 TMDL for organochlorine compounds in the Newport Bay watershed and to develop an implementation plan for the TMDL. Regional Board staff and authors of separate studies have asserted that these compounds – most notably DDT – have the potential to cause impacts, including chronic toxicity and eggshell thinning in avian populations, at concentrations currently found in the watershed.

The comprehensive literature review and review of newer data presented here indicates that under current watershed conditions, relevant wildlife populations are not exposed to levels of DDT that would cause *chronic* toxicity. DDT concentrations have been declining in the environment since DDT was banned in 1972 and will continue to decline in the future, making it highly unlikely that DDT concentrations in wildlife tissue will increase in the future from these nontoxic levels. Species examined in the literature review include the brown pelican, the osprey, cormorants and terns, and a variety of large marine mammals. Available evidence also indicates that organochlorines are not causing *acute* toxicity to aquatic species and wildlife in the watershed at current levels. Recent studies of acute toxicity in San Diego Creek and Newport Bay have concluded that acute toxicity is not caused by organochlorine compounds, but rather is likely attributable to organophosphate, carbamate, or pyrethroid pesticides (Lee and Taylor, 2001; Bay et al., 2004). Given that current DDT levels in the watershed are below levels that cause toxic effects, establishing a TMDL that would further limit DDT loads is unjustified.

Review of the scientific studies underlying the numeric DDT concentration targets proposed in the Regional Board's draft TMDL staff report (SARWQCB, 2006)—targets that include sediment threshold effect levels (TELs), fish tissue targets for the protection of human health and wildlife, and water column targets (see Table 1)—indicates that in many important cases the Regional Board's proposed standards are erroneous and scientifically unjustified. This is the case for the DDT freshwater and saltwater sediment TELs (6.98 ppb and 3.89 ppb respectively), the marine fish tissue DDT level to protect wildlife (50 ppb) and the fish tissue DDT level to protect human health (100 ppb). Dr. James Byard points out that the proposed sediment TELs are too low by at least one order of magnitude, and perhaps by more than two orders of magnitude, given flaws in the data sets used to calculate the TELs. Dr. Byard concludes that a marine and fresh water fish tissue target of 150 ppb protects sensitive marine wildlife. The 150 ppb level of total DDT in fish tissue is also the basis for the National Toxics Rule (NTR) and California Toxics Rule (CTR) criteria for DDT in the water column. Although DDT bioassay data are not available for marine mammals, Dr. Tjeerdema concluded that toxic consequences at current levels are unlikely. Finally, Dr. Byard points out that the OEHHA fish tissue guidance for human ingestion was never 100 ppb; 100 ppb was used as a screening level to identify locations for further study. The OEHHA guidance was targeted to be less than 1,000 ppb (corresponding to a 10^{-4} lifetime cancer risk), and is currently being revised to 560 ppb. It is also important to note that the State Board 303(d) listing policy explicitly states that OEHHA values should be used as listing guidances only, not regulatory levels. The literature review presented here indicates

that the numeric concentration targets used for DDT in the organochlorine TMDL are based on flawed and outdated datasets, resulting in erroneous target values. Thus, application of the proposed TMDL targets is inappropriate and without scientific basis.

The downward trends in DDT concentrations observed in the Newport Bay watershed must also be considered. The Problem Statement contained in the organochlorine TMDL drafted by the Regional Board relies on data from multiple sources, but a significant proportion of these data is between five and ten years old, and the Regional Board has not regressed these data to the current time. According to the most recent data, DDT concentrations have been steadily declining in the Newport Bay watershed for at least 20 years. This decline is evident in data for several different media—particularly fish tissue and mussels, in which the decline is statistically very strong. The consulting firm Quantitative Environmental Analysis (QEA) analyzed available concentration data in the watershed and concluded that the probability that there is not a substantial declining trend in DDT concentrations in the watershed is "vanishingly small" (QEA, 2006, Appendix A). If no toxic effects due to DDT are observable under current concentrations in the watershed, declines in DDT concentrations indicate that toxic effects will not likely be observed in the future. Further, the Regional Board's use of older data without regressing these data to the present time overstates current DDT levels and leads to erroneous conclusions.

The observed declines in DDT concentrations in the watershed are likely due to several factors. One important factor is the degradation of organochlorines over time. For example, based on an estimated DDT half-life of 2-15 years, and the fact that DDT use was banned in 1972, we would expect that the mass of DDT in the agricultural soils of the Newport Bay watershed would have declined by at least 75% over the past 34 years, and perhaps by much more, due solely to natural break-down. The statistically strong observed declines in DDT concentrations over the past 20 years demonstrated in QEA's analysis confirm this expectation. A second factor is the ongoing conversion of land from agricultural to developed uses. Organochlorines tend to have low water solubility and to sorb strongly to soil particles. Therefore, the predominant pathway for organochlorines to move in the watershed is soil erosion and transport, not soil leaching. Therefore, insofar as development covers over former agricultural areas—the original areas of organochlorine application and currently their dominant source in the watershed—development immobilizes both sediment and organochlorines, thereby reducing exposure to the chemicals throughout the watershed. The link between development and reduced erosion is evident in recent sediment load data, which show that sediment loads in the years 2000-2005 are significantly lower than they were during the years 1983-1999 (WRC, 2006). The cause of this drop in sediment loads seems to be development of agricultural land and channel stabilization. Between 1983 and 2000 agricultural land use in the watershed dropped from 22 percent to 7 percent while developed area rose from 48 percent to approximately 60 percent.

INTRODUCTION

In 2002 the EPA established a Toxic Pollutants Total Maximum Daily Load (TMDL) that developed target loads for organochlorines (including DDT, chlordane, toxaphene, and dieldrin) for the Newport Bay Watershed. EPA's TMDL found that existing loads of these compounds exceed EPA's calculated allowable loads, which were based upon sediment quality guidelines rather than observed effects. The Santa Ana Regional Water Quality Control Board (Regional Board) is currently working to revise EPA's 2002 TMDL for organochlorine compounds and to develop an implementation plan. Regional Board staff and authors of separate studies have also asserted that these compounds – most notably DDT – have the potential to cause impacts, including chronic toxicity and eggshell thinning, at current concentrations. These and other important scientific issues will drive critical decisions regarding TMDL implementation.

Use of most organochlorine pesticides in the United States ceased long ago. DDT was banned by EPA in 1972, dieldrin in 1974, chlordane in 1988, and toxaphene in 1990. However, prior to their ban, these chemicals were commonly used as pesticides in agricultural production. For example, DDT was applied in large quantities in California prior to 1972; approximately 1.1 million pounds of the pesticide were applied in 1970 alone (Mischke et al., 1985). The half-life of DDT is between 2 and 15 years in soil (U.S. Department of Human Health & Human Services, 1994). At high concentrations DDT has been found to negatively affect the hatching success of several avian species, including the brown pelican, the osprey, and the peregrine falcon. Other organochlorines differ from DDT in that they did not affect hatching success below acutely toxic levels, were applied in smaller quantities, and generally have shorter half-lives.

Since their ban, concentrations of organochlorine compounds in sediments, fish, and shellfish from the Newport Bay watershed have declined dramatically, and the mass of these compounds in watershed soils also continues to decline. Recent studies demonstrate that these compounds are not likely to be causing acute toxicity in the watershed; these studies have found that other compounds are likely to be the cause of acute toxicity in the waters and sediments of San Diego Creek and Newport Bay (Lee and Taylor, 2001; Bay et al., 2004). Despite the widely published conclusion that DDT does not cause mutations or embryo deformities, this issue has been raised by Regional Board staff and an unpublished non-peer reviewed study report funded by the Regional Board (Sutula et al., 2005).

Three studies have been proposed to address these issues and to develop potential alternatives to the implementation measures being considered by the Regional Board:

(1) A review of available toxicology data in the literature, to address the Regional Board's primary concerns regarding DDT in the watershed;
(2) A comprehensive identification of the causes of acute toxicity within the sediments and water of Newport Bay and San Diego Creek; and
(3) Continued monitoring for DDT and other organochlorines.

This report details the results of the first study, conducted under contract with The Irvine Company by a consultant team consisting of Flow Science Inc., James L. Byard, Ph.D., John P. Connolly, Ph.D., and Ronald S. Tjeerdema, Ph.D.

TOXIC EFFECTS OF DDT IN THE NEWPORT BAY WATERSHED

Perhaps the most important consideration indicating that the Regional Board's efforts to establish a TMDL for DDT in the Newport Bay watershed are unjustified is the fact that DDT is not causing toxic effects in the watershed, as demonstrated below. If DDT is not responsible for toxic effects in the watershed and considering that DDT concentrations will continue to decline naturally in the future, then there is no reason to reduce current DDT loads. In other words, the watershed has the capacity to assimilate current DDT loads and there is no need to artificially reduce loads at this time.

Acute Toxicity

Several recent studies suggest that DDT is not likely a cause of acute toxicity in Newport Bay water and sediment. Bay et al. (2004) found evidence of acute toxicity in sediment from the Bay, but explicitly noted that variations in sediment toxicity were not correlated with concentrations of DDTs, PCBs, or PAHs. They concluded that sediment toxicity seemed to be attributable to unmeasured organic compounds, such as organophosphate, carbamate, or pyrethroid pesticides. Similarly, Lee et al. (2001) note that while toxicity related to urban storm water runoff is present in Newport Bay, recent work has shown that the cause of the toxicity is not heavy metals or organochlorine compounds but rather organophosphate pesticides, such as diazinon and chlorpyrifos. Lee and Taylor (2001) also suggest that pyrethroid pesticides should be investigated further as a potential source of toxicity in the Bay. Thus, based on the most recent studies of acute toxicity in Newport Bay, DDT or other organochlorine compounds are not the cause of observed acute toxicity in the Bay.

Chronic Toxicity

To address the concern that wildlife species in the Newport Bay watershed could be exposed to DDE—a metabolite of DDT—concentrations that might harm their reproductive success, an extensive scientific literature review was undertaken to evaluate the current state of knowledge about DDE concentrations in the tissue of key wildlife species, trends in such DDE concentrations, and links between DDE tissue concentrations and reproductive success. The results of this literature review indicate that relevant wildlife populations are not currently exposed to levels of DDE in Newport Bay that are known to cause chronic toxicity, and that the expected continuing declines in DDE concentrations in the environment (due to the ban on its use in 1972) make it highly unlikely that DDE concentrations in wildlife tissue will increase from these nontoxic levels in the future.

Brown Pelican

During the era prior to the ban of DDT in 1972, levels of DDT and its metabolite DDE became elevated in sediment and in biota such as the northern anchovy on the Palos Verdes shelf (Risebrough et al., 1967). These elevated levels of DDE caused eggshell thinning and substantially reduced hatching success in brown pelican breeding on Anacapa Island, whose diet consists primarily of northern anchovy (Keith, 1969). For example, Dr. Byard writes that "in 1969 their [the brown pelicans'] reproductive effort was for all practical purposes a complete failure" (Appendix B, p. 3).

The brown pelican is a species particularly sensitive to elevated levels of DDE. DDE residue levels greater than 2.5 ppm in brown pelican eggs have been associated with eggshell thinning in excess of 15 percent and decreased hatching success. Conversely, residue levels lower than 2.5 ppm have not been associated with decreased brown pelican hatching success, though they are correlated with measurable eggshell thinning (e.g., Blus, 1984). As Dr. Byard points out, fish concentrations of 150 ppb DDE should correspond with egg residue concentrations of 1.7 ppm DDE for brown pelicans, which is below the threshold for reduced hatching success (Appendix B, p.1). Based on this conclusion, 150 ppb DDE in fish tissue amounts to a fish tissue level that would not reduce brown pelican hatching success. Indeed, after a thorough review of the relevant scientific literature, 150 ppb is the fish tissue concentration that Dr. Byard found to be protective of another sensitive species, the osprey (Appendix C). This fish tissue concentration is three times higher than the 50 ppb criterion proposed in the Regional Board's recent TMDL staff report (SARWQCB, 2006, Table 3-1). Therefore, the Regional Board's proposed numeric target for fish tissue is unjustified, especially considering that brown pelicans do not breed in the Newport Bay watershed.

Currently, DDE egg residue levels for all populations of brown pelicans in the U.S.—even those on Anacapa Island—are below the 2.5 ppm threshold for reduced hatching success, and hence brown pelican reproduction has returned to normal (e.g., Gress, 1995). Insofar as the Anacapa Island population represents a worst-case scenario for this species, populations in other parts of the U.S., including Newport Bay, are not currently at risk from DDT contamination and should not be in the future, given the expected continued decline in DDT residues.

Osprey

Similarly, Dr. Byard finds that the osprey—another avian species sensitive to the reproductive effects of DDT and subject to population declines during the DDT era—has recovered in all regions of the U.S. as DDT residues have declined in fish and eggs (Appendix C). However, he notes that despite significant declines in DDT sources in the U.S., the southern breeding grounds of the osprey in Latin America—where DDT continues to be used—remain an important source of DDT in osprey populations and have limited the decline of egg residues. An osprey pair successfully fledged three chicks from a nest in Newport Bay during the 2006 breeding season.

Cormorants and Terns

Dr. Byard also reported on the effects of DDT on cormorants and terns (Appendix D). Although these species are not the species most sensitive to DDT—the brown pelican, the osprey, and the peregrine falcon are among the most sensitive species—cormorants and terns breed in the Newport Bay watershed and are the selected receptors in a sediment-to-wildlife modeling study about to be reported by the San Francisco Estuary Institute (SFEI) (B. Greenfield, pers. comm., 2006). Therefore, the effects of DDT on cormorants and terns are important for the purposes of assessing potential impacts of DDE in the Newport Bay watershed. Dr. Byard's central conclusion is that DDE residues exceeding 10 ppm in eggs are required before significant DDT-related hatching failure will be observed in cormorant and tern populations. This DDE level results in eggshell thinning of approximately 15% or greater. Insofar as 10 ppm is significantly higher than the 2.5 ppm threshold for the brown pelican, and insofar as that 2.5 ppm threshold corresponds with a fish tissue concentration in excess of 150 ppb, the actual fish tissue threshold for cormorants and terns is likely to be in excess of 600 ppb, much higher than the 50 ppb residue level proposed by the Regional Board (SARWQCB, 2006, Table 3-1).

Marine Mammals

Dr. Ronald Tjeerdema's recent survey of the scientific literature relevant to DDT in marine mammals also supports the conclusion that wildlife species are not, and will not be, subject to harmful levels of DDT and its metabolites (Appendix E). The species Dr. Tjeerdema focused on are as follows: the California sea lion, the harbor seal, the Pacific bottlenose dolphin, the rough-toothed dolphin, the common dolphin, and two filter-feeding baleen whales—the minke whale and the migratory gray whale. These are the marine mammalian species that could have even a remote chance of spending a small amount of time in or near Newport Bay. Of these, only the California sea lion and the harbor seal have the potential to reside in the Bay for significant time periods.

From his survey of the relevant literature, Dr. Tjeerdema drew several important conclusions. First, he found that measurable concentrations of DDT and its metabolites have been reported in the tissue of these species since the 1960s. Blubber concentrations in fish-eating harbor seals, California sea lions, and Pacific bottlenose and common dolphins were found typically to be in the parts per million (ppm) range. Blubber concentrations in the two baleen whale species were found generally to be lower (in the parts per billion (ppb) range) since they feed at lower levels of the food chain. These observations suggest that the relevant species are indeed capable of accumulating DDT and its metabolites in their tissue. However, his second finding was that DDT tissue concentrations have been declining in these species since the 1970s. Given the expected general declines in environmental concentrations due to the ban on DDT, Dr. Tjeerdema concludes that mammalian tissue concentrations will continue the reported decline since the 1970s.

Dr. Tjeerdema also found that no studies to date have been able to demonstrate in a statistically significant population a link between DDT tissue concentrations and toxic

effects on marine mammals. He attributed this lack to the protected status of—and corresponding restricted access to—the relevant marine mammal species, and to the difficulty of conducting controlled experiments with significant sample sizes with such species given their relatively large size. Given that these limiting factors will likely persist in the future, Dr. Tjeerdema sees little prospect of such systematic toxicological studies being conducted in the future. Nevertheless, given that DDT concentrations are on the decline in the Newport Bay watershed, given that these marine mammals have only a transitory presence in the Bay, and given that DDT accumulation tends to occur in the relatively metabolically inactive blubber tissue of the species (a nontarget tissue), Dr. Tjeerdema found it "unlikely that sufficient concentrations will be accumulated in the region to cause toxic consequences" (Appendix E, p. 11).

PROPOSED DDT STANDARDS

In their forthcoming TMDL, the Santa Ana Regional Board proposes to apply several standards for DDT and its metabolites (i.e., total DDT) to DDT levels in different media. For example, the Board intends to apply a chronic criterion of 1 pptr (or 0.001 ug/L) to both fresh and salt water, freshwater and saltwater sediment threshold effect levels (TELs) of 6.98 ppb and 3.89 ppb respectively, and National Academy of Science (NAS) fish tissue standards of 1000 ppb and 50 ppb in freshwater and marine fish respectively. Table 3-1 from the Regional Board's TMDL staff report (SARWQCB, 2006) summarizes the proposed numeric targets for organochlorines and is reproduced below in Table 1.

Table 1: Numeric Sediment, Fish Tissue, and Water Column TMDL Targets, Newport Bay Watershed Organochlorine TMDL.

Sediment Targets[1]; units are ug/kg dry weight				
Location	**Total DDT**	**Chlordane**	**Total PCBs**	**Toxaphene**
San Diego Creek and tributaries	6.98	4.5	4.1	0.1
Upper & Lower Newport Bay	3.89	2.26	21.5	
Fish Tissue Targets for Protection of Human Health[2]; units are ug/kg wet weight				
San Diego Creek and tributaries	100	30	20	30
Upper & Lower Newport Bay	100	30	20	
Fish Tissue Targets for Protection of Aquatic Life and Wildlife[3]; units are ug/kg wet weight				
San Diego Creek and tributaries	1000	100	500	100
Upper & Lower Newport Bay	50	50	500	
Water Column Targets for Protection of Aquatic Life, Wildlife & Human Health[4]; (ug/L)				
San Diego Creek and tributaries				
Acute Criterion (CMC)	1.1	2.4		0.73
Chronic Criterion (CCC)	0.001	0.0043	0.014	0.0002
Human Health Criterion	0.00059	0.00059	0.00017	0.00075
Upper & Lower Newport Bay				
Acute Criterion (CMC)	0.13	0.09		
Chronic Criterion (CCC)	0.001	0.004	0.03	
Human Health Criterion	0.0059	0.00059	0.00017	

[1] Freshwater and marine sediment targets are TELs from Buchman, M.F. 1999. NOAA Screening Quick Reference Tables, NOAA HAZMAT Report 99-1, Seattle WA, Coastal Protection and Restoration Division, National Oceanic and Atmospheric Administration, 12 pp.
[2] Freshwater and marine fish tissue targets for protection of human health are OEHHA SVs.
[3] Freshwater and marine fish tissue targets for protection of aquatic life and wildlife are from Water Quality Criteria 1972. A report of the Committee on Water Quality Criteria, Environmental Studies Board, National Academy of Sciences, National Academy of Engineering. Washington, D.C., 1972.
[4] Freshwater and marine targets are from California Toxics Rule (2000).
Source: SARWQCB, 2006, Table 3-1.

Moreover, the Board regards the Environment Canada fish tissue residue guideline (TRG) of 14 ppb—aimed at protecting fish-eating avian species—as potentially relevant to their regulatory activity.

The target concentrations proposed by the Regional Board would appear to require a significant reduction of DDT loads in the watershed (SARWQCB, 2006). As the previous section on toxic effects indicates, current loads are below levels that would cause toxic effects, both acute and chronic, indicating that additional load reductions are unnecessary. In addition, a recent literature review indicates that in several cases the concentration targets proposed for use by the Regional Board are flawed. In other words, not only are these targets not applicable to the Newport Bay watershed at this time (due to the lack of observed DDT-related toxic effects in the watershed), but the targets themselves are scientifically incorrect, and their application would be inappropriate in any context. This conclusion will be demonstrated in the following section.

CTR Water Criterion

As noted, the CTR criterion for DDT in water is 1 pptr or 0.001 ug/L. This criterion is based primarily on a study by Anderson et al. (1975) of the effects of DDT and its metabolites (particularly DDE) on the reproduction of brown pelicans on Anacapa Island (see Appendix B). As noted previously, the levels of DDT and its metabolites in water, sediment, and biota such as the northern anchovy, were elevated around Anacapa Island, causing eggshell thinning and substantially reduced hatching success in brown pelicans, whose diet consists primarily of northern anchovy. The 1975 study by Anderson et al. found both that anchovy concentrations were approximately 150 ppb, and that brown pelican reproduction was still inhibited despite almost complete recovery. The EPA used the 150 ppb concentration in northern anchovy—along with several other factors, including a bioconcentration factor (BCF)—to derive the CTR water criterion of 1 pptr.

However, as Dr. Byard points out (see Appendix B), the study by Anderson et al. also found that the 150 ppb value in anchovy populations represented a 27-fold decline in tissue concentrations since the pre-1972 DDT era, while brown pelican egg residue DDE concentrations had declined only 9-fold over the same period. The difference between the magnitudes of the two declines indicates that egg residue DDE concentrations were not at a steady-state equilibrium concentration at the time of the Anderson et al. study. If egg residue concentrations ultimately would decline at least 27-fold as anchovy concentrations did, then the ultimate geometric mean egg residue DDE concentration would be 1.7 ppm. Insofar as the threshold DDE residue level for hatching success is approximately 2.5 ppm, the 150 ppb anchovy concentration and corresponding 1.7 ppm egg residue concentration are below the threshold for hatching failure in brown pelicans. Thus, the 1 pptr CTR criterion (which is based on the 150 ppb anchovy concentration) likely represents a level below the threshold for brown pelican hatching failure, and therefore represents a NOEL (No Observed Effect Level) for the effects of DDT on wildlife and is below the level of DDT necessary to protect this beneficial use.

Sediment TELs

A review of the basis for the currently applicable freshwater and saltwater total DDT sediment TELs—6.98 ppb and 3.89 ppb respectively—indicates that the TELs are flawed, resulting in values that are not appropriate for use in this TMDL (Appendix F). The first problem with the TELs is that they are based on weak and/or erroneous data. For example, some data points underlying the TELs were erroneously interpreted, selected arbitrarily, or "double-counted." Moreover, some sediment concentrations underlying the TELs—i.e., those derived from water concentrations and sediment-water partition coefficients (e.g., K_{ow}'s, K_{oc}'s)—were based on outdated and incorrect partition coefficients. Also, in some cases low DDT residue data points were used when higher level residue data points were shown to have no effect. As Dr. Byard points out, "If these flaws were corrected, the TEL values would be considerably higher" (Appendix F, p. 1).

The second problem with the TELs is that they are based primarily on the co-

occurrence of toxicity and DDT in sediments, not on dose-response data. In many cases there were numerous other toxic substances present in the sediments used to derive the TELs, which could account for the observed toxicity. In some cases authors of the underlying scientific studies ascribed toxicity to compounds other than DDT and specifically exonerated DDT (Bay et al., 2004). Many of these other compounds are also in Newport Bay sediments and these results further highlight the need to identify and address the true causes of toxicity. Moreover, instances of dose-response data—such as spiked sediment bioassays and studies of benthic communities highly contaminated with DDT—were weighted too lightly in the derivation of the TELs. Such dose-response data indicate that "the toxicity threshold for total DDT to benthic organisms is more than two orders of magnitude higher than the TELs proposed for use in Newport Bay and San Diego Creek" (Appendix F, p. 1). Therefore, the TELs proposed by the Regional Board are flawed and should not be applied in the Newport Bay watershed (or anywhere else). This conclusion is in agreement with the State Board, which does not recommend the use of TELs in their listing policy.

Department of Interior Guidance on DDT Residue in Avian Eggs

Dr. Byard found that a key report by the Department of the Interior (DOI) (1998) on toxicity thresholds for DDT in avian species contained several errors and serious misrepresentations of published scientific studies (Appendix D, pp. 1, 7-8). For example, the DOI report lists 1 ppm DDE in western grebe eggs as causing 1 % shell thinning. However, the DOI report cites Boellstorff et al. (1985), who reported a concentration of 1.4 ppm—not 1 ppm—and who state clearly that they do not regard the reported eggshell thinning to be statistically significant. As another example, the DOI report cites Lindvall and Low (1980) as reporting that an egg residue of 5.4 ppm DDE caused 2.3 % eggshell thinning and reduced hatching success in the western grebe. However, Lindvall and Low actually reported a DDE residue of 6.6 ppm and a thinning of 3.1 %. Furthermore, Lindvall and Low explicitly concluded that the reported thinning levels had little to no effect on reproductive success. In addition to these problems, Dr. Byard points out at least four more serious problems with the DOI's use of relevant scientific literature in their report. Dr. Byard draws the following conclusion about the DOI report: "At best the report is done incompetently and at worst is an intentional misrepresentation to achieve a higher potency for DDT in avian species than is supported by scientific study." This conclusion is significant for the Newport Bay watershed since a key report on which the Regional Board depends in establishing regulatory limits for DDT (and which the Board funded)—Sutula et al. (2005)—relies on the DOI findings.

NAS Guidance on DDT Residue in Fish Tissue

As noted previously, the Regional Board plans to apply in its forthcoming TMDL the 1972 recommendations of the NAS on DDT limits in freshwater and marine fish tissue. The recommendations are 1000 ppb and 50 ppb in freshwater and marine fish respectively, and were produced by two separate NAS panels 34 years ago.

However, as a recent study by Dr. James Byard demonstrates, these

recommendations are flawed in several significant ways (Appendix C). First, the 20-fold difference between the two values is unjustified since both values were based on essentially the same data. Dr. Byard reasons that since both criteria are based on eggshell thinning and reproductive failure in similarly sensitive avian species, the criteria should be similar. Second, Dr. Byard found that the 1972 panels overlooked important information available to them at the time of their recommendations. Third, the panel's recommendations do not incorporate results from the abundant study of this topic which has been conducted in the over 30-year period since 1972.

Based on a survey of the most up-to-date relevant information pertaining to the reproductive effects of DDT in fish on sensitive avian species, Dr. Byard concludes that the guidance for DDT in fish tissue ought to be 150 ppb for marine species. He notes that a 150 ppb guidance value would be consistent with the current CTR criterion for DDT in water, which is based on a 150 ppb DDT concentration in fish, a concentration which he estimated to be below the threshold for reproductive toxicity in the DDT-sensitive brown pelican. Insofar as the Regional Board staff's proposed 50 ppb target for marine fish tissue is one-third of this 150 ppb value, the board's value is unjustified. Conversely, the freshwater value of 1000 ppb proposed by Regional Board staff seems too high and likely is not adequately protective of sensitive avian species.

Canadian Fish Tissue Residue Guideline

In 2000, Environment Canada published a fish tissue total DDT residue guideline (TRG) aimed at protecting fish-eating avian species from the reproductive effects of DDE, a DDT metabolite (Environment Canada, 2000). The published TRG was 14 ppb. However, Dr. Byard concludes that the TRG was based on several questionable assumptions that led to an erroneous value that is too low (see Appendix G). First, Environment Canada selected two species of duck—the mallard and the black duck—as the test species for formulating the TRG. However, neither species of duck normally eats fish; both are primarily herbivores. Thus, it does not make sense to use these duck species as models for the effect of DDE in fish on avian species. Rather, Environment Canada should have used at least a carnivorous species such as the American kestrel (sparrow hawk), which is sensitive to the reproductive effects of DDE, and for which excellent dose-response data regarding eggshell thinning, DDE residue in eggs, and hatching failure are readily available. Although present in California, other more sensitive species such as the Brown Pelican were not considered in the development of the Environment Canada TRG since populations of such species are generally not significant in Canada.

Second, Environment Canada chose to use eggshell thinning, not hatching failure, as the toxic endpoint upon which to evaluate the reproductive effects of DDE on avian species. However, it is widely recognized that eggshell thinning below the threshold for hatching failure is not detrimental to avian wildlife, and thus is not known to be a toxic endpoint (Appendix G). Instead of eggshell thinning, Environment Canada should have used hatching failure, the most sensitive toxic endpoint for chronic DDE exposure in birds.

Third and finally, Environment Canada chose to assume in their TRG calculations the daily food intake rate of the Wilson's storm petrel. This choice was inappropriate since fish make up only a minor part of the Wilson's storm petrel diet, and since petrels have been shown to be less sensitive to the reproductive effects of DDE than species such as the osprey, the brown pelican, and the peregrine falcon. Instead of the food intake rate of the Wilson's storm petrel, Environment Canada should have used the intake rate of the osprey, a fish-eating species that both is sensitive to DDE and has a relatively high daily food intake rate. As Dr. Byard has noted, if appropriate assumptions described here had been used, Environment Canada would have calculated a TRG of 250 ppb, a value 18 times higher than the value published in 2000 (Appendix G, p. 7). Thus, the Environment Canada value is excessively protective.

OEHHA Sport Fish Guidance for Human Ingestion

In 1991, the California Office of Environmental Health Hazard Assessment (OEHHA) published a guidance report on sport fish consumption in Southern California (Pollock et al., 1991). The guidance has been updated various times for other areas of the State. Dr. Byard points out that the Santa Ana Regional Board staff have misinterpreted the OEHHA fish guidance for total DDT to claim impairment of sport fishing in Newport Bay (Appendix H). The OEHHA guidance cautions against using the recommended 100 ppb OEHHA target as a standard. The objective of the OEHHA guidance was to achieve a potential cancer risk of less than 1/10,000 (less than 1,000 ppb) at each site. This objective is met in Newport Bay. The guidance states that the linear dose extrapolation procedure used to estimate cancer risk likely overestimates the actual risk. Studies confirm that DDTs (DDT, DDE and DDD) are not genotoxic and produce cancer in rodent livers by a threshold-promoting activity. This understanding was part of the original FDA action level of 5,000 ppb in commercial fish. Dr. Byard also points out that OEHHA recently has issued new draft guidance that sets the fish fillet screening level at 560 ppb total DDT. The new guidance uses the 1/10,000 cancer risk level and considers the decay of DDTs in the environment. This new guidance is also met in Newport Bay. Dr. Byard concludes that DDTs are not impairing sport fishing in Newport Bay.

DDT CONCENTRATIONS

The proposed establishment of a TMDL for DDT by the Regional Board is inappropriate for another reason—the statistically strong downward trends in organochlorine concentrations in the Newport Bay watershed. Rather than incorporating these trends into their analysis, the Regional Board is relying upon data that are in many cases five to ten years old, and has failed to project well-established trend data to the present time (SARWQCB, 2006). Insofar as toxic effects caused by DDT are not observed in the watershed under current loadings, toxic effects due to DDT are highly unlikely in the future given the observed and projected decline of DDT concentrations over time. According to the most recent data, organochlorine concentrations have been steadily declining in the Newport Bay watershed for at least 20 years. This decline is evident in data for several different

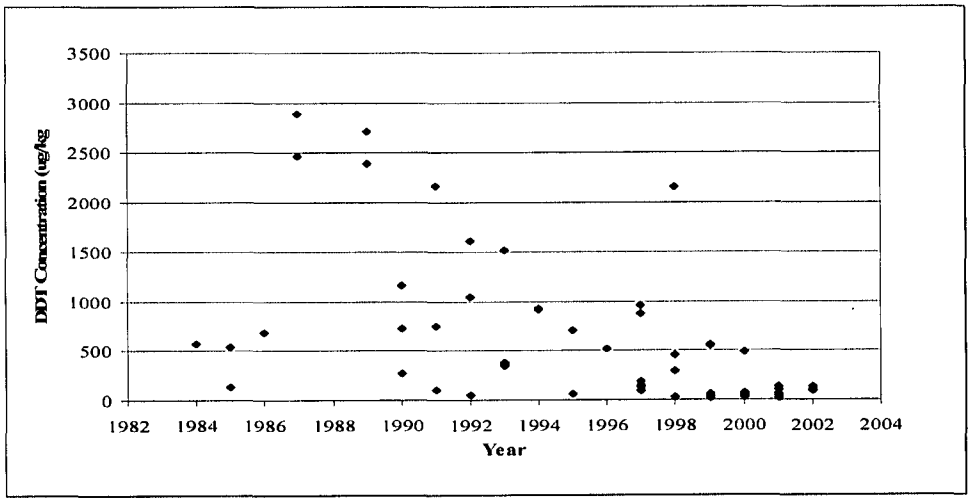
media—particularly fish tissue and mussels, in which the decline is statistically very strong—and will be shown in the following sections.

Quantitative Environmental Analysis (QEA) conducted a rigorous statistical analysis of Newport Bay DDT concentration data for three different media: fish tissue, mussel tissue, and Bay sediment. QEA's conclusion was that these three lines of evidence indicate strongly that DDT concentrations are declining in the Newport Bay Watershed: "There [are] statistically significant declines in DDT in red shiners, and in mussels in SDC [San Diego Creek], UNB [Upper Newport Bay], and LNB [Lower Newport Bay]. Additionally there are declining trends in seven other fish species although there are not enough data for robust statistical analysis in these species. *The likelihood of having so many independent data sets show a declining trend if a downward trend did not exist is vanishingly small.* For example, if there were no trend at all, there is a 50% chance of randomly getting a positive or negative trend from any given data set, and the probability of getting 11 negative trends is 0.0005 (i.e., 0.5^{11})" (Appendix A, QEA, p. 8, emphasis added). QEA's technical memorandum (Appendix A) contains complete results of their analysis.

Fish Tissue

Trends in DDT concentrations are evident in data collected for approximately 20 years in the Newport Bay watershed. In the case of the fish species red shiner, data showing substantial decline in tissue DDT concentrations are available from 1983 through 2002 (Figure 1). Red shiner may be taken as an indicator species for DDT in the watershed since there are sufficient data to clearly establish concentration trends for that species and since this species is short-lived (approximately 2 years; Baird and Girard, 1853) and residents do not range outside of the Newport Bay watershed.

Figure 1: Red Shiner DDT Concentration Data, Newport Bay Watershed



Exponential regression (ln[red shiner DDT concentration] vs. year) was used to evaluate the strength of the declining trend in DDT concentration in red shiner tissue over

time. For the regression incorporating data from all available years, QEA reported a "highly significant exponential decline" in DDT concentrations in red shiner tissue, and calculated the rate of decline (without outliers) to be -0.183 per year (Appendix A, p. 3). This rate of decline amounts to a DDT half-life in the watershed, as calculated from the surrogate endpoint of red shiner tissue, of 3.8 years, which is significantly lower than the 20-year half-life assumed for soil (see section "Natural DDT Removal"). QEA also performed a regression analysis for two 10-year sub-periods within the data set, 1983-1992 and 1993-2002. The purpose of this split analysis was to evaluate whether rates of DDT loss in this species have changed over time. This analysis showed that the rate of DDT concentration decline in DDT fish tissue was lower for the later period (-0.135 per year) than for the earlier period (-0.245 per year), but that more recent decline rates are still "highly significant" (Appendix A, p. 4).

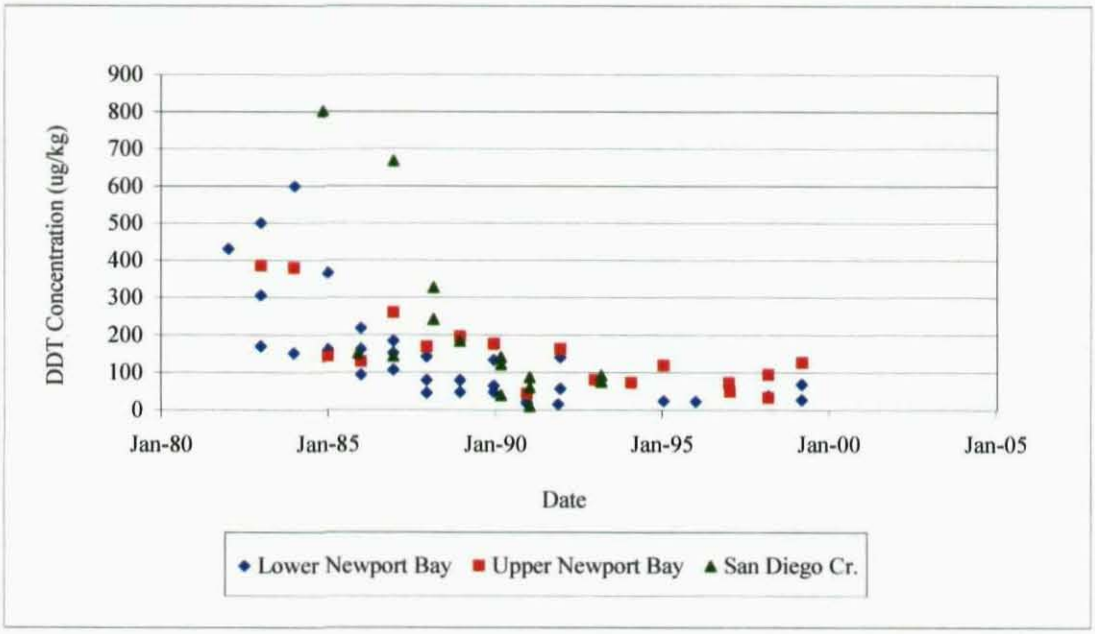
Fish species other than red shiner also show similar declines in DDT concentrations, despite far fewer data for each species. QEA evaluated seven other fish species (black perch, California halibut, California killifish, diamond turbot, spotted sand bass, striped mullet, and yellow fin croaker) for which three or more DDT concentration data points were available during a time range of five or more years and found that each species exhibited a declining trend over time. These negative trends are consistent with the red shiner data analysis, which showed that DDT levels in the biota of Newport Bay are declining, although several factors suggest that each of the trends observed in the seven fish species would not, in isolation, support strong inferences. This is largely because the datasets for these seven additional fish species contain too few data points to infer long-term trends from data from any one of those species taken alone. Nevertheless, when taken together, data from these seven species are consistent with the strong trend evident for red shiner, lending far more weight to the conclusion that fish tissue DDT concentrations are declining in the watershed than could be concluded from data from any single species considered alone. Detailed discussion is provided in Appendix A.

Mussels

Mussel tissue data from three locations in the Newport Bay system—San Diego Creek, Upper Newport Bay, and Lower Newport Bay—were evaluated for trends in DDT concentrations over time. The central conclusion from this analysis is that like red shiner data mussel tissue data show statistically significant declines in DDT concentrations dating to 1982 (Figure 2). QEA performed an exponential regression analysis of mussel data including the entire period of record (1982-1999). This analysis showed a significant DDT concentration decline rate in mussels both when all three locations were considered together (-0.133 per year), and when the three locations were considered separately (San Diego Creek = -0.292 per year; Upper Newport Bay = -0.095 per year; Lower Newport Bay = -0.156 per year). The decay rate for all three locations considered together (-0.133 per year) can be used to estimate a half-life for DDT in the watershed of 5.2 years, which is significantly lower than the 20-year half-life assumed for soil (see section "Natural DDT Removal"). A split analysis was also performed on mussel data for the two nine-year periods 1982 to 1990 and 1991 to 1999. The rate of decline of DDT concentrations in mussel tissue was

statistically significant only for the earlier period and not for the later. As with the red shiner data, the rate of decline was lower for the later period than for the earlier. The later period regressions have low statistical power (i.e., the probability that the declining trend is not erroneous is low), partly due to small sample size. Nevertheless, the most important conclusion is that when the entire mussel data sets (1982-1999) for each of the three locations are considered, each set reflects statistically significant declines in DDT tissue concentrations.

Figure 2: Mussel DDT Concentration Data, Newport Bay Watershed

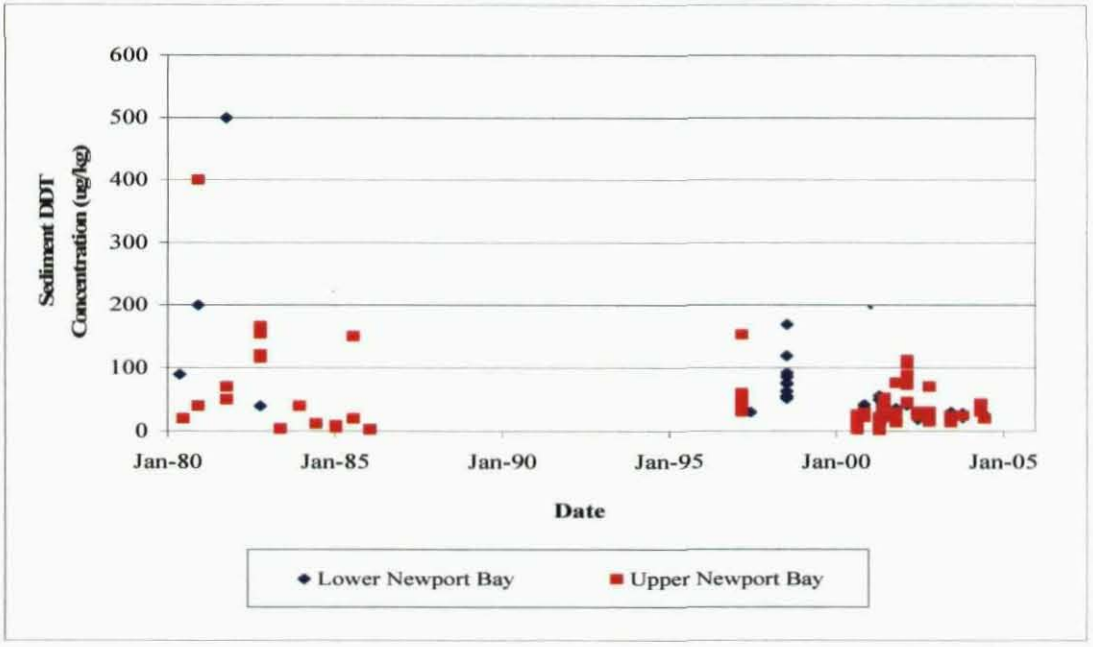


Bay Sediment

Sediment data are available for Lower and Upper Newport Bay for the period 1980 through 2004, with a long gap from 1987 to 1995 (Figure 3) (OC PFRD, 1980-86; SCCWRP, 1998; Bay et al., 2004; The Irvine Company, 2000-2004; U.S. EPA, 2002; Masters and Inman, 2000). However, it is difficult to infer Bay-wide trends in sediment DDT concentration over time from these data for several reasons. First, sampling was conducted by multiple agencies, using multiple methodologies, at varying locations and sample depths. Given this diversity in sampling approach and location, direct comparisons between data from year to year are inappropriate. Second, there is significant movement of sediment into, out of, and within the Bay such that even samples taken in the same location at two different times may not represent the change in DDT concentration for a specific quantity of sediment. Sediment movement results both from the natural flow of water and sediment in the Bay, as well as from periodic major dredging projects, which have occurred in the years 1983, 1985, 1988, and 1999. Third, sediment concentrations in Newport Bay may be more indicative of DDT loads from years or decades past, since Bay sediments are

transported from the upper watershed in a highly variable, episodic manner. Thus, DDT concentrations in Bay sediments reflect DDT that was applied many years ago in the upper watershed, and then sorbed to sediments in that location, which were subsequently eroded into a creek channel and transported to the Bay. Finally, Bay sediment DDT concentrations do not necessarily indicate bioavailability. This is especially true of samples collected from deeper sediment cores. While sample depths were not available for all data plotted in Figure 3, data from 1980 through 1986 (sampled by Orange County Public Facilities and Resources Department [OCPFRD], currently called the Resources and Development Management Department [RDMD]) reflect sample depths between two and 25 feet, with an average of 11 feet, well below the biologically active layer which extends only to a depth of approximately 6 inches. Thus, these early sediment samples are not indicative of concentrations available to biota in the Bay. For all these reasons, the available sediment data for Newport Bay are not reliable indicators of bioavailable DDT concentration trends in the watershed and should not be used independent of all other available data. As noted in Appendix A, QEA's analysis confirms this conclusion.

Figure 3: Bay Sediment DDT Concentration Data, Newport Bay



Water

Only minimal DDT water concentration data were available for the Newport Bay watershed. Table 2 summarizes these data. It is generally very difficult to measure the low levels at which DDT is present in the Bay. As the data show, only 3 of 12 data points were above detection limits. Also, the data are very temporally limited as they are based on samples from 2001 and 2002 only. For these reasons, no meaningful trend analysis could be

performed on the DDT water concentration data. The CTR human health regulatory threshold for DDT in water is 0.00059 ug/L, or 0.59 ng/L.

Table 2: DDT concentrations in water, Newport Bay

Date	Location	Sample Station	Kind of sample	Total DDT (ng/L)
4/23/2001	Lower Bay	Turning Basin	Water	1.29
4/23/2001	Lower Bay	PCH Bridge	Water	1.04
3/12/2002	Rhine Channel	NB3	Water	ND*
3/13/2002	Upper Bay	NB10	Water	ND*
3/7/2002	San Diego Creek	Campus Drive	Water (dry weather)	ND*
3/7/2002	San Diego Creek	Campus Drive	Water (stormflow)	ND*
5/2/2002	San Diego Creek	Campus Drive	Water (dry weather)	ND*
5/2/2002	San Diego Creek	Campus Drive	Water (dry weather)	ND*
8/12/2002	San Diego Creek	Campus Drive	Water (dry weather)	ND*
8/12/2002	San Diego Creek	Campus Drive	Water (dry weather)	ND*
11/8/2002	San Diego Creek	Campus Drive	Water (stormflow)	3
11/8/2002	San Diego Creek	Campus Drive	Water (stormflow)	ND*

* Detection limit = 1.0 ng/L Sources: Bay and Greenstein, 2003; Bay et al., 2004.

Agricultural Soils

Table 3 presents historical DDT concentrations at different depths for agricultural soils in the Newport Bay watershed. In general, agricultural soils in the watershed seem to exhibit a downward trend in DDT concentrations over time, which is expected given a DDT half-life of less than 20 years (Lichtenstein and Schultz, 1959; Racke et al., 1997; Stewart and Chisholm, 1971) and the fact that DDT use was discontinued in the early 1970s. However, it is crucial to note that the data reported in Table 3 were not sampled from the same locations. Rather, from year to year, soil concentrations were sampled in completely different locations. Given that no data were available showing the amounts of DDT historically applied to different areas of the watershed, the agricultural soils DDT data cannot be used to assess trends over time or local DDT half-life. Sampling locations and interpolated zero- to six-inch DDT contours for several sampling years are presented in Figures 4 through 8.

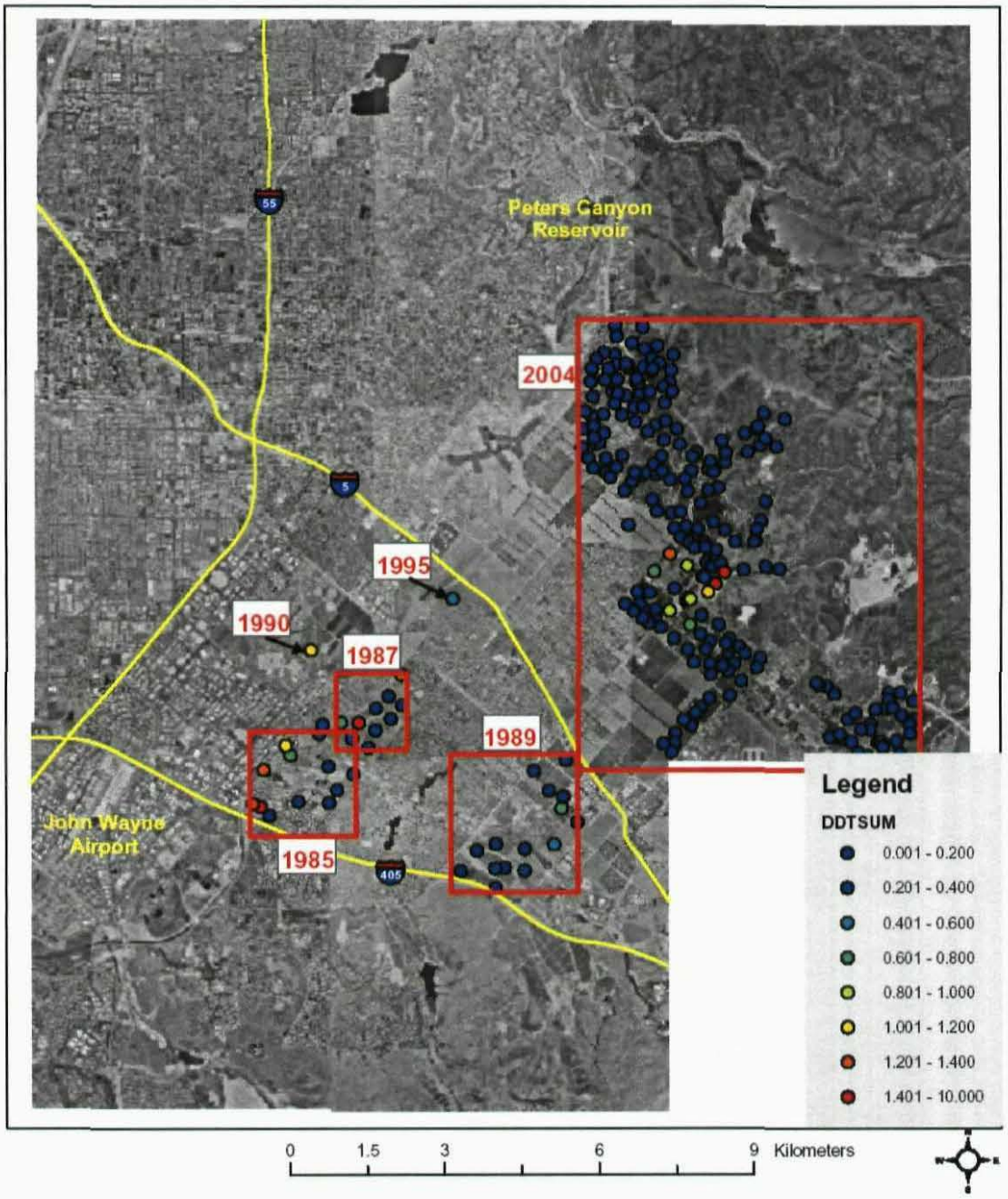
Table 3: Historical DDT concentrations, agricultural soils, Newport Bay watershed

Year	0-6 inch Sample Depth			12-18 inch Sample Depth			>24 inch Sample Depth		
	Average Observed Total DDT (ppm)	Max Observed Total DDT (ppm)	Sample Size (n)	Average Observed Total DDT (ppm)	Max Observed Total DDT (ppm)	Sample Size (n)	Average Observed Total DDT (ppm)	Max Observed Total DDT (ppm)	Sample Size (n)
1985	0.57	1.75	12						
1987	0.43	1.50	10	0.56	2.14	10	ND	ND	10
1988	0.29	1.09	10	0.12	0.15	10	0.55	0.55	10
1989	0.25	0.79	15	0.27	0.71	10	0.13	0.33	19
1990	0.40	0.90	4	0.51	0.91	2	0.09	0.20	7
1991	0.35	0.49	17	0.14	0.49	16			
1995	0.39	0.81	19						
2004	0.22	2.00	230				0.093	0.300	45

Note: No data were available for shaded areas.

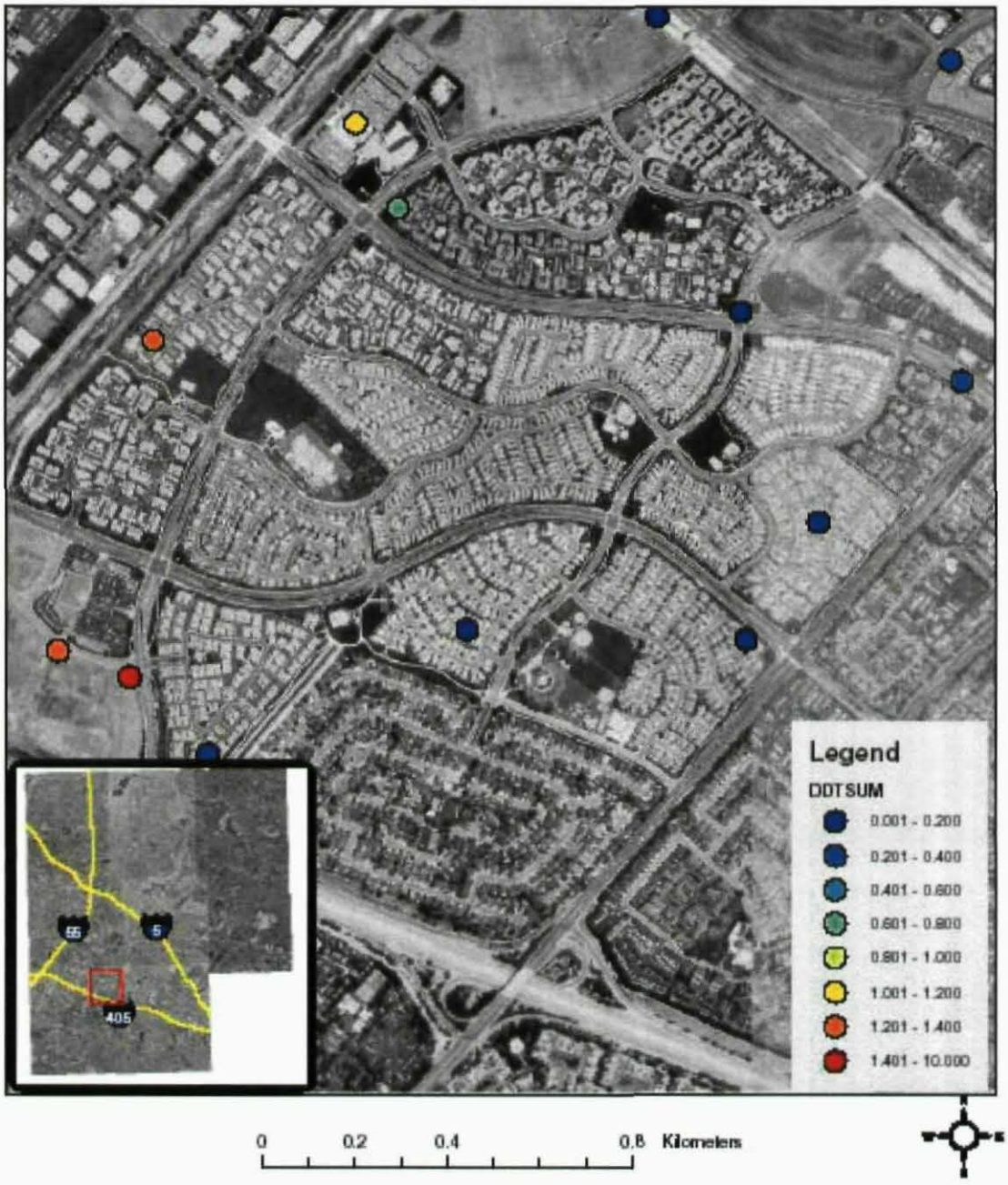
Sources: SA RWQCB, 1985; Leighton & Associates, 1985; Byard, 1985; Byard, 1987; Byard, 1988; Byard, 1989; Byard, 1990; Del Mar Analytical, 1990; Mittelhauser, 1991; NMG Geotechnical, 1996; The Irvine Company, 2006.

Figure 4: Agricultural Soils DDT Concentration Sample Locations



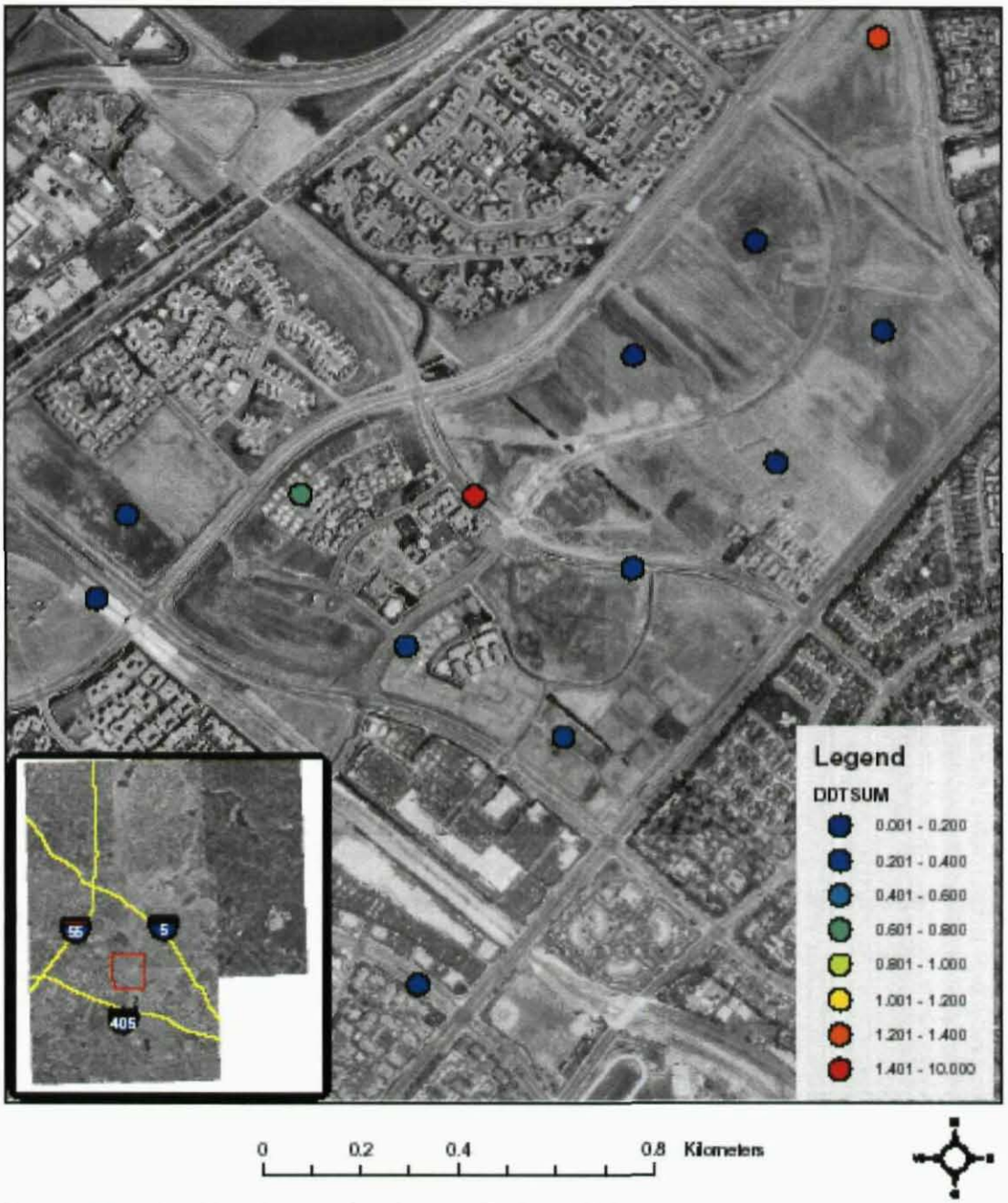
Source: Byard, 1985, 1987, 1989; The Irvine Company, 2006.
Composite photo underlay: 1994, 1995.

Figure 5: DDT Concentrations in Agricultural Soils, 1985



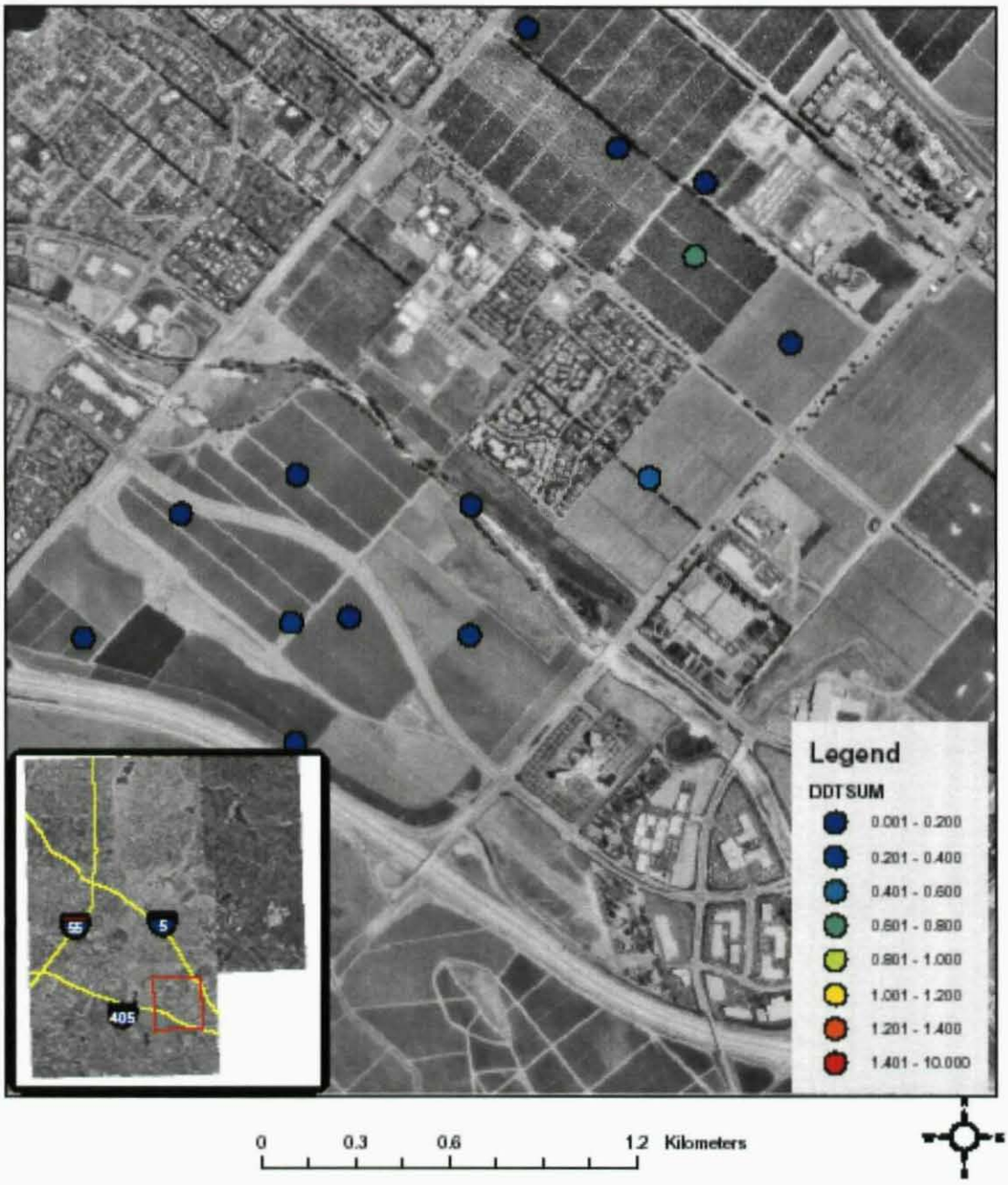
Source: Byard, 1985. Composite photo underlay: 1994.

Figure 6: DDT Concentrations in Agricultural Soils, 1987



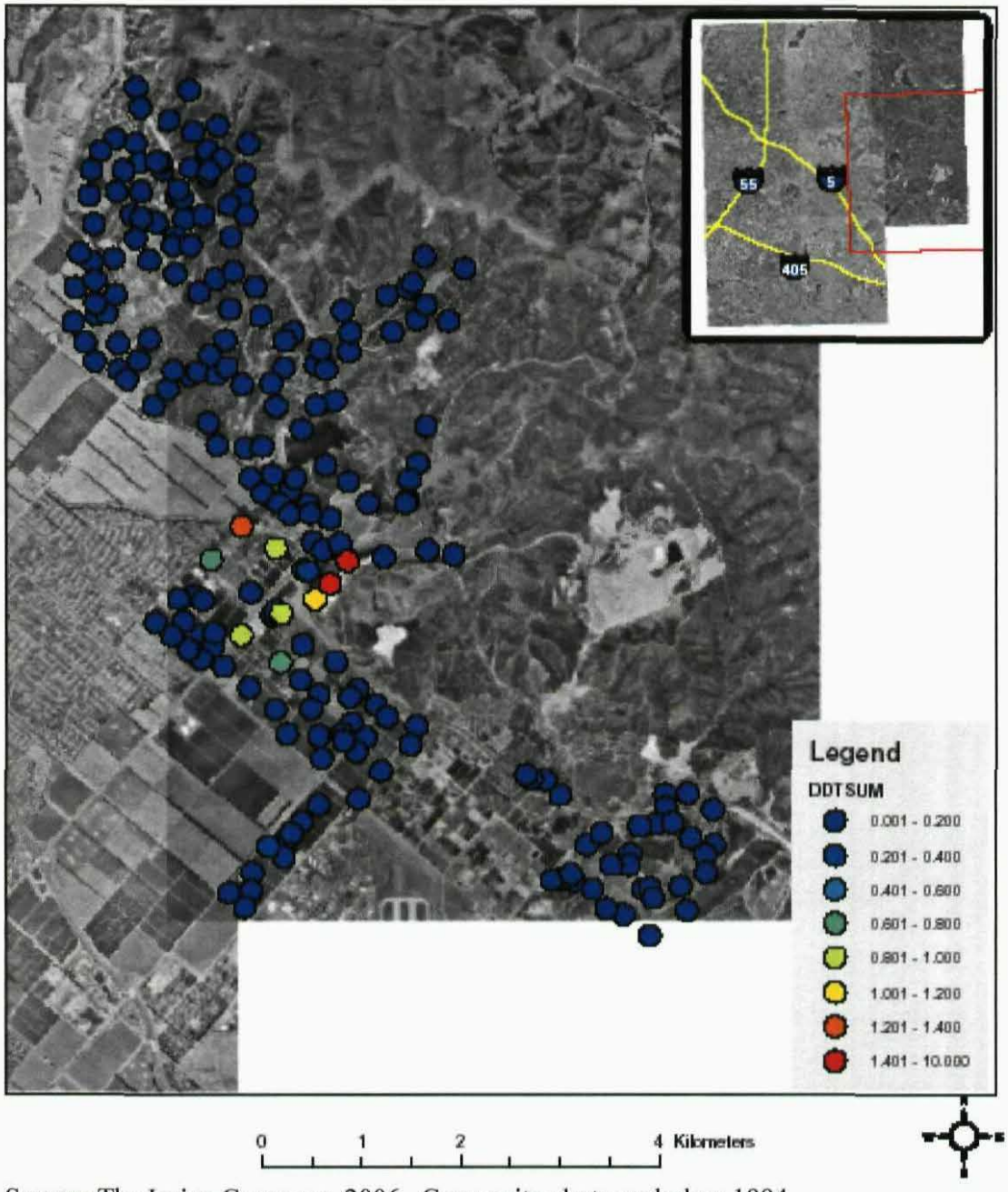
Source: Byard, 1987. Composite photo underlay: 1994.

Figure 7: DDT Concentrations in Agricultural Soils, 1989



Source: Byard, 1989. Composite photo underlay: 1994.

Figure 8: DDT Concentrations in Agricultural Soils, 2004



Source: The Irvine Company, 2006. Composite photo underlay: 1994.

For several reasons, the data presented in Table 3 and Figures 4 through 8 cannot be used to assess trends over time in agricultural soil DDT concentrations. First, as noted previously, the samples for each year were taken in different locations in the watershed, since the original purpose of the samples was not to determine concentration trends over time, but rather to assess site conditions for planning and development purposes. If concentrations were significantly different at each of the sampling locations in 1985, it would not be possible to determine whether there was in fact a trend in concentrations over time by sampling at different locations each year—the manner in which the samples reported in Table 3 were taken.

Second, the vast majority of the most recent samples from 2004 returned concentrations below the limit of detection for DDT. Of the zero- to six-inch depth samples, 168 of 230 samples (73%) were below the detection limit of 0.006 ppm (6 ppb). Thus a significant portion of these 168 samples could in fact be significantly lower than the concentration value of 0.006 ppm used for each non-detect (ND) sample in calculating the average concentration. If this is the case—which is highly probable from a strictly statistical perspective—then the average 2004 agricultural soil concentration could be significantly lower than the 0.07 ppm reported in Table 3. For other earlier years the number of ND samples was also significant, suggesting that average concentrations for those years could also be lower than reported in Table 3. This is particularly true of the earlier years since the detection limit was higher at those times than it currently is, due to less sensitive sample analysis techniques. Table 4 summarizes the number of ND values for each year and the corresponding detection limits.

Table 4: Number of DDT non-detect values and detection limits for zero- to six-inch depth agricultural soil samples in Newport Bay watershed

Sampling year	Detection limit (ppm)	Number of ND values	Total number of samples	ND percentage
1985	--	0	12	0 %
1987	--	0	10	0 %
1988	0.640	1	10	10 %
1989	0.016	6	15	40 %
1990	0.016	1	4	25 %
1991	0.016	2	17	12 %
1995	0.016	1	24	4 %
2004	0.006	168	230	73 %

It is also worth noting that several agricultural soil DDT data points were reported in Mischke et al. (1985) for Orange County. Total DDT concentrations in that report ranged from 0.321 ppm to 2.958 ppm for three different sample locations. However, the precise locations of these samples could not be identified from the report, and thus the data were not useful for establishing trends in agricultural soil DDT concentrations in the Newport Bay watershed.

Although agricultural soil data cannot be used to draw conclusions about local trends in DDT concentration over time, DDT decay rates in similar soils have been established in

the literature. These data indicate that overall DDT mass in the watershed must be declining since the use of DDT has stopped and since the fact that DDT decays over time is well established. The remaining mass of DDT in the watershed is less available for washoff and transport to Newport Bay given the ongoing changes in landuse from agricultural to developed conditions (see FACTORS AFFECTING ENVIRONMENTAL ORGANOCHLORINE CONCENTRATIONS).

FACTORS AFFECTING ENVIRONMENTAL DDT CONCENTRATIONS

Natural DDT Removal

The uniform downward trend in concentrations of DDT in fish and mussels in the watershed may be due to several factors. One important factor is simply the natural loss of these organochlorine compounds over time. The loss of DDT from soils is attributable to both volatilization and biodegradation. Volatilization tends to be the more important removal mechanism initially while biodegradation is more important later in the removal process (U.S. Dept. of Health and Human Services, 2002). As a result of both of these processes, DDT removal from soils tends to be non-linear, and thus the first 50% of the DDT tends to be removed from soil more quickly than subsequent halves. In other words, the half-life of a given quantity of DDT may decrease over time (Ibid).

A variety of studies have attempted to characterize the half-life of DDT and its metabolites. Dissipation of DDT is reported to be much quicker in tropical than in temperate regions. For 13 countries in tropical and sub-tropical regions, studies have shown the half-life of total DDT to range from 22 to 327 days (Racke et al., 1997). In temperate regions, the half-life of DDT has been reported to range from 2.3 years to 16.7 years (Lichtenstein and Schultz, 1959; Racke et al., 1997; Stewart and Chisholm, 1971). Dimond and Owen (1996) reported a mean half-life for the disappearance of DDT residues in sprayed forests in Maine of 20-30 years. Racke et al. (1997) reported the mean lifetime of DDT in temperate U.S. soils to be approximately 5.3 years.

If we conservatively assume a half-life of 20 years for DDT in soil, given that the use of DDT was banned in 1972 and excluding other loss or removal mechanisms, the mass of DDT in the agricultural soils of the Newport Bay watershed would have declined by at least 60% over the past 34 years due solely to natural removal. As noted previously, the empirically established concentration declines in Red Shiner and Mussels amount to DDT half-lives in tissue from those two species of 3.8 years and 5.2 years respectively, suggesting that the percentage of DDT removed from the watershed as a whole since 1972 may be much higher than 60%. Natural removal likely explains at least part of the empirically established concentration declines in Red Shiner, Mussels, and other key media presented previously.

Land Use

A steady conversion of land from agricultural to developed uses continues to reduce the quantity of DDT "available" for transport in storm flows. Organochlorine compounds

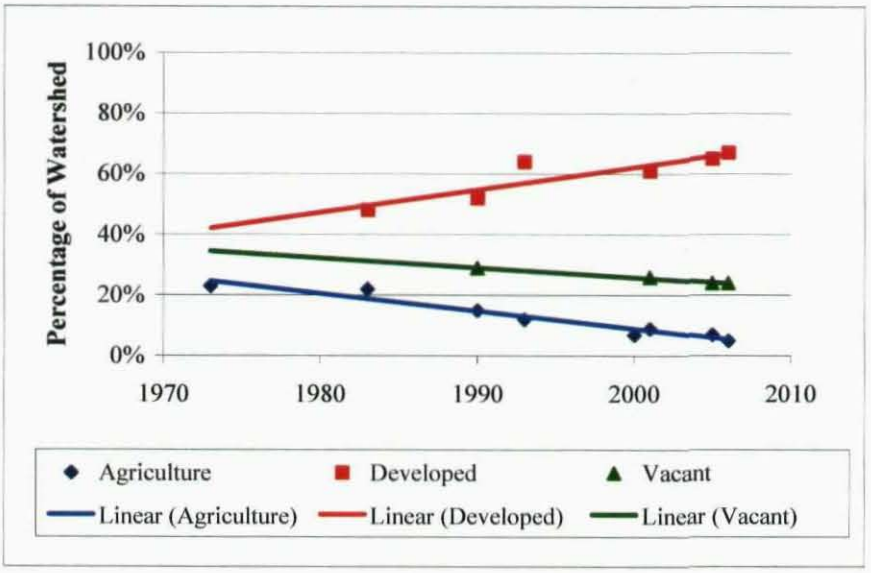
such as DDT have extremely low solubility in water and thus tend to sorb strongly to soil particles. Therefore, the predominant pathway for organochlorines to move in the watershed is soil erosion and transport, not soil leaching. As soils are transported to creeks and bays, organochlorine compounds sorbed to the soil particles are also transported. Thus, exposure of humans and biota to DDT in Newport Bay depends in large part upon the erosion and transport of sediment from the watershed to the Bay.

However, a recent report by WRC (2006) suggests that soil erosion and sediment loads in the watershed are declining. For example, for the San Diego Creek at Campus Drive monitoring station, WRC showed that while average annual flow volume for the years 2000-2005 is 85% of average annual flow volumes for 1983-1999 (indicating a rough parity between the two periods), average annual sediment discharge for 2000-2005 is only 42% of average annual sediment discharge for 1983-1999. This result is significant since sediment discharge is generally correlated with flow volume, and thus a reduction in sediment load would not be expected without a reduction in flow volume. WRC attributes this reduction in sediment load to development and erosion control measures in the watershed: "As the San Diego Creek watershed becomes further developed, less and less watershed supply of sediment is released during storm events" (WRC, 2006, p. 17).

This link between development and sediment load reductions suggests that development in the watershed has and will continue to reduce the amount of DDT available to biota in the watershed. Since development involves covering former agricultural areas—the original areas of organochlorine application and currently their dominant source in the watershed—by immobilizing sediment, development tends to immobilize DDT, reducing concentrations in downstream watershed areas. Given that development of formerly agricultural areas is occurring rapidly in the watershed, we would expect the availability of organochlorines from agricultural soils to be declining.

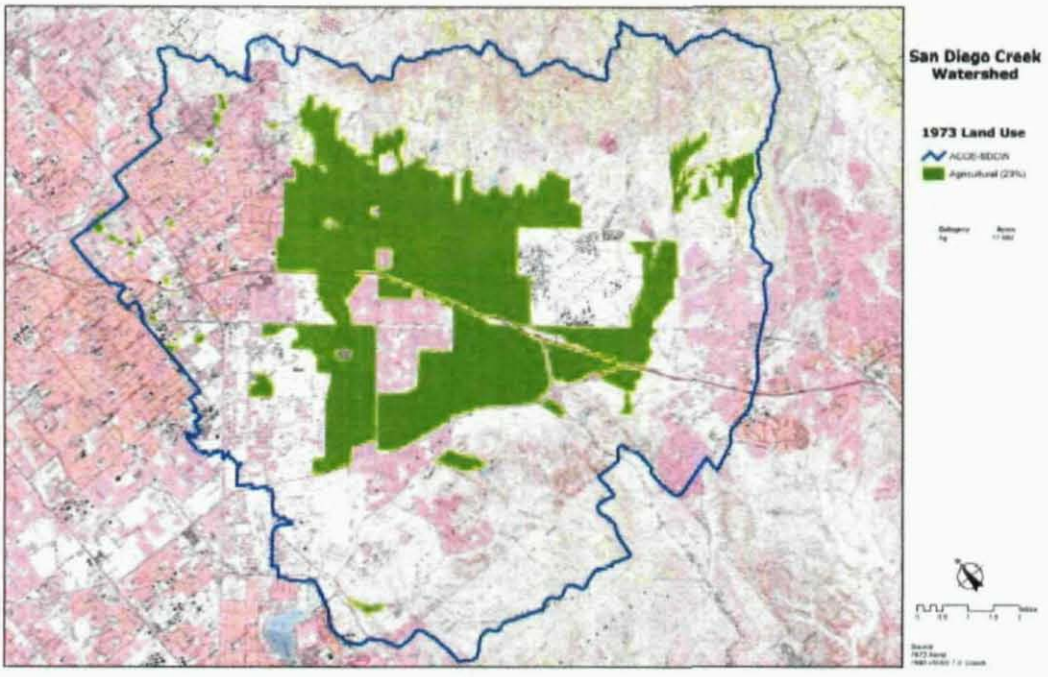
The extent of land-use change in the watershed in the recent past is significant. In 1983 agricultural uses accounted for 22 percent of the Newport Bay watershed while urban uses accounted for 48 percent. In 1993 agricultural use had declined to 12 percent of the watershed while urban use had increased to 64 percent (U.S. EPA, 1998). As of 2000 agricultural uses had dropped to approximately 7 percent of the watershed (U.S. EPA, 2002). These changes in land-use are evident in Figures 12, 13, and 14. Figure 12 provides a graphical representation of land in agricultural use in years 1973, 1983, 1990, 1993, 2000, 2001, and 2005 with projections for 2006. Given this established land-use trend, it is reasonable to expect the continued reduction of DDT concentrations in the watershed.

Figure 9: Land Use Trends in the San Diego Creek Watershed



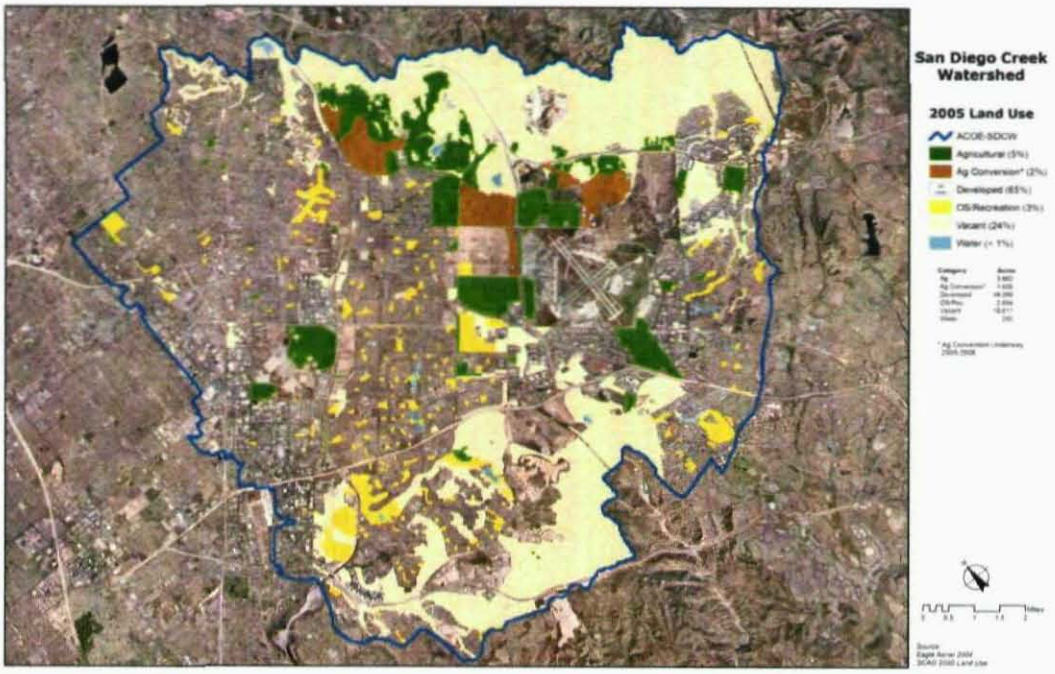
Land use data for years 1973, 1990, 2001, and 2005 was determined by GIS analysis of San Diego Creek Watershed land use maps by The Irvine Company (2005).
Land use data for years 1983, 1993, and 2000 are from USEPA (1998) and USEPA (2002).

Figure 10: Agricultural and Vacant Space Land Use in San Diego Creek Watershed, 1973



Source: The Irvine Company, 2006.

Figure 11: Agricultural and Vacant Space Land Use in San Diego Creek Watershed, 2005



Source: The Irvine Company, 2006.

Dicofol

It is sometimes suggested that other non-organochlorine pesticides, which continue to be used in the watershed, include small amounts of organochlorines such as DDT, and thus constitute an ongoing source of organochlorines in the watershed. Dicofol is sometimes offered as an example of this kind of pesticide. The following summary is offered at a web-site for the Extoxnet program, a cooperative pesticide information program supported by Cornell University, Oregon State University, the University of Idaho, the University of California at Davis, and Michigan State University:

> Dicofol is an organochlorine miticide used on a wide variety of fruit, vegetable, ornamental and field crops. Dicofol is manufactured from DDT. In 1986, use of dicofol was temporarily canceled by the EPA because of concerns raised by high levels of DDT contamination.[1]

However, the EPA has reinstated dicofol as a legal pesticide after finding that dicofol contains minimal levels of DDT. Mischke et al. (1985) concluded that DDT levels in dicofol were too low to account for the DDT soil residues found in their 1985 study. Thus, only minimal quantities of DDT are associated with dicofol. Therefore, even though small amounts of dicofol continue to be used in the Newport Bay watershed—available data from

[1] http://extoxnet.orst.edu/pips/dicofol.htm.

the California Department of Pesticide Regulation suggests that approximately 1 pound per year of dicofol has been applied annually in the watershed since 1990—dicofol does not represent a significant source of DDT in the watershed.

Sources Outside Newport Bay

Some biota can be helpful indicators of the level of organochlorine compounds still extant within the Newport Bay watershed. For example and as noted previously, red shiners and mussels in San Diego Creek and Newport Bay indicate a consistent declining trend in organochlorine concentrations in the watershed. However, these species are good indicators only because their entire life-cycle is localized within the watershed. Therefore, their exposure to organochlorines is directly related to the presence of these chemicals in the watershed. Other fish species that are year-round residents of Newport Bay may also be helpful indicators of organochlorine levels in the watershed. The following fish species have been collected in Newport Bay in both summer and winter and are thus believed to be year-round residents of the Bay: California killifish, Pacific staghorn sculpin, spotted sand bass, barred sand bass, black perch, arrow goby, California halibut, and diamond turbot (Allen et. al., 2004, p. 14).

The corollary to this point is that non-resident fish—fish that ordinarily spend significant portions of their life-cycle outside Newport Bay—will not be good indicators of organochlorine levels in the Newport Bay watershed, insofar as such species could have accumulated organochlorines in their tissue at ocean locations outside the Bay. As James Allen notes in his recent study of contaminant bioaccumulation in Newport Bay fish, "monitoring studies are needed to determine if elevated DDT levels in the popular sport fishes noted above are due to contamination in the bay or to sources outside the bay" (Allen et al., 2004). Until it is established that a particular fish species is a year-round resident of the Bay, and thus is not subject to organochlorine sources outside the Newport Bay watershed, it is not scientifically justifiable to infer the presence of organochlorine compounds in the watershed from the presence of such compounds in the tissue of the particular species.

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Appendix A: Quantitative Environmental Analysis Technical Memorandum

Quantitative Environmental Analysis, LLC

∂c/∂t

TECHNICAL MEMORANDUM

TO:	Susan C. Paulsen - Flow Science, Inc.	**DATE:**	October 3, 2006
FROM:	Deborah Chiavelli, Ph.D. and John Connolly, Ph.D., P.E., DEE	**RE:**	Analysis and Results of the Newport Bay and San Diego Creek DDT Trends
CC:	Files	**JOB#:**	IRVnew:110

Quantitative Environmental Analysis, LLC (QEA's) Task: Assess the temporal trends in DDT concentration and statistical power of the combined evidence of these trends in multiple sources.

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1. DATA SOURCE

QEA received the data in a Microsoft Excel file from Aaron Mead of Flow Science on April 5, 2006. QEA was informed by Mr. Mead that all relevant data had been combined in the *FISH DDT SUMMARY*, *MUSSEL DDT SUMMARY*, and *SEDIMENT DDT SUMMARY* pages of the Excel file, and QEA did not attempt to analyze any data not on these summary pages.

2. DATA DESCRIPTION AND ASSESSMENT

2.1 Locations

QEA was asked to separate the data among three general locations for analysis: San Diego Creek (SDC), Upper Newport Bay (UNB) and Lower Newport Bay (LNB).

2.2 Fish

2.2.1 Red Shiners

After removing the two Delhi Creek samples, which were collected outside the study area of concern, all red shiner data are from SDC. The data set includes 54 samples spanning the time period from 1983 to 2002. There are multiple samples per year, no missing years, and the majority of the data are from one source. Each sample is a composite of at least 15 fish, and typically more than 30. These characteristics make the red shiner data set particularly strong as a basis for trend analysis. Preliminary regression analysis revealed three outlying points (Figure 1). These outliers were removed in order to improve the predictive power of the regression. All analyses for red shiners were conducted with and without the outliers removed.

2.2.2 Other Fish

The data sets for all other fish species contained too few samples to conduct robust independent trend analysis. However, these data do provide a basis for assessing whether the temporal patterns in the sampled species are consistent with that seen for the red shiners. DDT trends in other fish species were compared to the red shiner data (method described later) for species with three or more data points from the same tissue type for a time range spanning five or more years.

2.3 Mussels

Mussel data are available from SDC, LNB, and UNB. The LNB and UNB mussel data cover the period from 1982 to 1999; N = 18 and 35 respectively. The SDC data cover the period from 1984 to 1993, N = 15. The shorter time span, lack of recent data and low N (15) make the SDC data less robust than the data from the other sites for predicting future trends.

2.4 Sediment Data

Sediment data are available from LNB and UNB. Sample dates range from 1980 to 2004, but there is a long gap where no sampling occurred (1987 – 1995), making regression analysis for the entire time period somewhat problematical. Multiple data sources apparently also contributed to a poor fit to the log-linear model, making the sediment data generally weak. This is discussed further in the Results section.

3. RESULTS AND CONCLUSIONS

3.1 Temporal Trends for the Full Time Span

Red shiners - Following convention, an exponential model is used to describe trends. ln(red shiner DDT concentration) is regressed against year, as exact sample dates are not available. There is a highly significant exponential decline in DDT concentration for red shiners (Table 1, Appendix), and the rate of decline increases from -0.174 to -0.183 per year with the three outlying points removed from the data set (Appendix). Residual analysis indicates a good fit of the log-linear regression to the data after the outliers are removed, and the placement of the outliers does not suggest any alternative regression model would be more appropriate.

Mussels – ln(mussel DDT concentration) is regressed against Julian day, and slope and 95% Confidence Interval (CI) are multiplied by 365 to obtain yearly DDT decline rate (Appendix). There is a significant decline in DDT concentration for mussels when all locations are analyzed together and also for each of the three locations analyzed separately (Table 1, Appendix). Residual analysis indicates a good fit of the log-linear regressions to the mussel data in all cases.

Sediments – ln(sediment DDT concentration) is regressed against Julian day and slope and 95% CI are multiplied by 365 to obtain yearly rates (Appendix). Consistent with the shiner and mussel data, a declining trend is shown; although the decline rate is lower than that in red shiners or mussels. This was true whether both locations were combined or UNB and LNB were considered separately (Table 1), and the 95% CI for all three sediment slopes encompassed zero (Appendix). Hence the declining trend is not statistically significant, but the ten-year gap between early and

late data, and the poor fit of the data in residual analysis (Figure 2) argue against drawing any conclusions from this result.

3.2 Temporal Trends in Split Time Spans

Rationale - Data have been split by early and later times for all three data types in order to assess whether the decline rate has changed for red shiners and mussels, and because the large temporal gap in the sediment data makes regression analysis of the entire time span somewhat questionable. Furthermore, identical DDT levels for the UNB location of UNBSDC and the LNB location of LNBRIN for the dates May 12-13, 1983, December 9, 1983, June 8, 1984, and January 18, 1986 indicate that there may be a recording error in the early sediment data that QEA received, although perhaps these levels reflect the laboratory detection limits for those sample dates.

Split placement - For red shiners, early data range from 1983 to 1992, and late data from 1993 to 2002, giving a ten-year span for both early and late data. For mussels, early data range from 1982 to 1990, and late data from 1991 to 1999, giving a nine-year span for both early and late data. QEA has not analyzed late data for the SDC location, because sampling ended there in 1993. For sediments, early data have a seven-year span from 1980 to 1986 and late data have a nine-year span from 1996 to 2004.

Early vs. late comparison - For all three data types (red shiners, mussels, and sediments) decline rates of DDT were greater during the early time period, and the 95% CI around the slopes for the early time spans do not include the slopes of the later time spans for red shiners and for mussels in LNB (Table 1, Appendix). This indicates that the rate of decline of DDT in the Newport Bay system has slowed over time and that prediction of future DDT levels should probably rely on more recent data rather than on the entire time span if possible.

Table 1. Regression summary for Newport Bay DDT data.

For each DDT pool (red shiners, mussels, sediments) analyses have been conducted for all locations combined and for SDC, UNB, and LNB separately; and analyses have been conducted for the entire time span of the samples as well as early and late samples separately (see text). In each field, Row 1 = N, Row 2 = yearly rate of decline (regression slope), Row 3 = R2 of the regression, and Row 4 = P value of the regression. See Appendix for confidence intervals around slopes and for projections of future DDT levels.

	Red shiners*			Mussels			Sediments		
Location	All	Early	Late	All	Early	Late	All	Early	Late
San Diego Creek	51 -0.183 0.775 <0.0001	24 -0.245 0.759 <0.0001	27 -0.135 0.296 0.003	15 -0.292 0.458 0.006	10 -0.316 0.467 0.029	NA	NA	NA	NA
Upper Newport Bay	NA	NA	NA	18 -0.095 0.537 0.0005	9 -0.169 0.499 0.033	9 -0.072 0.141 0.319	84 -0.021 0.030 0.117	22 -0.378 0.248 0.018	62 -0.0615 0.027 0.206
Lower Newport Bay	NA	NA	NA	35 -0.156 0.507 <0.0001	26 -0.268 0.569 <0.0001	9 -0.029 0.020 0.719	67 -0.043 0.116 0.005	15 -0.621 0.486 0.004	52 -0.184 0.199 0.0009
All Locations Combined	NA	NA	NA	68 -0.133 0.392 <0.0001	45 -0.236 0.440 <0.0001	23 -0.011 0.002 0.828	151 -0.030 0.059 0.003	37 -0.473 0.345 0.0002	114 -0.107 0.075 0.003

Notes:
**Statistics for red shiners are with three outlying points removed. Statistics with outliers included are in Appendix.*

Late trend in red shiners – The decline rate of DDT in red shiners for the late time span (with outliers removed) is approximately half that seen in the early time span, but still highly significant (Table 1). Residual analysis indicates a good fit of the log-linear regressions to the late red shiner data after the previously-mentioned outliers are removed.

Late trends in mussels – The late time span decline in DDT with time for mussels is not statistically significant in either UNB or LNB, and the rates are approximately one half and one tenth of the early time span rates, respectively. Residual analysis indicates a good fit of the log-linear regressions for late mussel data. Power (probability that a Type II error did not occur) was calculated for these non-significant mussel trends and was found to be very low (0.16 and 0.06 for UNB and LNB respectively). The low sample size (N = 9 for both LNB and UNB) is a contributor to the low power of these regressions.

As their slopes are not significantly different, late UNB and LNB mussel data were combined in an analysis of covariance (ANCOVA) in order to increase the statistical power to test for the temporal effect on DDT; however ANCOVA results still found no significant effect of date on DDT concentration.

It is important to remember that the time division for early vs. late is an arbitrary one based on splitting the time span in half and that the decline in mussel DDT levels is significant when the entire time span is analyzed.

Late trends in sediments - There is a significant decline in sediment DDT for the late time span in LNB, and DDT declined in UNB, but not significantly. However, there is a poor fit to the model according to residual analysis, and for both locations the regression is strongly affected by a set of early data points for each location (Figure 2). These data points are all from the same period (March 15, 1997 for UNB and July 21-23, 1998 for LNB) and same source for each location and all have positive residuals to the regression line (with one exception for UNB). There is also a relatively large time gap between these data points and later data. Residuals from some of the other one-source/one-date sample groups are predominantly negative (Figure 2). If the early group of data points is removed, DDT rate of change for the late time span becomes positive for both locations, significantly so for UNB (Appendix). The obvious source/agency bias in residuals, and the strong effect of the one set of early points in each location on the direction of DDT trend with time makes drawing inferences from the late sediment data problematical in spite of the relatively large number of data points and high power for the LNB regression (power is 0.24 and 0.93 for UNB and LNB respectively).

There are several additional factors that likely reduce the dependability of the sediment data. The samples analyzed here have not all been collected from the same depths, which adds variability to the data that cannot be accounted for as exact sample depths are currently unavailable. Additionally, there has been periodic dredging of Newport Bay, and sediment transport rates from the watershed have declined over time as the watershed has become less pervious and channels have been stabilized. Given these problems, it is preferable to draw conclusions about DDT trends in Newport Bay from the trends seen in fish and mussels rather than from sediment data.

3.3 Predicted DDT Values

For all regression analyses with either the entire sample time period or the later time spans, QEA predicted DDT concentrations with a 95% prediction interval. The Appendix contains both short-term predictions (for 2006, 2008, and 2010; Section 5.1.1), and long-term predictions (every 5 years from 2006 to 2076, a 70-year span; Section 5.1.2). The long-term predictions were calculated because one of the main uses for the numbers is to compare concentrations in biota with human health tissue thresholds, which were derived based on the assumption that tissue

concentrations would remain constant over a 70-year human lifetime. This assumption is clearly inapplicable for this pollutant in these circumstances. There is furthermore a statistical problem in that the prediction intervals as one moves forward in time quickly become large due to uncertainty in predicting trends when using models with a high level of unexplained variability. We have presented values for a 70-year period only for those data sets where the level of unexplained variability is low enough such that the 95% prediction interval in 2076 is less than the relevant regulation threshold (100 ppb; see Appendix).

3.4 Other Fish

Temporal trends in fish other than red shiners - The seven fish species other than red shiners with three or more DDT concentration data points from the same tissue type for a time range spanning five or more years all have declining trends in DDT concentration with time. The fish species meeting these criteria (black perch, California halibut, California killifish, diamond turbot, spotted sand bass, striped mullet, and yellow fin croaker) each have only three to five data points, and some samples are from only one fish as opposed to being a mean or composite of multiple fish. In most cases there is also a gap of approximately 20 years between the early and late data points. There was no attempt to further parse these data by location (i.e., SDC, LNB, and UNB). All of these problems weaken any inferences drawn from these data, however the fact that negative trends in DDT concentration are seen in all seven species supports the conclusion that DDT levels in the biota of this system are declining.

Comparison of other fish to red shiner temporal trend - The following additional analysis has been performed for these data. For each fish species, all data points are multiplied by a constant to achieve least squares minimization between observed data points and predicted DDT levels in red shiners for that year (Table 2; using the red shiner regression with outliers removed). This is done to scale the data for the other fish species to the red shiner data. Then each data point for each fish is checked to see if it falls within the 95% prediction interval for red shiner DDT concentration for that year (Table 2).

All of the data points for four of the seven fish species fall within the 95% PI for red shiners, indicating a similar decline with time. Half or more data points fall within the 95% PI for the other three fish species, indicating the decline was faster for diamond turbot, and slower for striped mullet and yellow fin croaker.

Table 2. Comparison of DDT trends in other fish species to red shiner predicted values based on the red shiner regression equation.

									95% Prediction Interval for given year		
Fish Species	Year	DDT (ppb)	ln(DDT)	Scaling Constant	Observed = ln(DDT)*constant	Expected = red shiner predicted	$(obs-exp)^2$	Sum of $(obs-exp)^2$	Lower	Upper	Observed is within PI?
Black Perch	1992	48	3.87	1.63	6.31	6.89	0.34	0.96	5.89	7.89	Y
	1999	28	3.33	1.63	5.43	5.61	0.03		4.59	6.62	Y
	2001	40	3.69	1.63	6.01	5.24	0.59		4.22	6.27	Y
California Halibut	1980	628	6.44	1.36	8.78	9.08	0.09	0.37	8.01	10.15	Y
	2000	51	3.93	1.36	5.35	5.42	0.01		4.41	6.44	Y
	2001	69	4.23	1.36	5.77	5.24	0.28		4.22	6.27	Y
Spotted Sand Bass	1978	680	6.52	1.30	8.46	9.45	0.98	5.37	8.36	10.53	Y
	1990	277	5.62	1.30	7.29	7.25	0.00		6.25	8.26	Y
	1991	110	4.70	1.30	6.10	7.07	0.95		6.07	8.07	Y
	2001	68	4.22	1.30	5.47	5.24	0.05		4.22	6.27	Y
	2002	204.9	5.32	1.30	6.90	5.06	3.40		4.03	6.09	Y
Striped Mullet	1978	4210	8.35	1.14	9.49	9.45	0.00	4.90	8.36	10.53	Y
	1978	1440	7.27	1.14	8.27	9.45	1.37		8.36	10.53	N
	1980	2070	7.64	1.14	8.69	9.08	0.15		8.01	10.15	Y
	2002	428.2	6.06	1.14	6.89	5.06	3.37		4.03	6.09	N
Yellowfin Croaker	1978	200	5.30	1.55	8.20	9.45	1.56	5.98	8.36	10.53	N
	1980	310	5.74	1.55	8.88	9.08	0.04		8.01	10.15	Y
	1999	23	3.13	1.55	4.84	5.61	0.59		4.59	6.62	Y
	1999	47	3.85	1.55	5.96	5.61	0.12		4.59	6.62	Y
	2001	102	4.62	1.55	7.16	5.24	3.66		4.22	6.27	N
Diamond Turbot*	1997	152	5.02	1.42	7.14	5.97	1.35	4.02	4.79	7.16	Y
	1999	18	2.89	1.42	4.11	5.70	2.55		4.52	6.89	N
	2001	36	3.58	1.42	5.09	5.43	0.12		4.24	6.63	Y
California Killifish*	1993	353	5.87	1.12	6.56	6.52	0.00	0.03	5.32	7.71	Y
	1993	364	5.90	1.12	6.59	6.52	0.01		5.32	7.71	Y
	2002	100	4.61	1.12	5.15	5.30	0.02		4.09	6.50	Y

*Notes: *Used red shiner late regression to fit the data because all years were in late regression.*

4. SUMMARY

The available sediment data are too problematical to use for trend analysis and it is not recommended that the sediment data be used to infer any current or future trends in DDT levels in the Newport Bay system.

There is strong evidence that DDT levels are declining in the biota of the Newport Bay system when the entire temporal span of the available data is considered. There are statistically significant declines in DDT in red shiners, and in mussels in SDC, UNB, and LNB. Additionally there are declining trends in seven other fish species although there are not enough data for robust statistical analysis in these species. The likelihood of having so many independent data sets show a declining trend if a downward trend did not exist, is vanishingly small. For example, if there were no trend at all, there is a 50% chance of randomly getting a positive or negative trend from any given data set, and the probability of getting 11 negative trends is 0.0005 (i.e., 0.5^{11}).

If only more recent data are considered, there is still a significant negative trend in DDT for the red shiners, but the power of the available data to indicate DDT trends is too weak in the mussel data for UNB and LNB and there are no recent mussel data for SDC. Data for two of the fish species other than red shiners also indicate negative DDT trends in more recent years (since 1993). Repeating the above exercise, the probability of getting 5 negative trends by chance is 0.03 – still relatively small. In conjunction with the data for red shiners, which comprise the one data set adequate to indicate trends in more recent years, this provides support for the conclusion that DDT levels are still in general decline in the Newport Bay system.

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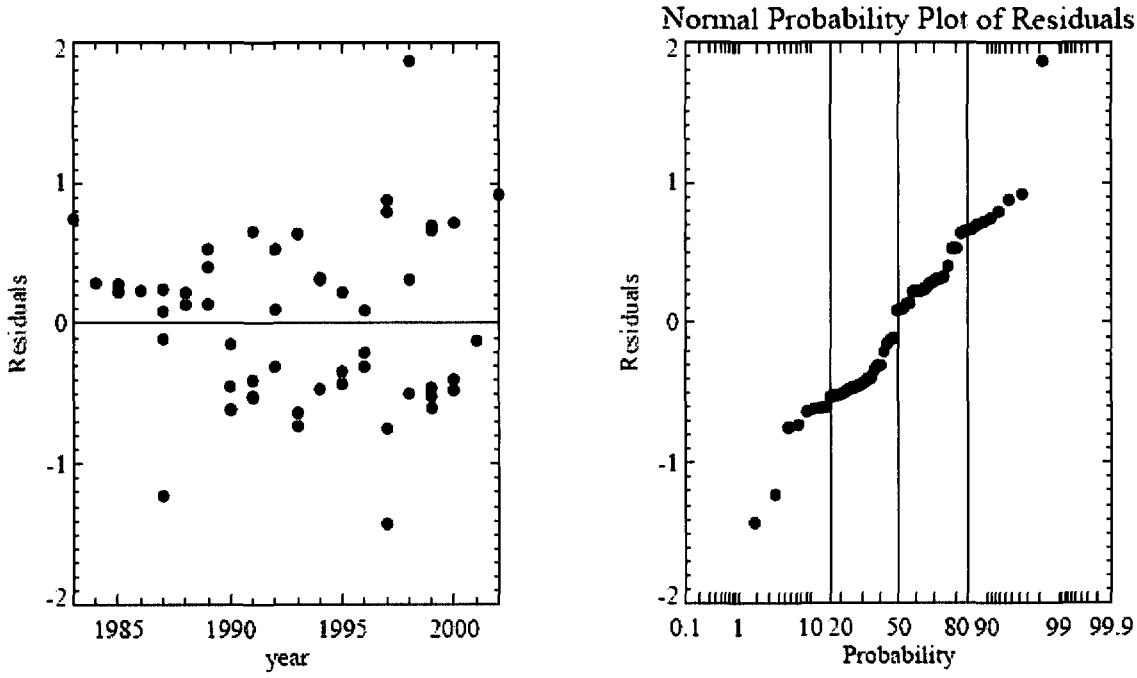
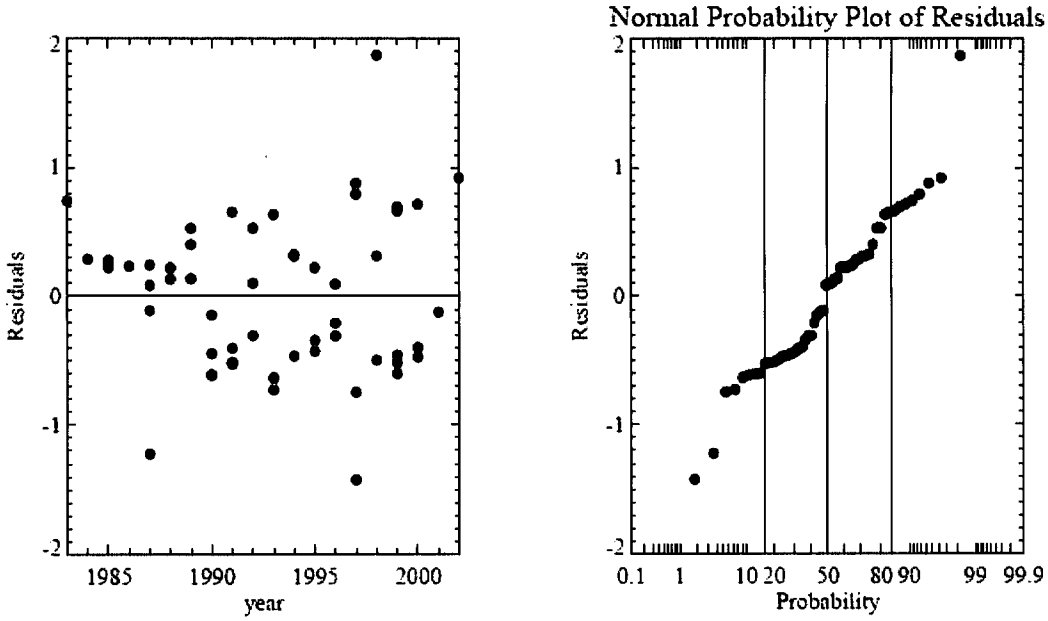
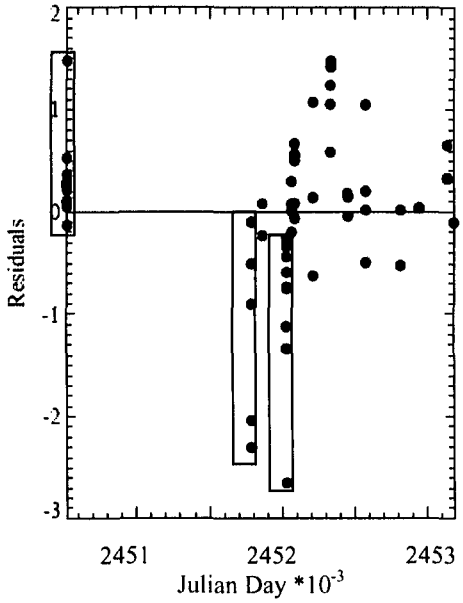


Figure 1. Residual plots for regression of red shiners ln(DDT) vs. year, clearly showing the three outliers.
Regression analysis was performed both with and without outliers.

Figure 1. Residual plots for regression of red shiners ln(DDT) vs. year, clearly showing the three outliers. Regression analysis was performed both with and without the outliers.

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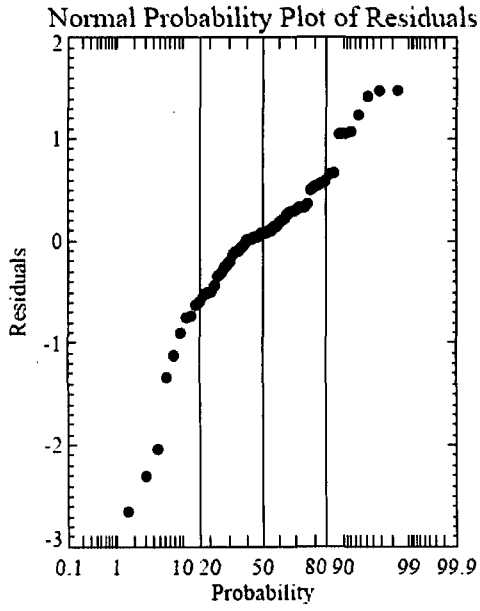
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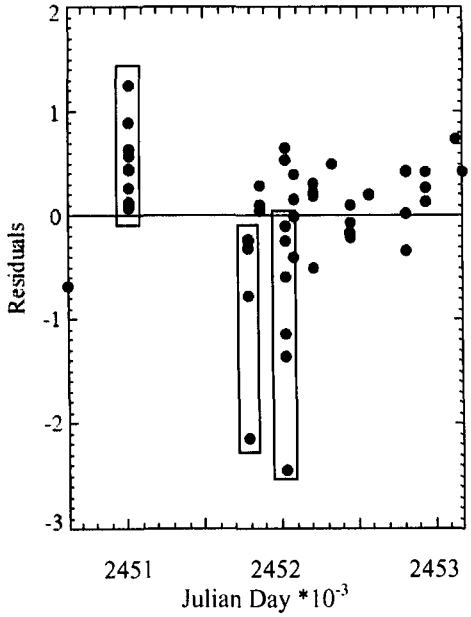
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2453
Julian Day *10-3

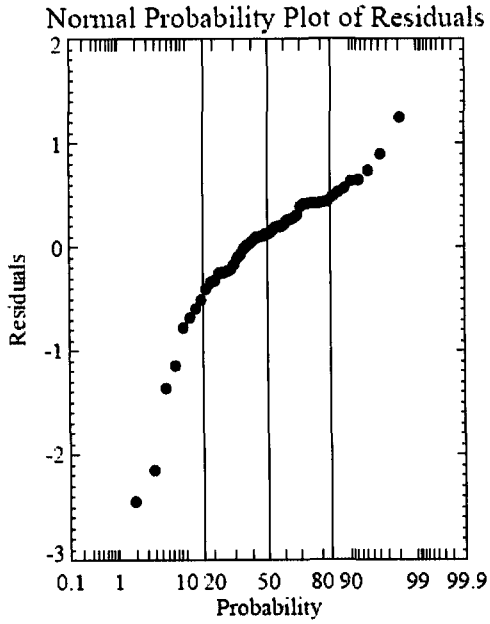
Normal Probability Plot of Residuals
Residuals
2
1
0
-1
-2
-3
0.1
1
10 20
50
80 90
99
99.9
Probability

Figure 2. Agency bias seen in residual plots for regression of sediment ln(DDT) vs. Julian day from 1996 through 2004.

Upper panels: Upper Newport Bay. Lower panels: Lower Newport Bay. Note that for each location, an early group of samples from the same date, which were taken by the same agency, have predominantly positive residuals. Also note two sets of negative residuals all on the same day in each location, which were from two additional sampling agencies.

5. APPENDIX

5.1 Analysis Program Output

5.1.1 Regression Parameters and Short Term Regression Predictions

This section contains regression parameters and 95% confidence intervals around slopes, for all Upper and Lower Newport Bay (UNB, LNB) and San Diego Creek (SDC) DDT analyses. Projections of future DDT values are presented for both entire time span and late time span data sets. Observed DDT data and the predicted regression model values are plotted for all regressions. For mussel and sediment data, slopes from Julian day regressions are multiplied by 365 to give yearly decline rates. Note that red shiner analyses are included with and without outliers removed (NO_OUTRM/OUTRM) and that late period sediment analyses are included with and without the early single-source data points removed (UNB_97RM and LNB_98RM; see text and Figures 1 and 2 for details).

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REGRESSION PARAMETERS FOR RED_SHINERS_NO_OUTRM

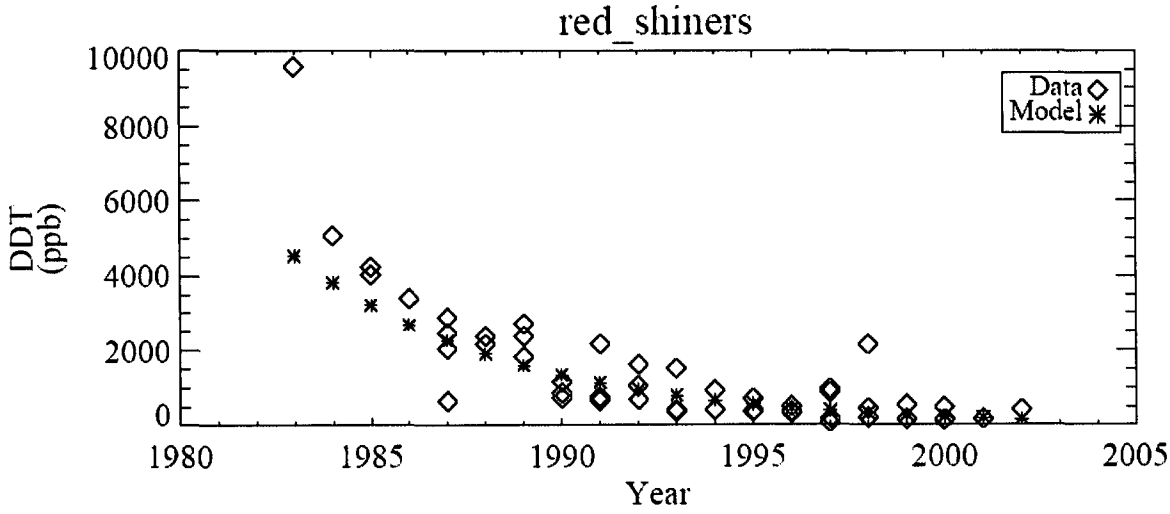
N: 54
R: 0.82138577 R2: 0.67467458
P: 2.7755576e-014 F: 107.83995

Intercept: 353.04215 Variance of Intercept: 1119.5741

Slope: -0.17378767 Variance of Slope: 0.00028182994

95% slope Confidence Interval
lower: -0.20736916
upper: -0.14020618

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 83.436119 +/- 3.6293264
2008 estimate: 58.939174 +/- 3.7177681
2010 estimate: 41.634561 +/- 3.8195652



REGRESSION PARAMETERS FOR RED_SHINERS_OUTRM

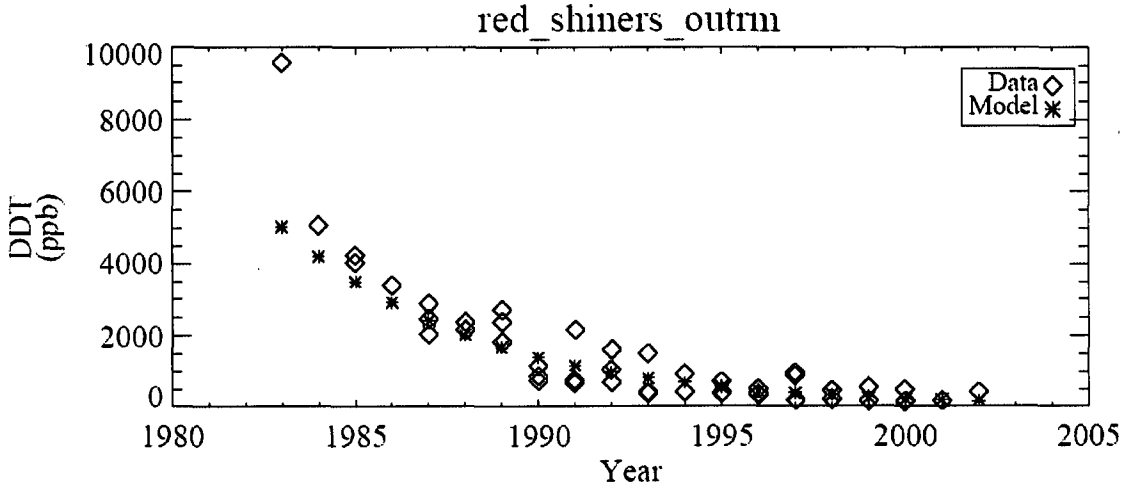
N: 51
R: 0.88046974 R2: 0.77522696
P: 0.00000000 F: 168.99767

Intercept: 370.91249 Variance of Intercept: 783.25042

Slope: -0.18274629 Variance of Slope: 0.00019717771

95% slope Confidence Interval
lower: -0.21099590
upper: -0.15449667

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 75.447468 +/- 2.8758861
2008 estimate: 52.349587 +/- 2.9365017
2010 estimate: 36.323012 +/- 3.0059241



Quantitative Environmental Analysis, LLC

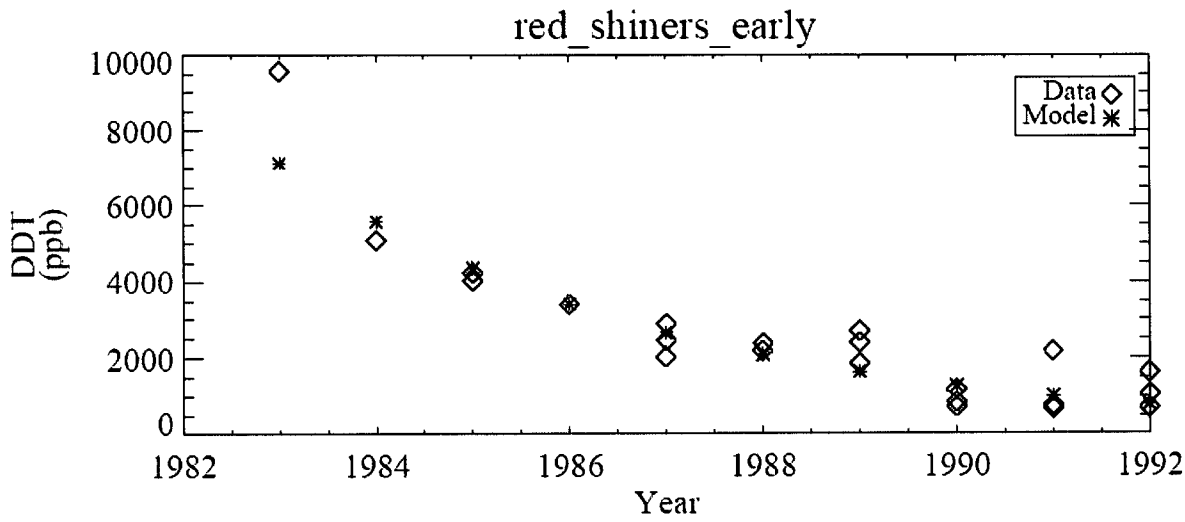
REGRESSION PARAMETERS FOR RED_SHINERS_EARLY_OUTRM

N: 24
R: 0.87114113 R2: 0.75888686
P: 3.0517636e-008 F: 69.243473

Intercept: 495.56046 Variance of Intercept: 3535.0105

Slope: -0.24543045 Variance of Slope: 0.00089389024

95% slope Confidence Interval
lower: -0.30659804
upper: -0.18426286



REGRESSION PARAMETERS FOR RED_SHINERS_LATE_OUTRM

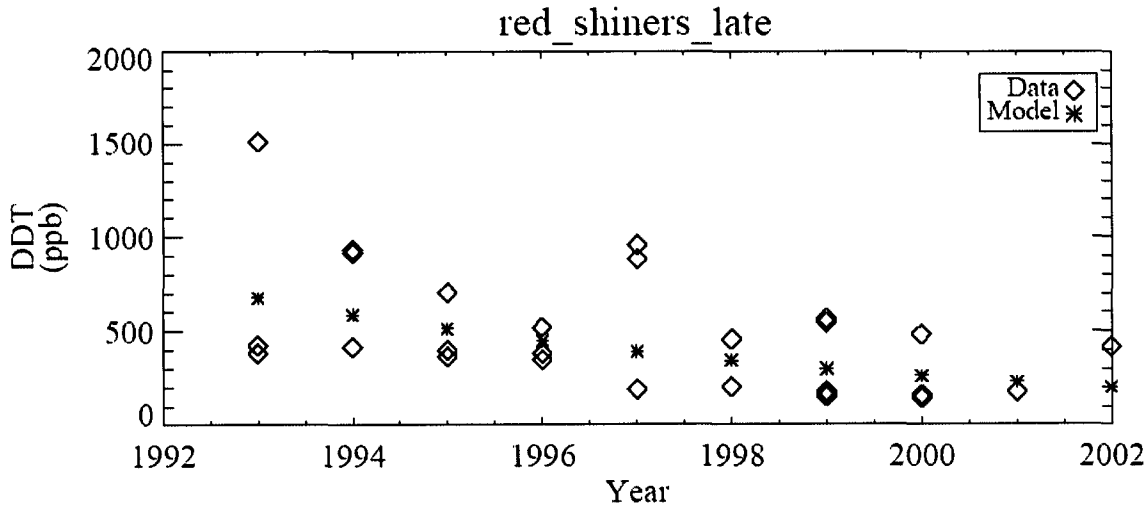
N: 27
R: 0.54434948 R2: 0.29631635
P: 0.0033306361 F: 10.527328

Intercept: 276.23456 Variance of Intercept: 6829.1442

Slope: -0.13533295 Variance of Slope: 0.0017124167

95% slope Confidence Interval
lower: -0.22123721
upper: -0.049428689

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 116.35716 +/- 4.0061304
2008 estimate: 88.765541 +/- 4.4387959
2010 estimate: 67.716687 +/- 4.9769836



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REGRESSION PARAMETERS FOR RED_SHINERS_LATE_NOOUTRM

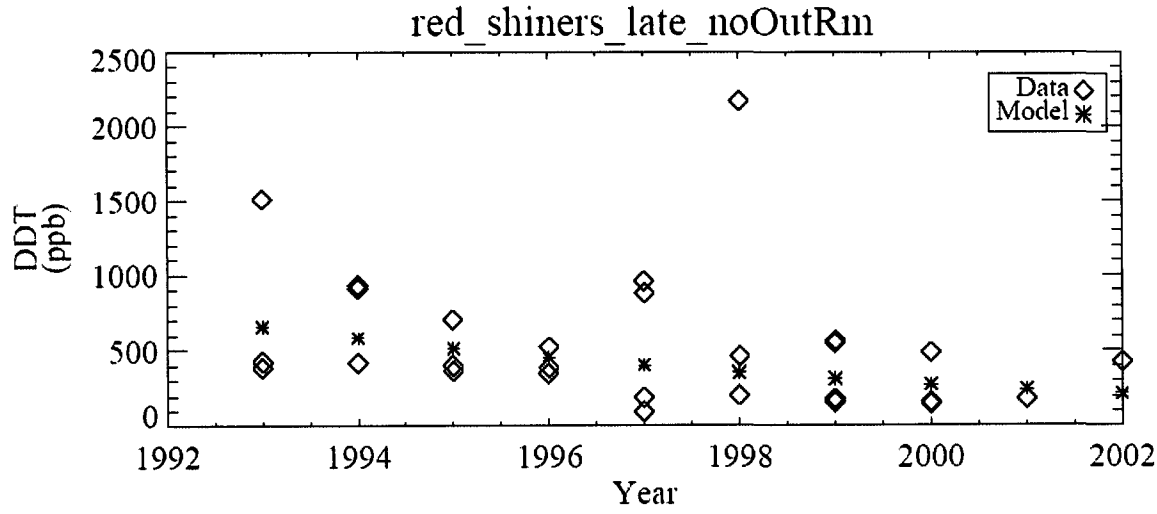
N: 29
R: 0.42084146 R2: 0.17710754
P: 0.023004859 F: 5.8110916

Intercept: 256.08298 Variance of Intercept: 11268.329

Slope: -0.12523474 Variance of Slope: 0.0028254504

95% slope Confidence Interval
lower: -0.23182992
upper: -0.018639551

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 129.29506 +/- 5.6026661
2008 estimate: 100.64783 +/- 6.3598223
2010 estimate: 78.347820 +/- 7.3270052



REGRESSION PARAMETERS FOR MUSSELS_ALL_LOCATIONS_COMBINED

N: 68
R: 0.62574206 R2: 0.39155313
P: 1.1589056e-008 F: 42.472905

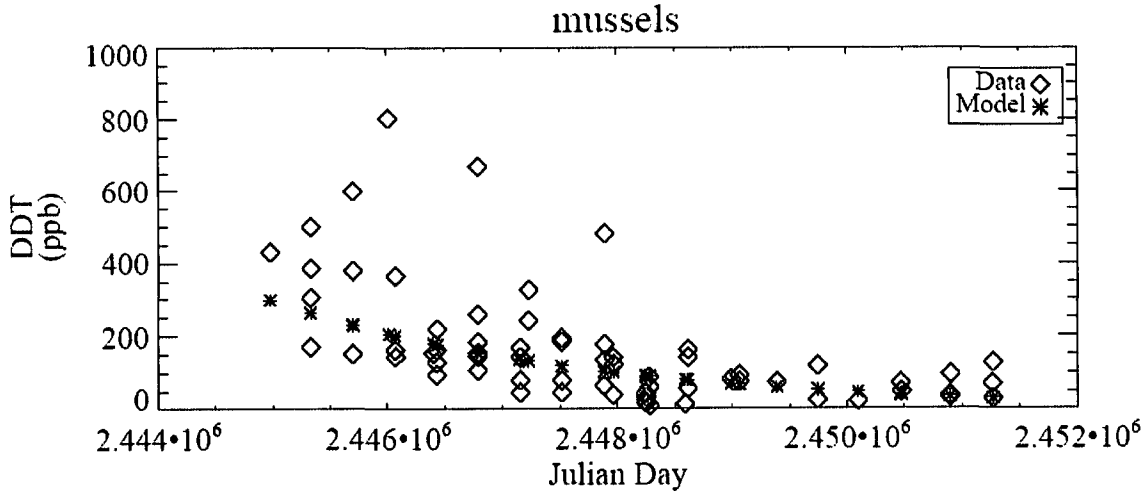
Intercept: 895.31354 Variance of Intercept: 18677.066

Slope: -0.00036385209 Variance of Slope: 3.1170069e-009

95% slope Confidence Interval
lower: -0.00047532058
upper: -0.00025238359

yearly rate (slope * 365) -0.13280601
yearly lower slope CI: -0.17349201
yearly upper slope CI: -0.092120009

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 11.719759 +/- 5.3290373
2008 estimate: 8.9826989 +/- 5.5154996
2010 estimate: 6.8848580 +/- 5.7263803



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REGRESSION PARAMETERS FOR MUSSELS_SDC

N: 15
R: 0.67696386 R2: 0.45828007
P: 0.0055706136 F: 10.997640

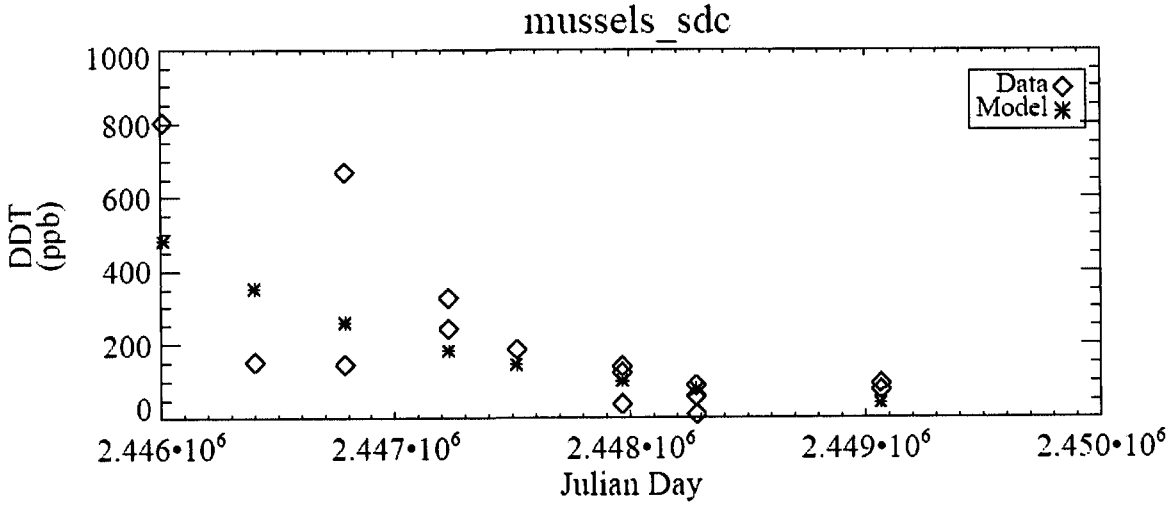
Intercept: 1965.0688 Variance of Intercept: 349384.86

Slope: -0.00080085073 Variance of Slope: 5.8318138e-008

95% slope Confidence Interval
lower: -0.0013225617
upper: -0.00027913980

yearly rate (slope * 365) -0.29231052
yearly lower slope CI: -0.48273501
yearly upper slope CI: -0.10188603

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 0.87881890 +/- 40.661958
2008 estimate: 0.48938865 +/- 57.061376
2010 estimate: 0.27252629 +/- 80.636058



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REGRESSION PARAMETERS FOR MUSSELS_UNB

N: 18
R: 0.73264393 R2: 0.53676713
P: 0.00054405210 F: 18.539863

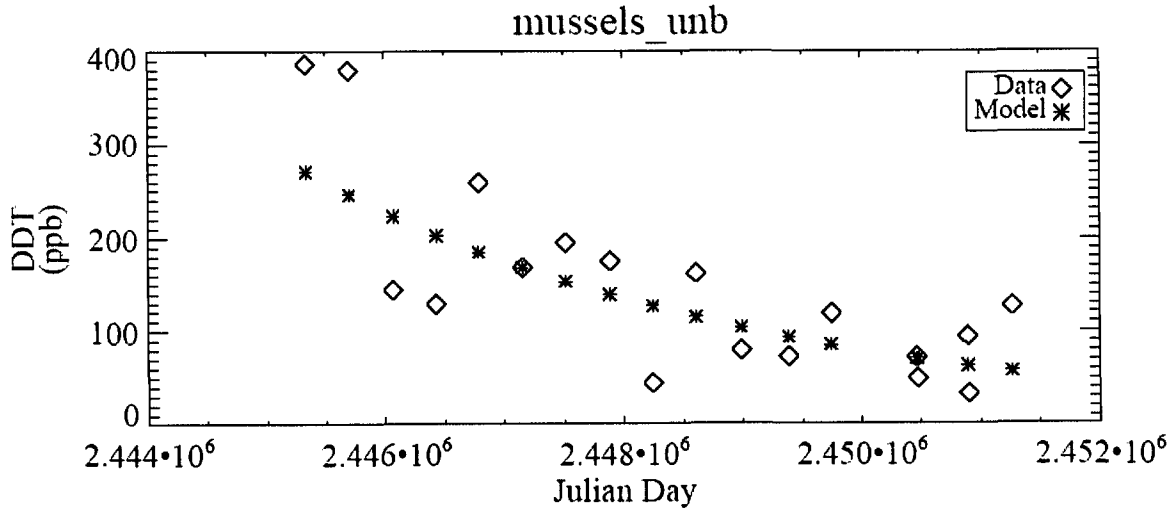
Intercept: 643.10577 Variance of Intercept: 21976.558

Slope: -0.00026070179 Variance of Slope: 3.6659074e-009

95% slope Confidence Interval
lower: -0.00038905516
upper: -0.00013234842

yearly rate (slope * 365) -0.095156153
yearly lower slope CI: -0.14200514
yearly upper slope CI: -0.048307172

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 29.159305 +/- 3.4570656
2008 estimate: 24.099740 +/- 3.6534045
2010 estimate: 19.918084 +/- 3.8773425



REGRESSION PARAMETERS FOR MUSSELS_LNB

N: 35
R: 0.71188219 R2: 0.50677626
P: 1.6249398e-006 F: 33.906755

Intercept: 1047.7585 Variance of Intercept: 32099.545

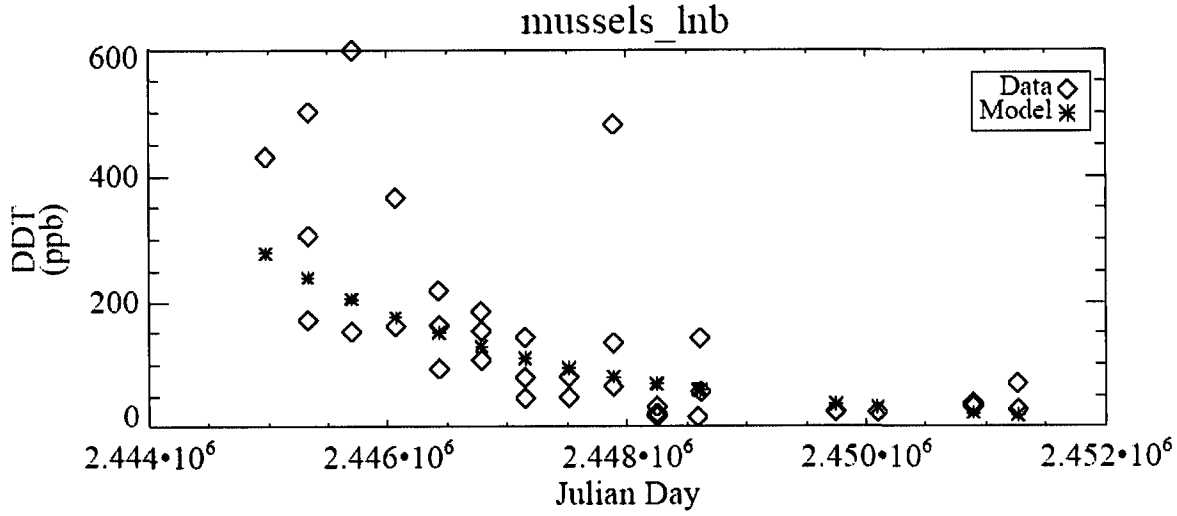
Slope: -0.00042623130 Variance of Slope: 5.3580215e-009

95% slope Confidence Interval
lower: -0.00057515488
upper: -0.00027730772

yearly rate (slope * 365) -0.15557442
yearly lower slope CI: -0.20993153
yearly upper slope CI: -0.10121732

DDT projected values (ppb) +/- 95% Prediction Interval

2006 estimate:	6.2626199 +/-	5.9840905
2008 estimate:	4.5860704 +/-	6.3476466
2010 estimate:	3.3583455 +/-	6.7628821



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REGRESSION PARAMETERS FOR
MUSSELS_EARLY_ALL_LOCATIONS_COMBINED

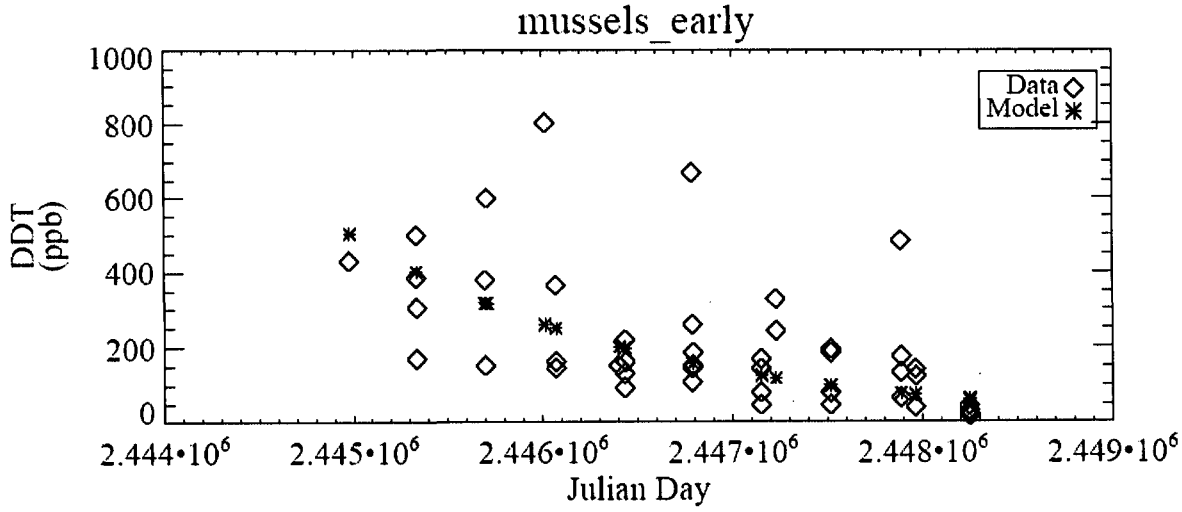
N: 45
R: 0.66357163 R2: 0.44032731
P: 6.7583844e-007 F: 33.830621

Intercept: 1586.7276 Variance of Intercept: 73953.934

Slope: -0.00064642800 Variance of Slope: 1.2351803e-008

95% slope Confidence Interval
lower: -0.00087056061
upper: -0.00042229539

yearly rate (slope * 365) -0.23594622
yearly lower slope CI: -0.31775462
yearly upper slope CI: -0.15413782



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$\partial c/\partial t$

REGRESSION PARAMETERS FOR
MUSSELS_LATE_ALL_LOCATIONS_COMBINED

N: 23
R: 0.048038496 R2: 0.0023076971
P: 0.82769246 F: 0.048573732

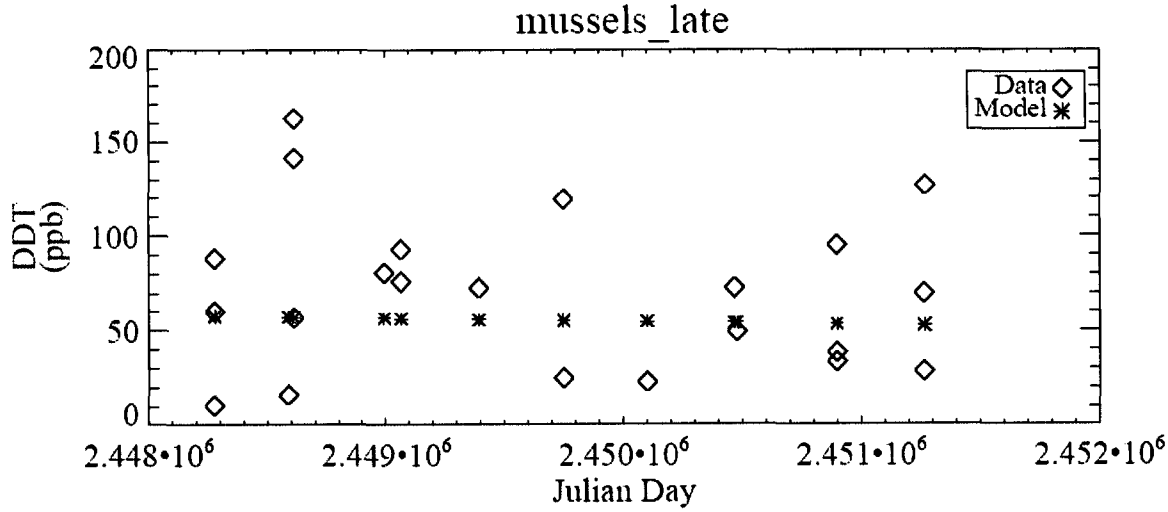
Intercept: 80.583787 Variance of Intercept: 120701.59

Slope: -3.1256447e-005 Variance of Slope: 2.0113041e-008

95% slope Confidence Interval
lower: -0.00032618834
upper: 0.00026367545

yearly rate (slope * 365) -0.011408603
yearly lower slope CI: -0.11905874
yearly upper slope CI: 0.096241538

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 48.616686 +/- 7.0701593
2008 estimate: 47.518463 +/- 8.1490779
2010 estimate: 46.445049 +/- 9.5033307



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REGRESSION PARAMETERS FOR MUSSELS_EARLY_SDC

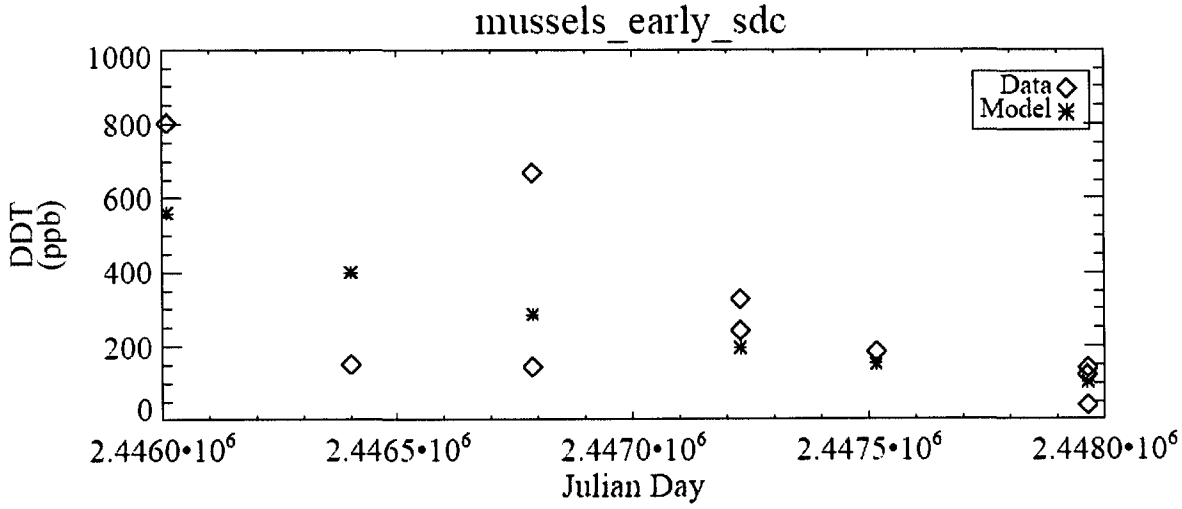
N: 10
R: 0.68301969 R2: 0.46651590
P: 0.029485943 F: 6.9957609

Intercept: 2125.1804 Variance of Intercept: 642365.85

Slope: -0.00086624707 Variance of Slope: 1.0726267e-007

95% slope Confidence Interval
lower: -0.0016214866
upper: -0.00011100752

yearly rate (slope * 365) -0.31618018
yearly lower slope CI: -0.59184261
yearly upper slope CI: -0.040517745



REGRESSION PARAMETERS FOR MUSSELS_EARLY_UNB

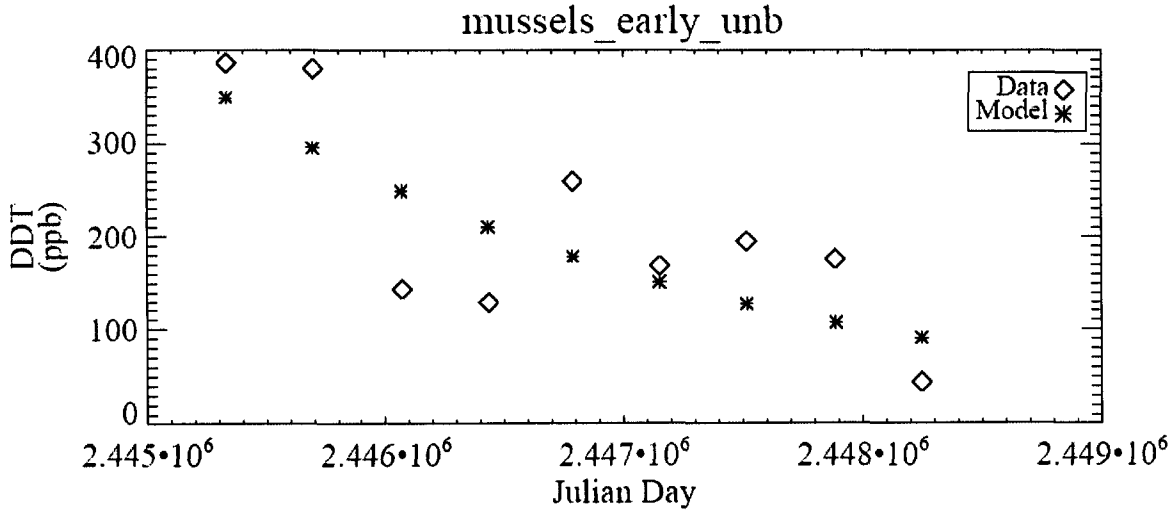
N: 9
R: 0.70662353 R2: 0.49931681
P: 0.033319829 F: 6.9808969

Intercept: 1135.3137 Variance of Intercept: 182955.57

Slope: -0.00046188248 Variance of Slope: 3.0559887e-008

95% slope Confidence Interval
lower: -0.00087525187
upper: -4.8513079e-005

yearly rate (slope * 365) -0.16858710
yearly lower slope CI: -0.31946693
yearly upper slope CI: -0.017707274



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REGRESSION PARAMETERS FOR MUSSELS_EARLY_LNB

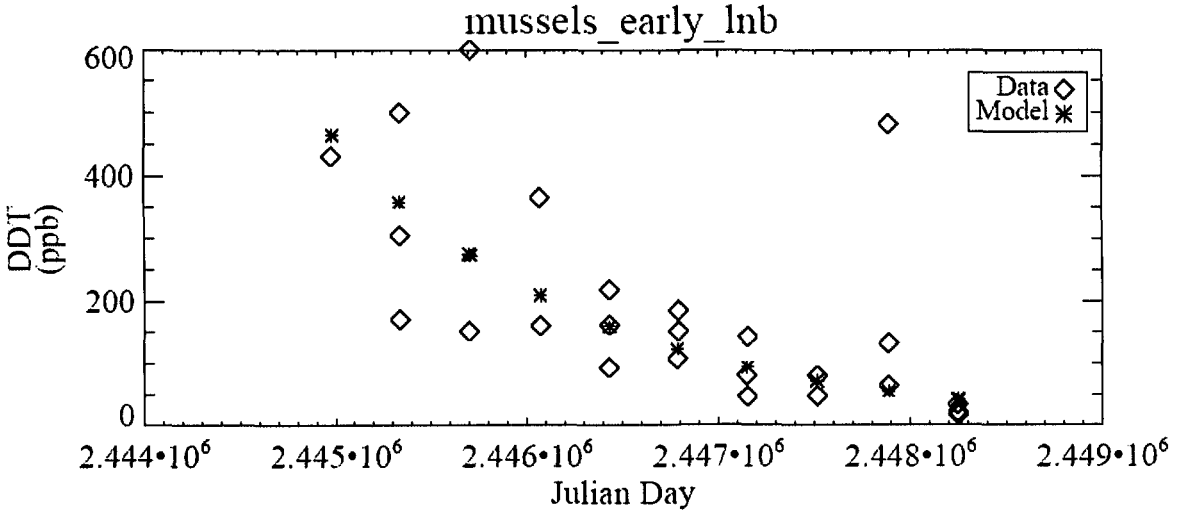
N: 26
R: 0.75458572 R2: 0.56939960
P: 8.4453139e-006 F: 31.736131

Intercept: 1802.5196 Variance of Intercept: 101834.32

Slope: -0.00073472024 Variance of Slope: 1.7009440e-008

95% slope Confidence Interval
lower: -0.0010038946
upper: -0.00046554593

yearly rate (slope * 365) -0.26817289
yearly lower slope CI: -0.36642151
yearly upper slope CI: -0.16992426



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REGRESSION PARAMETERS FOR MUSSELS_LATE_UNB

N: 9
R: 0.37566413 R2: 0.14112354
P: 0.31908627 F: 1.1501826

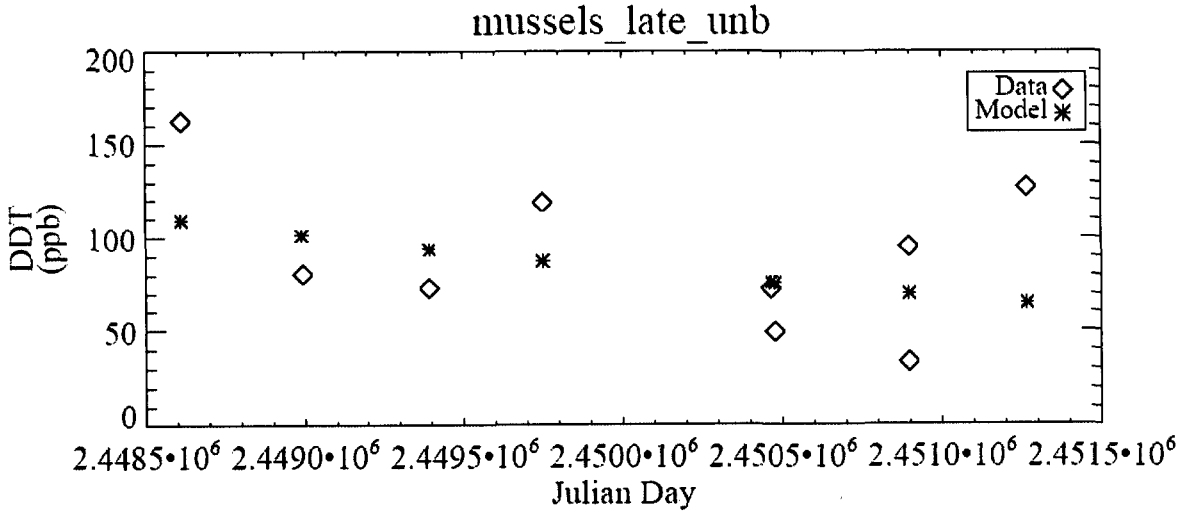
Intercept: 488.76926 Variance of Intercept: 203976.17

Slope: -0.00019769345 Variance of Slope: 3.3979559e-008

95% slope Confidence Interval
lower: -0.00063357784
upper: 0.00023819095

yearly rate (slope * 365) -0.072158108
yearly lower slope CI: -0.23125591
yearly upper slope CI: 0.086939695

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 38.539455 +/- 7.5273825
2008 estimate: 33.353697 +/- 9.8509241
2010 estimate: 28.865719 +/- 13.039222



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REGRESSION PARAMETERS FOR MUSSELS_LATE_LNB

N: 9
R: 0.14007425 R2: 0.019620794
P: 0.71926777 F: 0.14009432

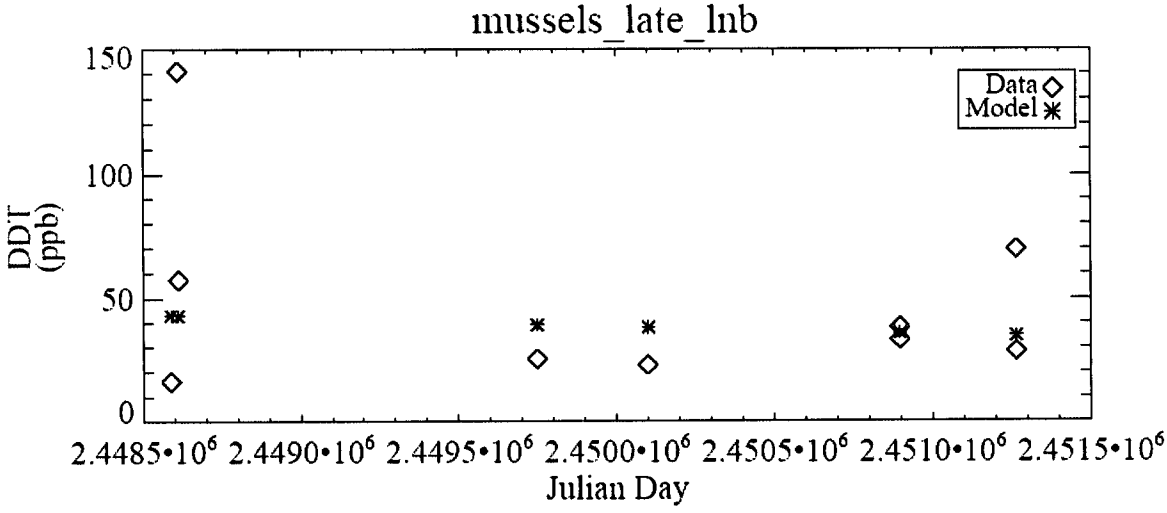
Intercept: 199.70530 Variance of Intercept: 274360.61

Slope: -8.0021229e-005 Variance of Slope: 4.5707756e-008

95% slope Confidence Interval
lower: -0.00058556341
upper: 0.00042552095

yearly rate (slope * 365) -0.029207749
yearly lower slope CI: -0.21373065
yearly upper slope CI: 0.15531515

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 28.280310 +/- 13.069423
2008 estimate: 26.673492 +/- 17.511763
2010 estimate: 25.157970 +/- 23.845064



Quantitative Environmental Analysis, LLC
$\partial c/\partial t$

REGRESSION PARAMETERS FOR SEDIMENTS_ALL_LOCATIONS_COMBINED

N: 151
R: 0.24313265 R2: 0.059113484
P: 0.0026287899 F: 9.3612874

Intercept: 206.60084 Variance of Intercept: 4409.5439

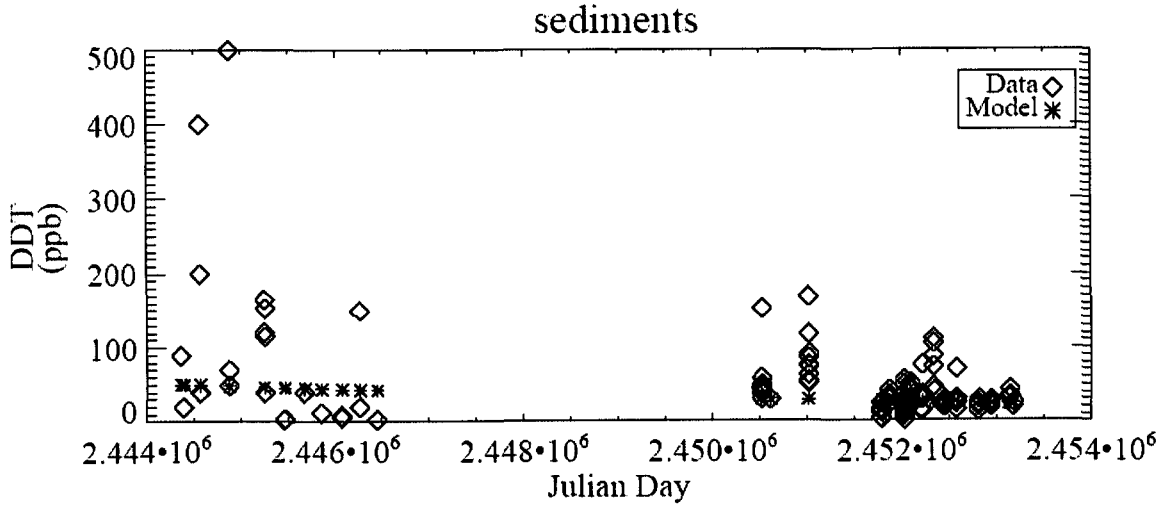
Slope: -8.2915892e-005 Variance of Slope: 7.3441236e-010

95% slope Confidence Interval
lower: -0.00013646592
upper: -2.9365868e-005

yearly rate (slope * 365) -0.030264301
yearly lower slope CI: -0.049810059
yearly upper slope CI: -0.010718542

DDT projected values (ppb) +/- 95% Prediction Interval

2006 estimate:	22.977861 +/-	7.2641894
2008 estimate:	21.626505 +/-	7.2942650
2010 estimate:	20.354625 +/-	7.3300398



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REGRESSION PARAMETERS FOR SEDIMENTS_UNB

N: 84
R: 0.17243035 R2: 0.029732226
P: 0.11677773 F: 2.5127522

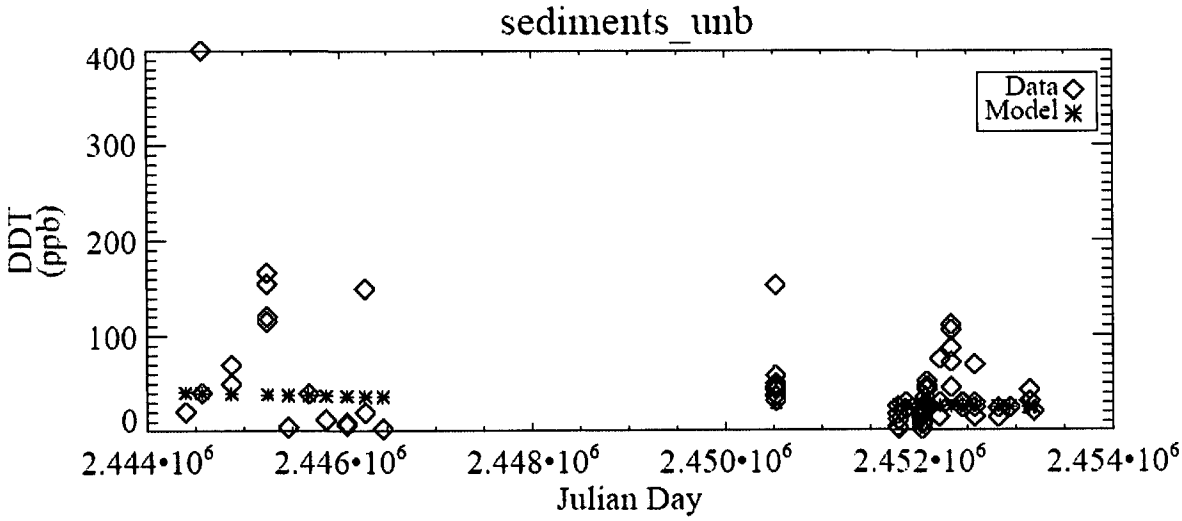
Intercept: 145.41857 Variance of Intercept: 8030.3591

Slope: -5.7974081e-005 Variance of Slope: 1.3375748e-009

95% slope Confidence Interval
lower: -0.00013072921
upper: 1.4781053e-005

yearly rate (slope * 365) -0.021160540
yearly lower slope CI: -0.047716163
yearly upper slope CI: 0.0053950844

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 23.492230 +/- 7.6289685
2008 estimate: 22.517453 +/- 7.6874308
2010 estimate: 21.583122 +/- 7.7568269



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∂c/∂t

REGRESSION PARAMETERS FOR SEDIMENTS_LNB

N: 67
R: 0.33988221 R2: 0.11551992
P: 0.0048929630 F: 8.4895009

Intercept: 291.74883 Variance of Intercept: 9786.8013

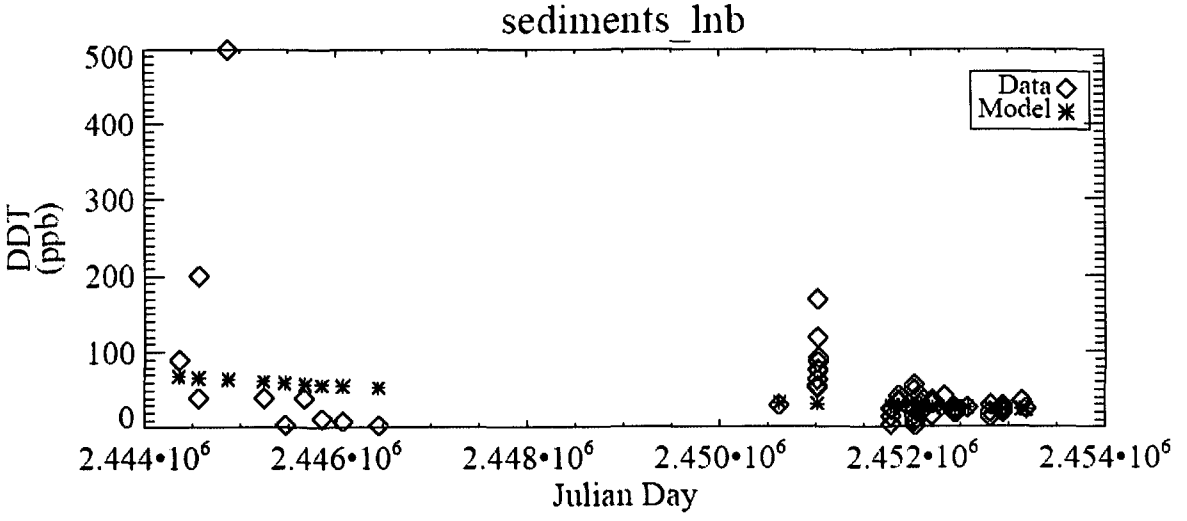
Slope: -0.00011762823 Variance of Slope: 1.6298249e-009

95% slope Confidence Interval
lower: -0.00019825495
upper: -3.7001502e-005

yearly rate (slope * 365) -0.042934303
yearly lower slope CI: -0.072363057
yearly upper slope CI: -0.013505548

DDT projected values (ppb) +/- 95% Prediction Interval

2006 estimate:	22.249806 +/-	7.2250941
2008 estimate:	20.416574 +/-	7.2909092
2010 estimate:	18.734389 +/-	7.3698321



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(512) 707-0090
(512) 275-0915 *fax*

REGRESSION PARAMETERS FOR SEDIMENTS_EARLY_ALL_LOCATIONS_COMBINED

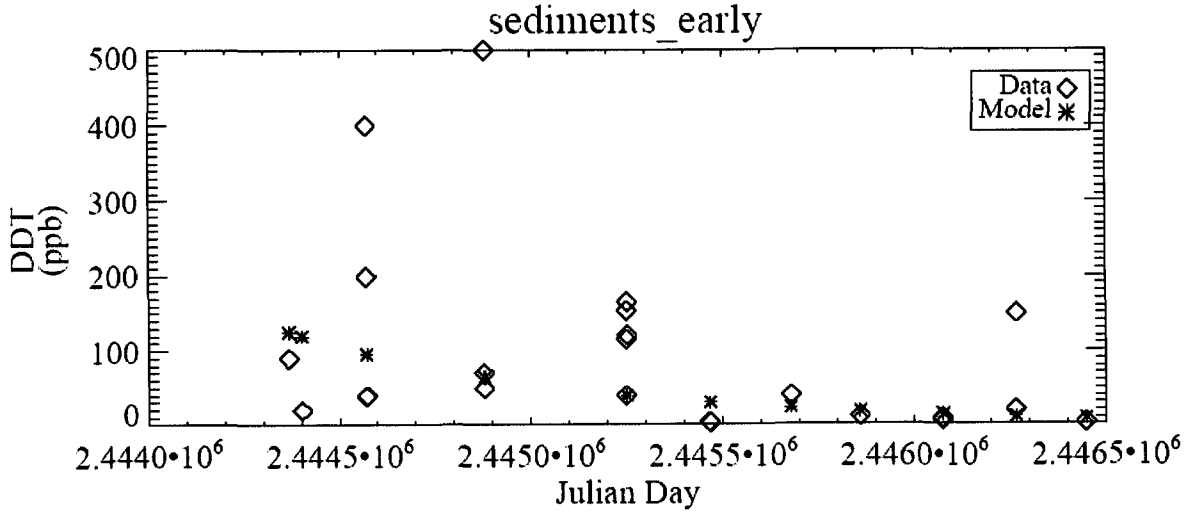
N: 37
R: 0.58720700 R2: 0.34481206
P: 0.00013314710 F: 18.419787

Intercept: 3174.3451 Variance of Intercept: 545765.28

Slope: -0.0012966600 Variance of Slope: 9.1278319e-008

95% slope Confidence Interval
lower: -0.0019100027
upper: -0.00068331731

yearly rate (slope * 365) -0.47328091
yearly lower slope CI: -0.69715100
yearly upper slope CI: 0.24941082



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$\partial c/\partial t$

REGRESSION PARAMETERS FOR SEDIMENTS_LATE_ALL_LOCATIONS_COMBINED

N: 114
R: 0.27399717 R2: 0.075074450
P: 0.0031785628 F: 9.0908272

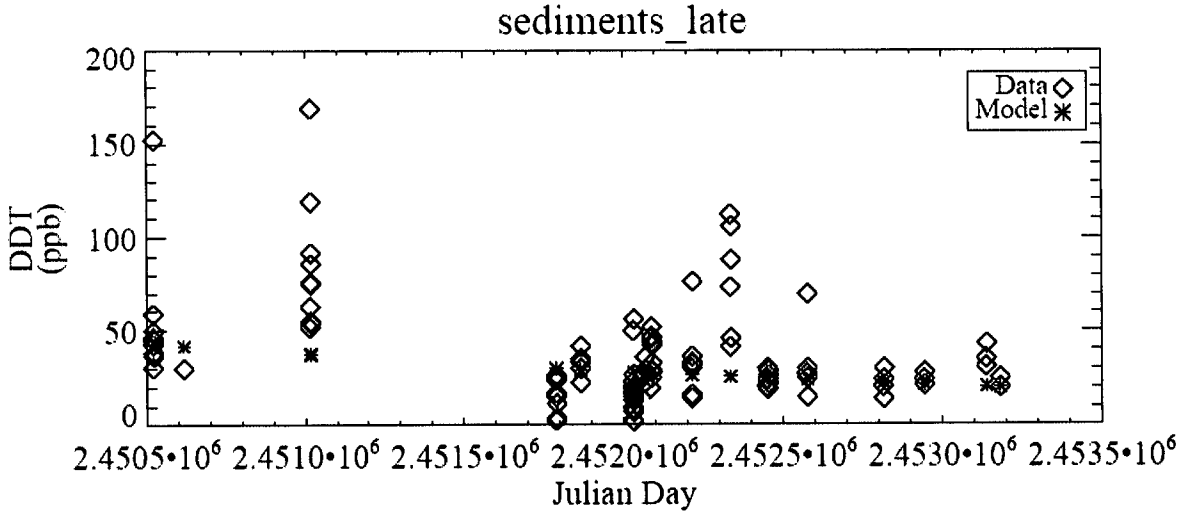
Intercept: 722.60987 Variance of Intercept: 56909.732

Slope: -0.00029334206 Variance of Slope: 9.4655371e-009

95% slope Confidence Interval
lower: -0.00048611176
upper: -0.00010057235

yearly rate (slope * 365) -0.10706985
yearly lower slope CI: -0.17743079
yearly upper slope CI: -0.036708908

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 16.140316 +/- 4.5968579
2008 estimate: 13.025216 +/- 4.7825499
2010 estimate: 10.511334 +/- 5.0325779



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REGRESSION PARAMETERS FOR SEDIMENTS_EARLY_UNB

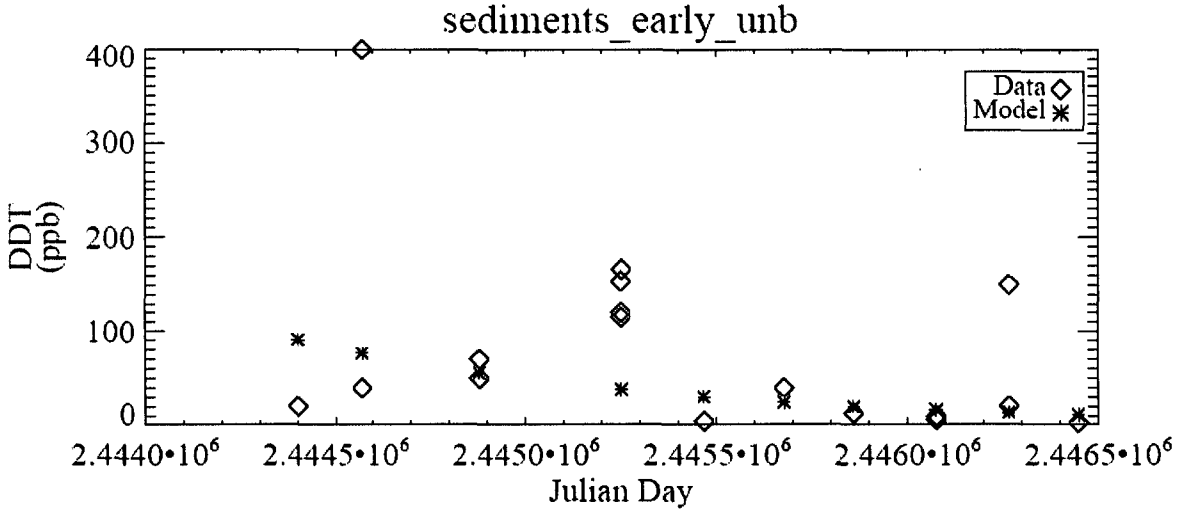
N: 22
R: 0.49779948 R2: 0.24780432
P: 0.018396539 F: 6.5888261

Intercept: 2536.2016 Variance of Intercept: 973512.66

Slope: -0.0010357102 Variance of Slope: 1.6280529e-007

95% slope Confidence Interval
lower: -0.0018773791
upper: -0.00019404140

yearly rate (slope * 365) -0.37803424
yearly lower slope CI: -0.68524337
yearly upper slope CI: -0.070825113



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REGRESSION PARAMETERS FOR SEDIMENTS_EARLY_LNB

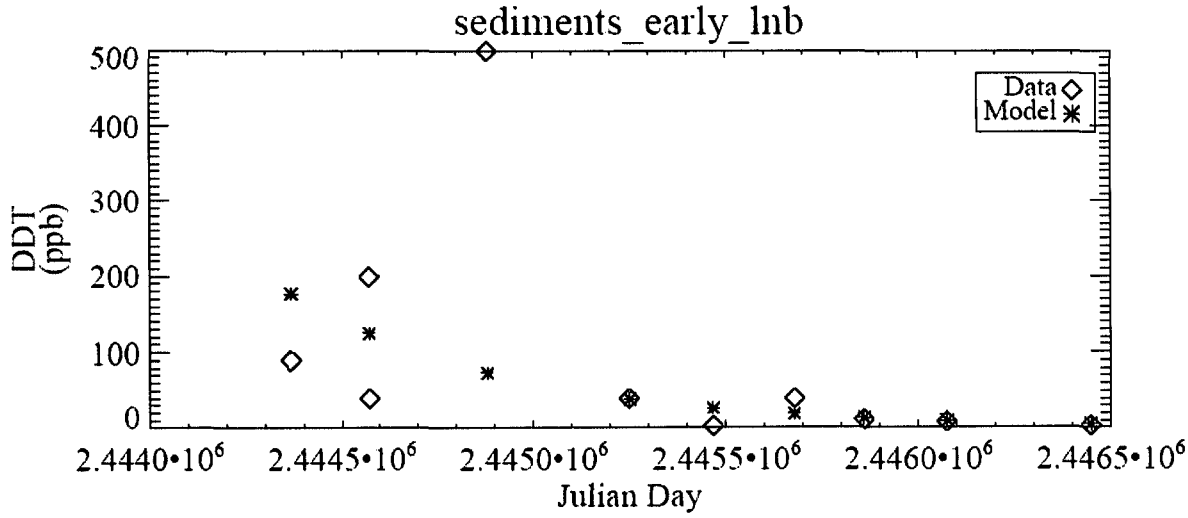
N: 15
R: 0.69730979 R2: 0.48624095
P: 0.0038569680 F: 12.303690

Intercept: 4166.3236 Variance of Intercept: 1408137.8

Slope: -0.0017023415 Variance of Slope: 2.3553638e-007

95% slope Confidence Interval
lower: -0.0027508142
upper: -0.00065386887

yearly rate (slope * 365) -0.62135466
yearly lower slope CI: -1.0040472
yearly upper slope CI: -0.23866214



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$\partial c/\partial t$

REGRESSION PARAMETERS FOR SEDIMENTS_LATE_UNB

N: 62
R: 0.16302514 R2: 0.026577195
P: 0.20550351 F: 1.6381697

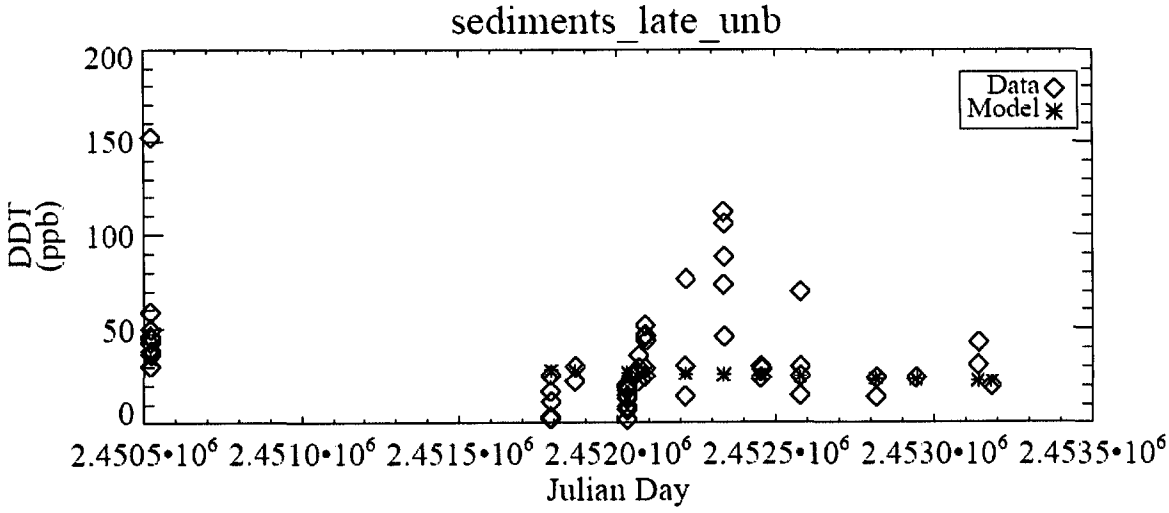
Intercept: 416.49726 Variance of Intercept: 104219.91

Slope: -0.00016851471 Variance of Slope: 1.7334716e-008

95% slope Confidence Interval
lower: -0.00043187684
upper: 9.4847426e-005

yearly rate (slope * 365) -0.061507868
yearly lower slope CI: -0.15763505
yearly upper slope CI: 0.034619311

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 19.708401 +/- 5.3213314
2008 estimate: 17.424212 +/- 5.6933763
2010 estimate: 15.404759 +/- 6.2014263



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$\partial c/\partial t$

REGRESSION PARAMETERS FOR SEDIMENTS_LATE_LNB

N: 52
R: 0.44624360 R2: 0.19913335
P: 0.00091376518 F: 12.432367

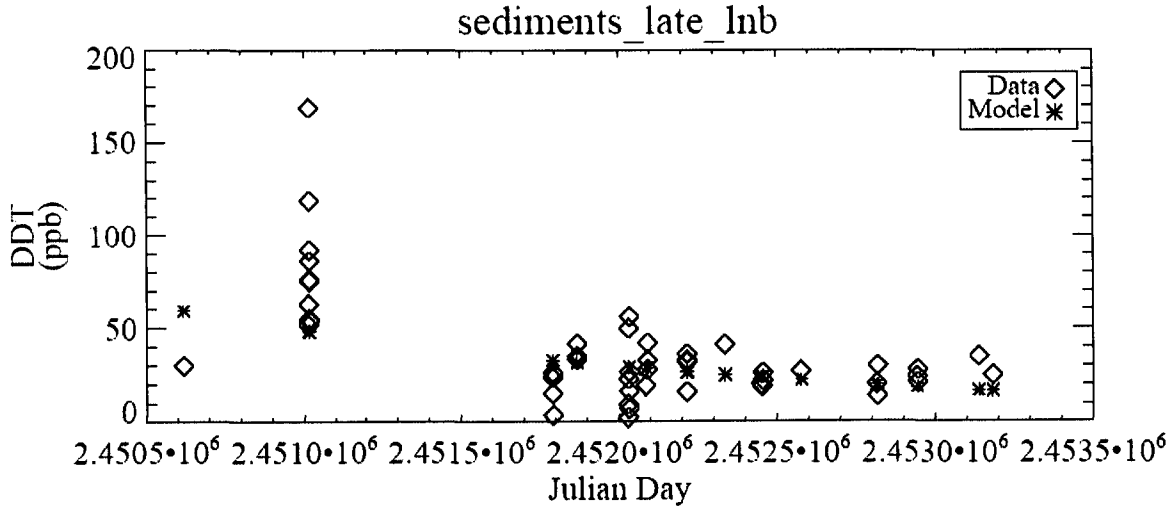
Intercept: 1236.6075 Variance of Intercept: 122331.20

Slope: -0.00050294502 Variance of Slope: 2.0346383e-008

95% slope Confidence Interval
lower: -0.00078944738
upper: -0.00021644266

yearly rate (slope * 365) -0.18357493
yearly lower slope CI: -0.28814829
yearly upper slope CI: -0.079001572

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 11.435676 +/- 4.2869450
2008 estimate: 7.9175762 +/- 4.6865237
2010 estimate: 5.4817934 +/- 5.2364700



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$\partial c/\partial t$

REGRESSION PARAMETERS FOR SEDIMENTS_UNB_97RM

N: 51
R: 0.29530450 R2: 0.087204750
P: 0.035397430 F: 4.6812609

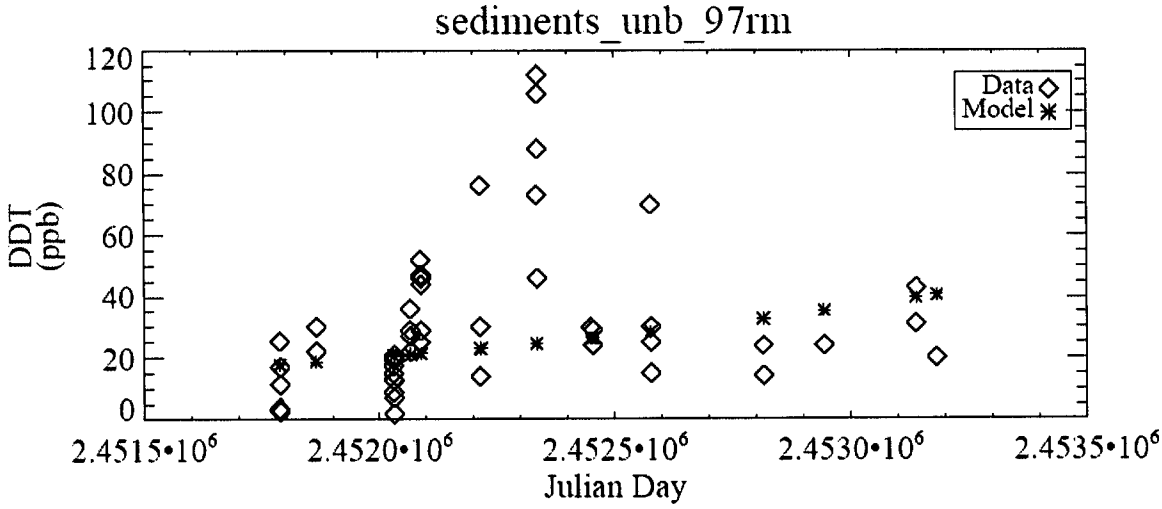
Intercept: -1446.4509 Variance of Intercept: 448902.59

Slope: 0.00059113250 Variance of Slope: 7.4646050e-008

95% slope Confidence Interval
lower: 4.2087725e-005
upper: 0.0011401773

yearly rate (slope * 365) 0.21576336
yearly lower slope CI: 0.015362020
yearly upper slope CI: 0.41616471

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 61.688310 +/- 6.0732172
2008 estimate: 95.031869 +/- 7.6074543
2010 estimate: 146.39818 +/- 10.002129



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REGRESSION PARAMETERS FOR SEDIMENTS_LNB_98RM

N: 42
R: 0.091793539 R2: 0.0084260539
P: 0.56315608 F: 0.33990622

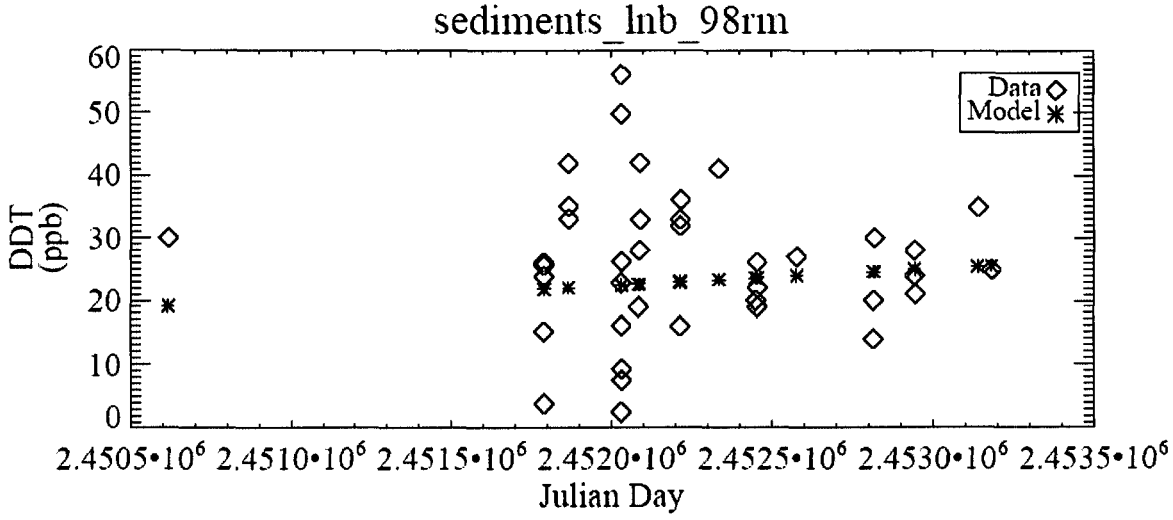
Intercept: -275.97475 Variance of Intercept: 229192.45

Slope: 0.00011381833 Variance of Slope: 3.8112309e-008

95% slope Confidence Interval
lower: -0.00028074387
upper: 0.00050838052

yearly rate (slope * 365) 0.041543689
yearly lower slope CI: -0.10247151
yearly upper slope CI: 0.18555889

DDT projected values (ppb) +/- 95% Prediction Interval
2006 estimate: 27.734278 +/- 4.0084578
2008 estimate: 30.140516 +/- 4.6775011
2010 estimate: 32.755521 +/- 5.6505153



5.1.2 Regression 70-Year Predicted Values

Predicted Values and 95% Prediction Intervals for Newport Bay DDT levels in Red Shiners and Mussels.

Values are predicted at 5 year intervals for a 70-year period: 2006 through 2076

RED_SHINERS

DDT projected values (ppb) +/- 95% Prediction Interval

2006 estimate:	83.436119 +/-	3.6293264
2011 estimate:	34.992822 +/-	3.8755604
2016 estimate:	14.675869 +/-	4.2082663
2021 estimate:	6.1550090 +/-	4.6340781
2026 estimate:	2.5813896 +/-	5.1622921
2031 estimate:	1.0826259 +/-	5.8052022
2036 estimate:	0.45404960 +/-	6.5784337
2041 estimate:	0.19042684 +/-	7.5013290
2046 estimate:	0.079864362 +/-	8.5974256
2051 estimate:	0.033494839 +/-	9.8950489
2056 estimate:	0.014047620 +/-	11.428042
2061 estimate:	0.0058915236 +/-	13.236660
2066 estimate:	0.0024708847 +/-	15.368653
2071 estimate:	0.0010362806 +/-	17.880538
2076 estimate:	0.00043461251 +/-	20.839156

RED_SHINERS OUTLIERS REMOVED

DDT projected values (ppb) +/- 95% Prediction Interval

2006 estimate:	75.447468 +/-	2.8758861
2011 estimate:	30.256323 +/-	3.0439619
2016 estimate:	12.133543 +/-	3.2679668
2021 estimate:	4.8658543 +/-	3.5501799
2026 estimate:	1.9513293 +/-	3.8942131
2031 estimate:	0.78253190 +/-	4.3051520
2036 estimate:	0.31381487 +/-	4.7896515
2041 estimate:	0.12584762 +/-	5.3560236
2046 estimate:	0.050468044 +/-	6.0143524
2051 estimate:	0.020238949 +/-	6.7766425
2056 estimate:	0.0081163250 +/-	7.6570152
2061 estimate:	0.0032548494 +/-	8.6719506
2066 estimate:	0.0013052761 +/-	9.8405903
2071 estimate:	0.00052344837 +/-	11.185091
2076 estimate:	0.00020991590 +/-	12.731048

MUSSELS

DDT projected values (ppb) +/- 95% Prediction Interval

2006 estimate:	11.702715 +/-	5.3299930
2011 estimate:	6.0220453 +/-	5.8421251
2016 estimate:	3.0988561 +/-	6.5177788
2021 estimate:	1.5946258 +/-	7.3799186
2026 estimate:	0.82057105 +/-	8.4587515

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2031 estimate:	0.42225381 +/-	9.7929352
2036 estimate:	0.21728561 +/-	11.431069
2041 estimate:	0.11181199 +/-	13.433561
2046 estimate:	0.057536807 +/-	15.874960
2051 estimate:	0.029607596 +/-	18.846858
2056 estimate:	0.015235634 +/-	22.461487
2061 estimate:	0.0078400337 +/-	26.856142
2066 estimate:	0.0040343662 +/-	32.198624
2071 estimate:	0.0020760256 +/-	38.693910
2076 estimate:	0.0010682923 +/-	46.592324

MUSSELS_UNB

DDT projected values (ppb) +/- 95% Prediction Interval

2006 estimate:	29.128913 +/-	3.4580671
2011 estimate:	18.095980 +/-	4.0009676
2016 estimate:	11.241905 +/-	4.7311915
2021 estimate:	6.9838955 +/-	5.6821454
2026 estimate:	4.3386593 +/-	6.9004350
2031 estimate:	2.6953388 +/-	8.4478634
2036 estimate:	1.6744461 +/-	10.404359
2041 estimate:	1.0402291 +/-	12.872015
2046 estimate:	0.64622956 +/-	15.980429
2051 estimate:	0.40146218 +/-	19.893591
2056 estimate:	0.24940345 +/-	24.818688
2061 estimate:	0.15493883 +/-	31.017269
2066 estimate:	0.096253845 +/-	38.819383
2071 estimate:	0.059796519 +/-	48.641456
2076 estimate:	0.037147853 +/-	61.008864

Appendix B: Dr. James L. Byard, "Scientific Commentary on the U.S. EPA Water Criterion for DDT to Protect Wildlife; Protecting the Brown Pelican:"

SCIENTIFIC COMMENTARY ON THE U. S. EPA WATER CRITERION FOR DDT TO PROTECT WILDLIFE; PROTECTING THE BROWN PELICAN

James L. Byard, Ph.D., D.A.B.T.

September 15, 2006

SUMMARY

The National criterion and State CTR standard for DDT in the water column is based on a fish residue of 150 ppb in northern anchovies and reduced hatching success in a sensitive avian species, the brown pelican. Following reductions in releases from a DDT manufacturing plant, residues in northern anchovies fell 27-fold to 150 ppb; residues in brown pelican eggs fell 9-fold during the same period. If the egg residues had declined 27-fold as would be expected, in time, in the long-lived brown pelican, the egg residue level would reach 1.7 ppm. This egg residue level is below the NOEL (no-observable-effect-level) for reduced hatching success. Assuming a 27-fold reduction in egg residues, the CTR of 1 pptr in water and 150 ppb in fish is based on a NOEL and not a LOEL (low-observable-effect-level). The NOEL for reduced hatching success was asertained from a literatue review of the effects of DDT on reproduction in brown pelicans. Reproduction was inhibited in populations in Louisiana, Florida, South Carolina, Texas and California during the DDT use era. Since the ban in 1972, residue levels have declined, eggshells have become thicker, and reproduction has slowly returned to normal. By the mid 1990s, reproduction in all populations of brown pelicans in the United States was no longer inhibited by DDE. Residues in eggs are below the 2.5 ppm level associated with reduced hatching success. The recovery of brown pelican breeding on Anacapa Island represents a worst case because of the very high concentrations of DDE in sediments and fish on the nearby Palos Verdes Shelf. The much higher level of DDE from manufacturing wastes on the Palos Verdes Shelf along with the recovery of the nearby Anacapa colony suggests that lower residues from agricultural uses should have no measurable effect on reproduction in brown pelicans.

INTRODUCTION

In 1980, the U. S. EPA published criteria for the protection of wildlife from DDT in the water column. The criterion was adopted as the California Toxics Rule (CTR) standard in 2002. The wildlife criterion of 1 pptr was based on the bioaccumulation of DDT from water into fish.

A fish target residue was chosen to be 150 ppb from a study by Anderson et al. (1975) in a recovering population of brown pelicans. Monitoring pptr levels of DDT in water is difficult and uncertain, limiting the utility of the criterion. Measuring levels of DDT in fish is much easier and more certain. Therefore, a criterion in fish is more useful than one in water. This brings us to the question of whether the 150 ppb DDT residue level in fish, that is the basis for the National criterion and CTR standard in water, will protect wildlife, considering what is known today. To remain consistent with the criterion, the fish residue should protect the brown pelican, one of the most sensitive species to the reproductive effects of DDT. This question is addressed herein by reviewing studies of the effects of DDT on reproduction in brown pelicans.

U. S. EPA CRITERION

The EPA 1980 criterion follows:

A residue value for wildlife protection of 0.0010 µg/l is obtained for both freshwater and saltwater using the lowest maximum permissible tissue concentration of 0.15 mg/kg based on reduced productivity of the brown pelican (Anderson, et al. 1975). Average lipid content of pelican diets is unavailable. Clupeids usually constitute the major prey of pelicans, and the percent lipid value of the clupeid, northern anchovy, is 8 (Reintjes, 1980). The northern anchovy is in some areas a major food source of the brown pelican. Therefore, the percent lipid value of 8 was used for the calculation of the Final Residue Value. The value of 0.15 mg/kg divided by the geometric mean of normalized BCF values (17,870) and by a percent lipid value of 8 gives a residue value of 0.0010 µg/l (Table 5).

Selection of the lowest freshwater and saltwater residue values from the above calculations gives a Freshwater Final Residue Value of 0.0010 µg/l and a Saltwater Final Residue Value of 0.0010 ug/l. The Final Residue Values may be too high because they are based on a concentration which reduced the productivity of the brown pelican.

The particular pelicans studied by Anderson et al. (1975) were reported to be feeding on northern anchovies. The northern anchovy diet of the recovering population of brown pelicans became the basis of the EPA chronic criterion to protect wildlife. The fish residue of 150 ppb is based on a study where the population of brown pelicans were still recovering. The level of reproduction was judged to be inadequate to sustain the population. However, the authors refered to a slow response of DDE residues in eggs compared to the fish diet. The fish residue had declined 27-fold during a period in which the egg residues had declined only 9-fold. Also, DDT and DDE were detected in fish in 1974, but only the more stable DDE was detected in brown pelican eggs that year. Therefore, DDE in brown pelicans and in their eggs appears to have not reached a steady-state with the more rapidly declining residues in the aquatic environment. If we assume that in time the DDE residue in the eggs would also decline 27-fold, the final egg residue would have a geometric mean of 1.7 ppm. Would this level be a no-effect level for reproductive effects in the brown pelican?

To answer this question, let us review in chronological order the studies of the effects of DDT on various populations of brown pelicans during and after the DDT era. The recovery of brown pelicans following the ban of DDT in 1972 provides a measure of dose-response and thesholds for the reproductive effects of DDT.

CHRONOLOGY OF BROWN PELICAN STUDIES

Risebrough et al. (1967) reported the accumulation of DDT in higher trophic levels along the California coast. "Fish from California coastal waters contained more residue, but in general total concentrations were 10-20 per cent of those in the birds." Bird species included Cassin's auklet, western gull, pelagic cormorant, Brandt's cormorant, brown pelican, common murre, ancient, murrelet, red phalarope, rhinoceros auklet, sooty shearwater and slender-billed shearwater. Whole bird tissue ranged from 1.0 to 15.4 ppm. Western gull and Cassin's auklet eggs contained 6.5 and 10.8 ppm, respectively. Fish included northern anchovy, English sole, Pacific jack mackerel, and hake. DDT levels in fish ranged from 0.2 to 2.8 ppm, with one sample of northern anchovy taken off Terminal Island, Los Angeles at 12.7 ppm DDT

In a 1969 conference at Oregon State University, James Keith (Keith, 1969) stated that scientists now have data to show that DDT is causing eggshell thinning in birds. Pelicans on Anacapa Island off the southern California coast produced good numbers of young in 1962, 1963 and 1966. In 1968 they were clearly in trouble, and in 1969 their reproductive effort was for all practical purposes a complete failure. In the same conference, Robert Risebrough (Terriere et al., 1969) stated in a panel discussion that DDT levels in northern anchovies were low around San Francisco Bay compared to 5-15 ppm in waters off southern California. "We are aware of certain massive "hot spots": Clear Lake, California, Lake Michigan and evidently the Southern California coast." DDT stored in fat is toxicologically inert unless mobilized due to mobilization of fat stores. In a separate paper at the conference, Risebrough, et al spoke of recent findings.

p,p'-DDE is the major cause of eggshell thinning in raptorial and fish-eating birds (Risebrough et al., 1969). The peregrine falcon, bald eagle and osprey are in decline due to DDE eggshell thinning. There is no evidence of thinning in eggshells of species that prey mostly on mammals, such as the Red-tailed hawk, golden eagle and great horned owl. Brown pelicans have declined 50 % in the past four years at Point Reyes. Brown pelican and double-crested cormorant reproduction on the Channel Islands and Islas Coronados near San Diego were decimated in 1969. Western gull eggs on Anacapa Island in 1969 were normal. Some eggshell thinning is evident in ashy petrel and murre from the Farallon Islands. A "No effect" level has not been established for eggshell thinning. The relationship between DDE residues and eggshell thinning is linear with an absence of a "no effect" range of concentrations. DDE plus DDD in eggs from white pelicans, at levels ranging from less than 0.5 ppm to 6 ppm, were associated with significant eggshell thinning. "The complex series of behavioral events that lead up to mating, next building, and egglaying were evidently not adversely affected." The likely mechanism of action is inhibition of calcium transport and mineralization in the shell gland. In the brown pelican, eggshell thickness is reduced about 15 % at 75 ppm DDE on a lipid basis (3.3 ppm fresh weight). At higher residue levels the slope of the residue-thinning curve decreases to zero thickness at 3,000 ppm DDE (132 ppm fresh weight).

Keith et al. (1970) also studied the brown pelicans on the Channel Islands. Brown pelican eggshells from Anacapa Island were 34 % thinner than pre-DDT era controls. DDE residues in the eggs were 29 to 183 ppm. DDE in brain tissue was high but not as high as the 30-60 ppm considered lethal.

Blus (1970) reported a study of eggshell thinning and breeding success in brown pelicans in Florida and South Carolina. Populations in both states were declining. Eggshells were 6-16 per cent thinner than pre-DDT eggshells. Brown pelicans have been extirpated in Louisiana and other Gulf Coast localities. The reproductive failure and population declines were attributed to eggshell thinning caused by DDE.

Risebrough et al. (1971) reported an account of almost complete reproductive failure of brown pelicans on the Channel Islands in 1969. Broken and crushed eggs were strewn about the breeding area. Eggshell thickness was reduced 50 %. Only 2 young were observed out of 1,272 nests.

A statistical analysis of the variability in eggshell thinning in brown pelicans implicated DDE as the causative organochlorine (Blus et al., 1971). Ten eggs from California contained DDE residues as high as 135 ppm with shell thinning of 25 to 35 %. DDE residues in eggs from 9 colonies in Florida ranged from 0.2 to 6.0 ppm. Eggs from 2 colonies in South Carolina had DDE residues ranging from 3.3 to 10.6 ppm. Blus et al. reported in 1972 that eggshell thinning of 15-20 % has been associated with declining populations of several species of birds. The dose-response of DDE residue in eggs and eggshell thinning in brown pelicans was log-linear. The estimated no-effect level was 0.5 ppm. The brown pelican is unusually sensitive to eggshell thinning by DDE. Fifteen per cent thinning occurs at 4-5 ppm DDE in eggs. The herring gull showed no thinning when DDE residues in eggs were 4-5 ppm. The level of DDE in eggs is taken as an indication of DDE residues in the female. Figure 1 below is reproduced from Blus et al. (1972).

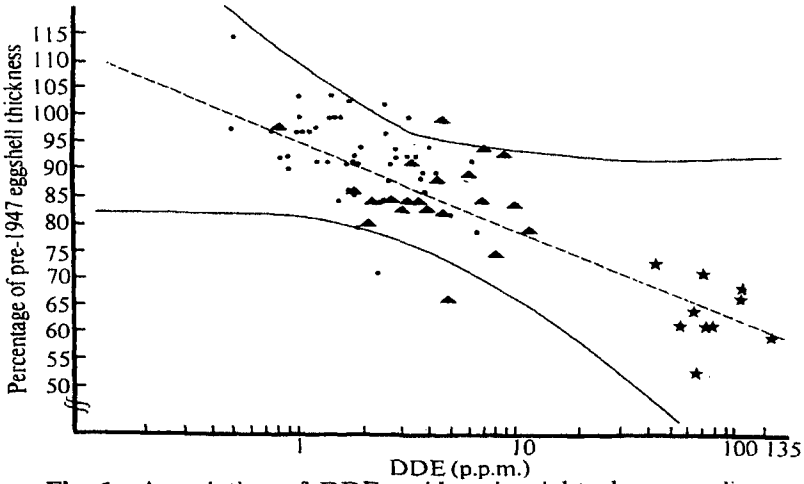

Fig. 1 Association of DDE residues in eighty brown pelican eggs from Florida (●), South Carolina (▲) and California (★) with the % of pre-1947 eggshell thickness. Solid lines represent 95% confidence limits. $\hat{Y}=95.787-15.689\ \log_{10} X$; $r=-0.80$ ($P<0.01$).

The paper by Blus et al. (1972a) in *Nature* was accompanied by a letter from William Hazeltine challenging the assertion that the DDE – eggshell thinning dose-response was log-linear. Moreover, Hazeltine questioned whether DDE causes eggshell thinning. He suggests scientists are acting irresponsibly to ban pesticides.

Risebrough (1972) also wrote a letter to *Nature*. His letter defended Blus et al. and refuted Hazeltine's comments. He states that in some cases the log-normal distribution provides an excellent fit to the brown pelican data, and: "In several other cases the gamma distribution more adequately describes the observed distribution of pollutants."

Switzer et al. (1972) also wrote a letter to *Nature* challenging Blus et al.'s conclusion that eggshell thinning in the brown pelican was caused by DDE. They pointed out that museum eggs, used to establish pre-DDT era shell thickness, were often selected as the best (and perhaps thickest) specimens for display in public exhibits.

Blus et al. (1972b) responded to comments by Hazeltine and by Switzer, et al in a follow-up report in *Nature*. They point out that lipid levels in eggs decrease about one-third from laying to hatching. Since, DDE residues are localized in the lipid, the lipid concentration of DDE will increase during incubation.

Schreiber and Risebrough (1972) published a review of the status of the brown pelican in the United States and Baja, Mexico. They also reported on Schreiber's work on brown pelicans in Florida. Hatching success in Florida decreased sharply with increasing frequency of inspection by wildlife biologists. The lipid content of Florida eggs was 5.0 %. The authors claimed that very low concentrations of DDE were associated with significant thinning and that the relationship is linear from zero concentrations of DDE. Thinning of eggshells greater than 20

% usually causes them to break during incubation. Total DDT residues in eggs collected in 1969 and 1970 in Florida were 1.2 to 2.9 ppm The 9 % reduction in eggshell thickness in Florida had not yet had an observable effect on population stability. There was no evidence that 9 % shell thinning has an effect on gas exchange or water retention.

Keith and Gruchy (1972) published a comprehensive review of the past five years of reports on the effects of DDE on avian wildlife. They noted a wide species variation in eggshell thinning response to DDE residues as illustrated in their Figure 7 below.

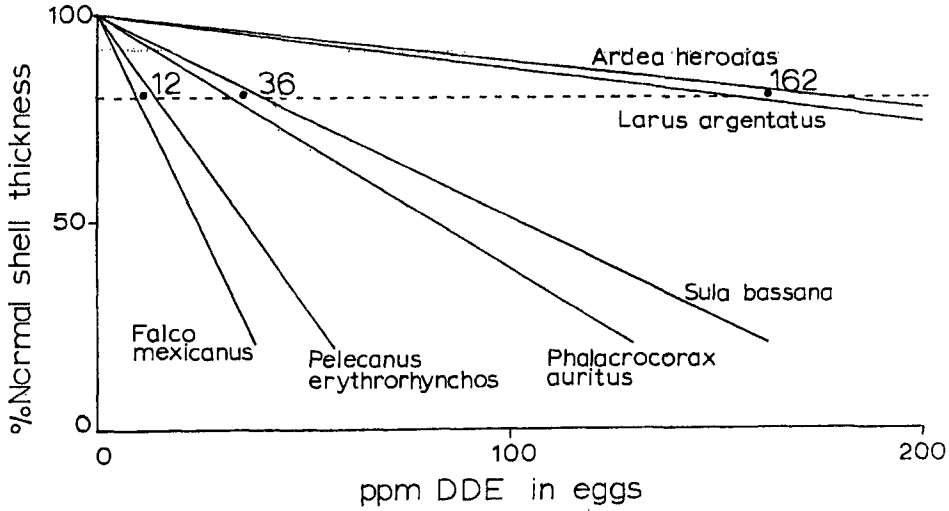

FIGURE 7. Variation between species in reproductive response to DDE. DDE values are of whole eggs on a wet-weight basis. Twenty-percent reduction in shell thickness (population damage threshold) is shown by a dotted line, and the calculated DDE values at that thickness are shown as 12, 36, and 162 ppm for the three pairs of slopes. Sources are *Pelecanus erythrorhynchos* and *Phalacrocorax auritus*, [1]; *Falco mexicanus*, [11]; *Larus argentatus*, [14]; and *Sula bassana*, unpublished data of J. A. KEITH.

Jehl (1973) reported on the status of brown pelicans on islands off the west coast of Baja, California. Breeding was severly impacted at most of the locations, with empty nests and broken shells. Observations were complicated by destruction of pelican eggs by gulls whenever nests were unattended. The source of DDE was attributed to the Los Angeles outfall. The dose-response for DDE in eggs and shell thinning is shown below in Figure 3 (DDE concentration in ppm lipid).

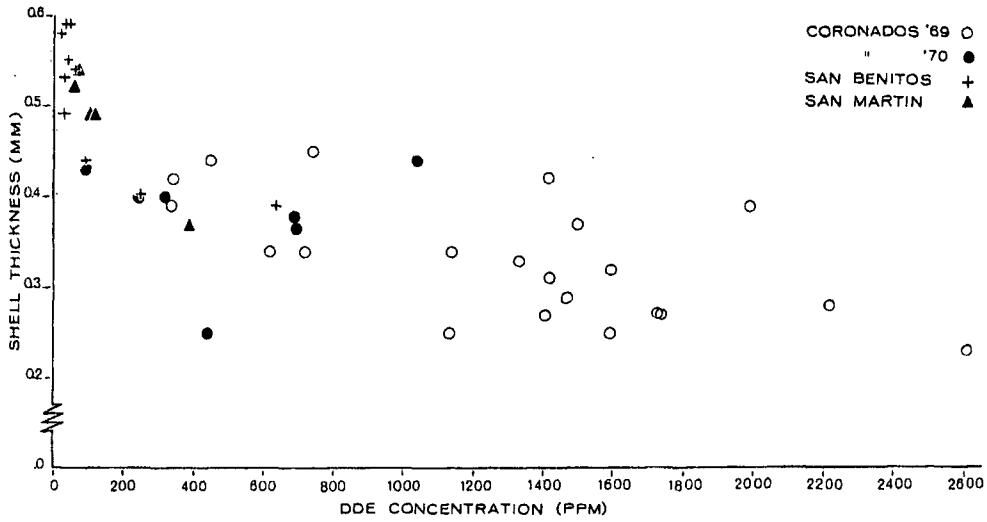

FIGURE 3. Relationship between DDE concentration and shell thickness in Brown Pelican eggs from northwestern Baja California.

Blus et al. (1974a) reported on studies of brown pelican eggs collected in 1969 and 1970 from California, Florida and South Carolina. Eggshells were thinner than pre-DDT era eggshells. DDE residues were highest in California eggs and lowest in Florida eggs. Shell thinning was highly correlated with levels of DDE in the eggs. The calculated no-effect level was 500 ppb DDE. Thinning was 4 % at 1 ppm and 15 % at 5 ppm. The observed logarithmic relationship was also reported by others for the double-crested cormorant and the prairie falcon. Dieldrin may have contributed to reproductive failure of brown pelicans. Serious population declines have occurred in California and South Carolina as a result of DDE eggshell thinning. "The 17 % eggshell thinning observed in South Carolina was associated with subnormal reproductive success." In areas with the greatest eggshell thinning, "Usually, the entire clutch exhibited the extreme thinning, and all the eggs were broken in some nests." Florida eggs from different breeding areas averaged 0.69 to 2.48 ppm DDE, with an average of 8 % shell thinning. "...the bulk of the residues in all areas of Florida are low enough that one would not expect these residues to induce widespread, long-term, adverse effects on the populations there." The log-linear relationship between DDE residues in eggs and shell thinning are illustrated in Figure 2 below:

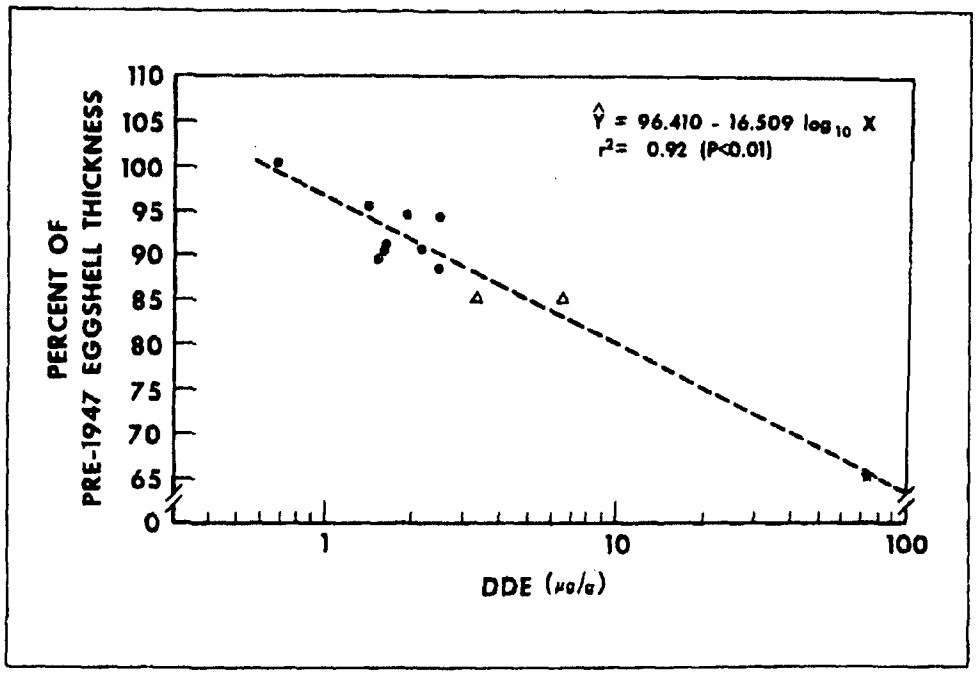

FIGURE 2.—*Association of DDE residues in brown pelican eggs from nine collections in Florida* [•], *two colonies in South Carolina* [△], *and one colony in California* [★] *with percent of pre-1947 eggshell thickness.*

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A more systematic study was done in 1971 and 1972 by Blus et al. (1974b) in a breeding colony of brown pelicans in South Carolina. One freshly laid egg was taken from each of 93 marked nests. In this way, residue level and shell thinning could be related directly to nest success. The effects of DDE on eggshell thinning and reproductive success were confounded by dieldrin. Reproductive success was normal in those nests in which a sample egg contained less than 2.5 ppm DDE.

Anderson et al. (1975) published the first report of the recovery of the brown pelican following the ban of DDT in 1972. The major source of DDT for the study populations was the wastes of the DDT manufacturer being released into the ocean by way of the Los Angeles County storm sewer outfall. Releases were greatly reduced after April, 1970. Recovery of brown pelican reproduction on offshore islands to the north and south improved quickly during the period 1971 to 1974 as shown in Table 1 below.

Table 1. Recent history of brown pelicans breeding off the coast of southern California and northwestern Baja California; productivity totals include Anacapa and Santa Cruz Islands and Isle Coronado Norte (*3*). Abbreviation: C.L., confidence level.

	No. nests built	No. young fledged		Eggshell thickness*				Reference	Anchovy abundance†
				Crushed/broken		Found intact			
		Total	Per nest	No.	$\bar{X}$ ± 95% C.L. (mm)	No.	$\bar{X}$ ± 95% C.L. (mm)		
1969	1125	4	0.004	53	0.288 ± 0.016	12	0.402 ± 0.019	(*14*)	140
1970	727	5	0.007	72	0.286 ± 0.014	16	0.393 ± 0.021	(*28*)	70
1971	650	42	0.065	17	0.310 ± 0.030	6	0.460 ± 0.026		80
1972	511	207	0.405	25	0.294 ± 0.034	4	0.438 ± 0.024		195
1973	597	134	0.225	26	0.343 ± 0.033	4	0.510 ± 0.068		275
1974	1286	1185	0.922	27	0.378 ± 0.033	59	0.482 ± 0.016		355

*Arithmetic means are given. Normal eggshell thickness for this population is 0.572 ± 0.010 mm (N = 11) (*9*); eggshells were measured by standard techniques (*9*). Intact eggs included some destroyed by predators. Thickness data for 1969 to 1973 are from Anacapa and Santa Cruz only; those for 1974 also include samples from Isla Coronado Norte, which were not significantly different. † This is an estimate of biomass expressed as thousands of schools per census in a fixed area off southern California during January to June, as derived from figure 6 of Mais (*4*).

Fledging rates increased from 0.004 to 0.922. Thicker shelled eggs and fewer broken eggs were observed with time during this period. The recovery was not complete, as a fledging rate of 1.2 to 1.5 is needed to achieve a stable population.

Direct observation confirmed that the northern anchovy was the major food item for this breeding colony of brown pelicans:

> 6. During banding at Anacapa from 1972 to 1974, we examined stomach contents regurgitated by young pelicans; the material consisted almost exclusively of anchovies. Our observations of feeding adults before and during the breeding season also indicated a heavy reliance on anchovies.

Residues of DDE in northern anchovies decreased 27-fold from 1969 to 1974. DDE in brown pelican eggs decreased 9-fold during this same period.

Table 2. Geometric mean residues of DDT and related compounds (DDE and TDE) (*12*) in anchovies and brown pelican eggs off the southern California and Baja California coasts. Abbreviations: Cr, crushed eggs; In, intact eggs; N.D., residues were not detected (< 2 ppm, lipid basis) (*24*).

Year	Anchovy whole bodies*				Brown pelican egg contents†				Reference
	Residue (ppm, fresh weight basis)				Residue (ppm, lipid weight basis)				
	No.	DDT plus TDE	DDE	Total	No.	DDT plus TDE	DDE	Total	
					Southern California and northwestern Baja California				
1969	11	1.03	3.24	4.27	73 (Cr)	49.0	1155.3	1204.3	(*14*)
					28 (In)	54.2	852.5	906.7	(*29*)
1970	15	0.56	0.84	1.40					
1971	6	0.47	0.87	1.34					
1972	8	0.38	0.74	1.12	10 (In)		220.9	> 220.9	
1973	4	0.11	0.18	0.29	4 (In)	6.5	174.9	182.9	
1974	4	0.03	0.12	0.15	39 (In)	N.D.	96.6	96.6	
					West-central Baja California				
1969	10	0.06	0.20	0.26	16 (In)	5.8	89.5	96.1	(*14*)

*Anchovies were collected from January to August each year. Individual fish were analyzed in 1969 and pools of 10 to 30 fish were analyzed thereafter; sensitivity was 0.01 ppm (*24*). The anchovies from west-central Baja California probably represent a different population (*5*). †Eggs from Coronado Norte were included only in 1969 and 1974. The pelican eggs from west-central Baja California were collected at Isla San Benito.

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The slower decline in residues in eggs compared to fish suggests that at a steady-state, the 150 ppb total DDT measured in northern anchovies in 1974 would result in an egg residue that is below the threshold for a reproductive effect.

Anderson et al. (1977) continued to study brown pelicans on Anacapa Island in 1975. The only breeding colonies in California observed by these investigators were on Anacapa Island and nearby scorpion rock. Only four eggs were collected and three of these were putrified. Lipid content of eggs was assumed to be 5 per cent. DDE residue analysis, shell thickness and productivity appeared to have leveled off in 1975 following the recovery from 1969 to 1974. PCBs were 5-10 ppm during this period.

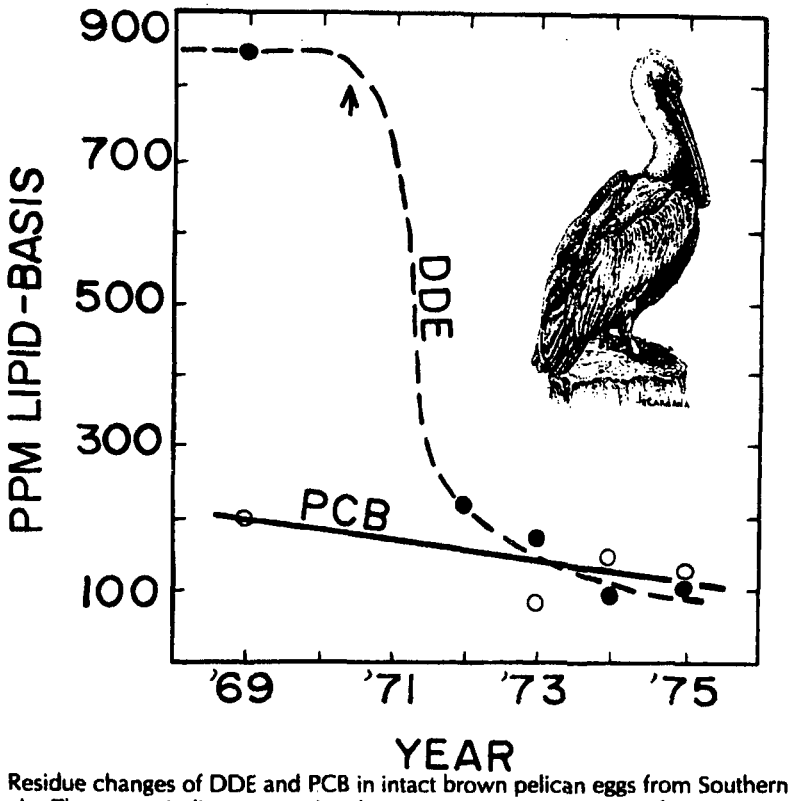

FIGURE 1. Residue changes of DDE and PCB in intact brown pelican eggs from Southern California. The arrow indicates a major drop in environmental input of DDT. According to published data, major input of DDT ceased in 1970 (Anderson et al. 1975) and by 1971 it had decreased to about 0.5% of previous levels (Jukes 1974, citing the DDT-manufacturing company president). There is some disagreement as to the actual levels of input before 1970 (Jukes 1974).

PCBs may have affected reproduction, preventing the full recovery of the colony. Limited observations in 1976 suggested that an inadequate food supply was also contributing to low productivity.

In 1977, Blus et al. published a follow-up report on the brown pelican breeding colonies in South Carolina. Shells of eggs collected from 1969 to 1973 averaged 14 to 17 per cent thinner than shells of eggs collected prior to the DDT era. Crushed shells were thinner than shells from eggs that hatched. Shells of freshly laid eggs were thinner than shells of hatched eggs. Residues of DDE in eggs decreased from 5.45 ppm in 1969 to 2.09 ppm in 1973. Reproductive success of 1.66 per nest in 1973 was considered excellent. Atlantic menhaden, a major food item of the brown pelican, contained a residue of 0.135 ppm total DDT as shown in Table 14 below.

TABLE 14. *Residues of organochlorine pollutants in Atlantic menhaden regurgitated by brown pelicans, South Carolina—1973*

Residues, µg/g fresh wet weight							
DDE	TDE	DDT	Dieldrin	Oxychlordane	*Cis*-chlordane[1]	Toxaphene	PCB's
0.04	0.04	0.04	0.03	0.01	—	0.03	0.08
0.06	0.04	0.05	0.03	0.01	0.01	0.04	0.17
0.07	0.02	0.03	0.02	—	—	0.02	0.25
0.06	0.03	0.03	—	—	0.01	0.04	0.14
0.05	0.03	0.02	0.02	—	0.01	0.02	0.10
0.08	0.03	0.02	0.02	—	0.01	0.02	0.25
0.15	0.07	0.06	0.04	—	0.02	0.04	0.24
GM 0.067	0.035	0.033	0.020			0.029	0.161
CL 0.045-0.099	0.024-0.050	0.022-0.049	0.011-0.038			0.021-0.039	0.105-0.248
Range 0.04-0.15	0.02-0.07	0.02-0.06	ND-0.04	ND-0.01	ND-0.02	0.02-0.04	0.08-0.25

NOTE: ND or — = no residue detected.
GM = geometric mean.
CL = 95 percent confidence limits.
[1] *Cis*-chlordane and/or *trans*-nonachlor.

The menhaden were recovered from regurgitated stomach contents in 1973. Biomagnification for total DDT from fish to egg was 18. Residues of total DDT in menhaden in the late 1960s was 0.295 ppm. "The migratory habits of the Atlantic menhaden (15, 17) and the brown pelican confound the significance of biomagnification noted in this study."

Thompson et al. (1977) reported on a 1970-1971 study of brown pelicans in Florida. Regurgitated food items from 14 colony sites were analyzed and found to contain an average of 0.074 ppm total DDT in 1970 and 0.047 ppm in 1971. Total DDT in fish collected in 1964-1965 averaged 0.174 ppm. Total DDT in brown pelican eggs collected in 1971 from three colony sites averaged 1.27 ppm.

King et al. (1978) reported on DDT residues and shell thinning in addled brown pelican eggs collected in 1970 along the Texas coast. The average total DDT residue was 3.23 ppm and was negatively correlated with an average 11 per cent shell thinning.

King et al. (1977) reported 10 % thinning in brown pelican eggs collected in Texas from 1970 to 1974. DDE levels declined from 3.2 ppm in 1970 to 0.86 ppm in 1974. Endrin toxicity accounted for mortality in adult pelicans and may have caused reproductive failure. Effects of DDE on reproduction during his period could not be assessed due to the small populations and confounding endrin toxicity.

Mendenhall and Prouty (1978) studied recovering populations of brown pelicans in South Carolina. A steady decline in DDE residues in eggs had a high negative correlation with increasing eggshell thickness as shown in Figure 1 below.

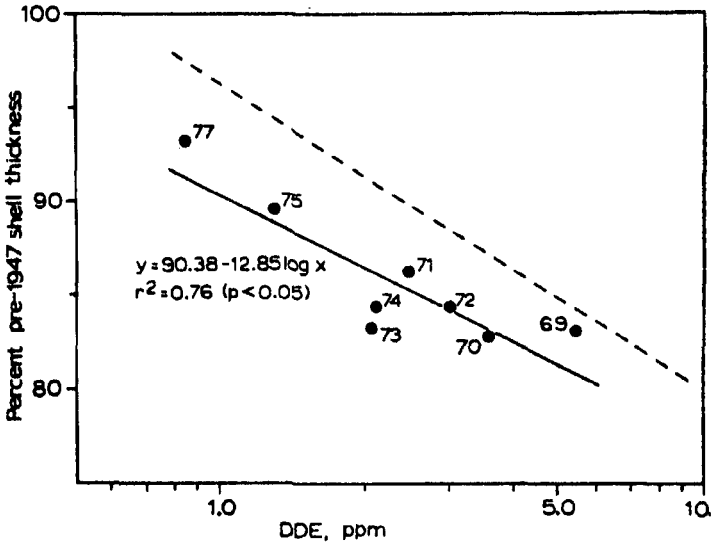

Fig. 1. (——) Change in eggshell thickness for South Carolina brown pelicans as related to DDE residues, 1969-1977. Each point shows mean shell thinning in relation to pre-1947 data (y) and mean wet-weight DDE residue (x) for one year. Sources of data as in Table 2. (----) Regression for 12 colonies in 3 states, 1969-70; $y = 96.410 - 16.509 \log_{10} x$, $r^2 = 0.92$ (Blus et al. 1974a).

Eggshell thickness in 1978 was only 6 % below the pre-1947 mean thickness. Fledgling rates continued to increase and reached a population sustaining level in 1976 as shown in Table 3 below.

TABLE 3.

Colony size (peak nest numbers) and fledging success for Brown Pelicans, South Carolina, 1969-1978.

	1969	1970	1971	1972	1973	1974	1975	1976	1977	1978
Nests	1266	1116	1469	1415	1646	1670	2400	2540	3376	3353
Fledged per nest	0.78	0.85	0.92	0.69	1.66	0.97	0.75	1.23	1.4*	1.35

* Approximate figure; see text.

Sources: 1969-76, as Table 2; 1977-78, present study.

The authors noted that in 1977 all eggs sampled were below 2.5 ppm DDE. DDE levels above 2.5 ppm had been associated with consistent nest failure.

Blus et al. (1979a) reported on a program to transplant brown pelicans from Florida to Louisiana. 765 young pelicans were transplanted in 1971 and began breeding and increasing in numbers until a severe die-off in 1975. The die-off was attributed to endrin. Eggshell thickness gradually decreased to 14 % below pre-DDT era thickness by 1974 and then began to increase thereafter. Endrin use was curtailed in 1976 and breeding improved to 1.47 fledged per nest. The authors considered fledgling rates of 1.2 to 1.5 to be necessary to maintain a stable population. DDE residues in eggs peaked at 1.36 ppm in 1972 and decreased to 0.92 ppm by 1976. The authors concluded that DDE-induced eggshell thinning was not high enough to interfere with reproductive success.

Blus et al. (1979b) reported on DDT residues, eggshell thinning and reproduction in brown pelicans in South Carolina and Florida. The primary food item of the breeding colonies, the Atlantic menhaden, were collected in 1974 and 1975 from regurgitated stomach contents in South Carolina and analyzed for DDT. From 1969 to 1975, the trend in total DDT residues in eggs from South Carolina was steadily downward from 7.81 to 1.80 ppm. DDE decreased from 5.45 to 1.40 ppm during the same period. By 1975, residues of parent DDT were barely measureable. Menhaden DDE residues were 0.016 ppm in 1974 and 0.014 ppm in 1975. Egg shells increased in thickness from 17 % thinner to 10 % thinner than pre-DDT era eggshells. Florida populations had been stable for several years. South Carolina populations were increasing. Fledgling rates in the South Carolina populations in 1975 were adequate to maintain a stable population.

Blus (1982) provided further interpretation of the relationship of DDT residues in brown pelican eggs to reproductive success. By collecting single eggs from a marked nest and following productivity in the same nest, residues of DDE could be associated directly with reproductive success. The critical level of DDE residues in eggs was 3 ppm. Residues below this level generally produced, at most, a slight reproductive effect. Residues in excess of this level were associated with a substantial effect on reproduction. A residue of 4 ppm in eggs was associated with total reproductive failure.

An overall decline in organochlorine residues in brown pelican eggs is illustrated by the authors in Figure 1 below.

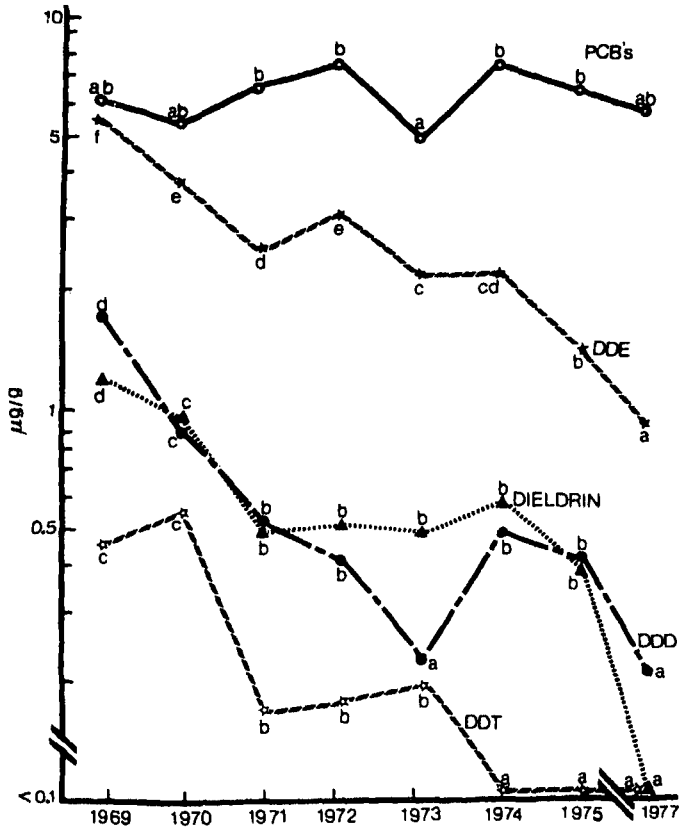

Fig. 1. Trends in five organochlorine residues detected in eggs of brown pelicans collected in South Carolina, 1969 to 1975. Means for each chemical that share a common letter are not significantly different ($p > 0{\cdot}05$) from one another.

In 1983, Anderson and Gress published an update on the status of populations of brown pelicans in the Southern California Bight. DDE residues in eggs and eggshell thinning were not measured. Fledgling rates were closely associated with stocks of northern anchovies since about 1974. The population of brown pelicans on Anacapa Island continued to increase even though fledgling rates were below one. "...1980 was the first year when reproduction was probably not drastically affected by pollution..."

Blus (1984) reported a comparison of regression and sample egg methods for predicting the reproductive effects threshold for DDE. Brown pelican eggs from California, Florida, Louisiana, and South Carolina were analyzed for DDE residue, eggshell thinning, and compared to reproductive success. Eggshell thinning of 18 % or greater had been reported to be associated with reproductive failure and population declines. An egg residue of 5 ppm DDE was associated with 18 % shell thinning by regression analysis (Figure 1 below).

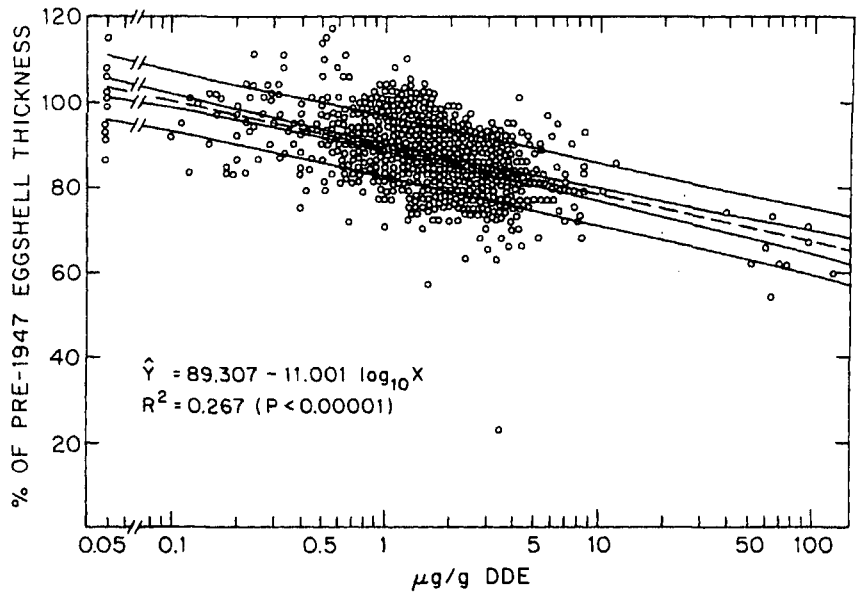

FIG. 1. Regression analysis showing the relationship of DDE residues in 813 eggs of Brown Pelicans to eggshell thickness; South Carolina, Florida, Louisiana, and California, 1969–1976. The dashed line is the regression line, the two pairs of solid lines delineate the 95% confidence limits for the population mean (inner pair) and for individual eggs (outer pair).

Using the sample egg method, reproductive effects occur at 3 ppm. The threshold is between 2.5 and 3 ppm DDE (Figure 2 below).

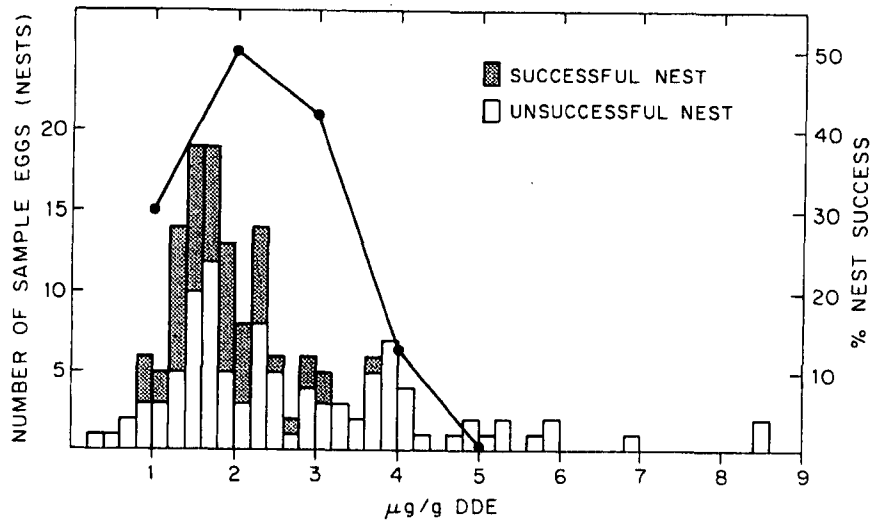

FIG. 2. Relationship of DDE residues in 156 sample eggs of Brown Pelicans to nest success. Bars represent success related to 0.2 μg/g intervals; dots on the line represent mean nest success by μg/g intervals.

The critical level of 3 ppm is associated with eggshell thinning of 16 % from the regression analysis of 813 eggs (Figure 1).

In 1985, King et al. reported on studies from 1975 to 1981 on colonies of brown pelicans in Texas. During this period, nesting pairs increased from 18 to 57. Fledgling rates were considered adequate in all years except 1975. DDE levels were about half that measured in 1970 and ranged from 0.9 to 2.3 ppm. "Current levels of DDE apparently pose a minimal threat to pelican reproduction." "Mean eggshell thickness was 4 to 14 % thinner than normal, but we found no evidence that shell thinning adversely affected reproduction." DDE residues in a major food item, the gulf menhaden, were measured at an average of 0.06 ppm in 11 fish in 1980. "DDT and metabolite residues may have been magnified 23 times from fish (0.06 ppm) to pelican eggs (1.36 ppm), but interpretation of this apparent biomagnification is complicated by the migratory habits of the pelicans and their prey."

Gamble et al. (1987) reported on a 1986 study of a colony of brown pelicans in Texas and two colonies in the Yucatan Peninsula in Mexico. DDE residues in eggs from Texas averaged 0.16 ppm. These levels reflected a ten-fold decline from 1975 levels. The authors concluded: "The concentrations of the organochlorine compounds in eggs from Texas and Mexico were below levels considered to be harmful."

In 1995, Franklin Gress published his doctoral thesis on 22 years of studies of brown pelicans on Anacapa Island. DDE residues in eggs declined slowly during the late 1970s and 1980s to approximately 2 ppm in 1992. Eggshells increased in thickness during this period. Thinning was about 5 % in 1992. Gress concluded: "... at present we have no evidence that

brown pelican reproduction in the SCB is measurably impaired by DDE-related eggshell changes..." The only breeding colonies in the Southern California Bight (SCB) are on West Anacapa Island, Santa Barbara Island and Islas Los Coronados.

ANALYSIS

Brown pelican reproduction was reduced by the direct action of the DDT metabolite, DDE, during and after the DDT use era. DDE was magnified up the aquatic food chain to the fish diet of the brown pelican and deposited in the lipid of the eggs. DDE residues above 2.5 ppm in eggs were associated with eggshell thinning in excess of 15 %, resulting in decreased hatching success. DDE egg residues below 2.5 ppm, although capable of producing measureable thinning of eggshells, were not asssociated with reduced hatching success or any other affect on reproduction. DDE residues in all populations of brown pelicans in the United States are currently below the threshold for reduced hatching success.

Brown pelicans in the Southern California Bight were most impacted by DDE during the 1960s and 1970s. The reason is the much higher contamination levels from the production wastes of DDT manufacture compared to agricultural residues generated throughout the regions populated by brown pelicans. The highly contaminated Palos Verdes shelf provides a continuing source of DDE to the northern anchovy diet of the breeding colonies of brown pelicans on Anacapa Island. For example, the Southern California Bight study of 1998 (Allen et al., 2002) found total DDT levels as high as 10.5 ppm in fish captured in the Palos Verdes Shelf area. This aquatic food-chain source explains the slow decline and leveling off of DDE residues in eggs collected on Anacapa Island. Breeding colonies further south, off Baja Califonia, have achieved much lower egg residues.

In spite of the high DDE levels on the nearby Palos Verdes shelf, the brown pelicans on Anacapa Island are apparently now below the threshold for reproductive effects (Gress, 1995). The steady-state residue level of 1.7 ppm DDE in eggs, estimated from the 1974 data, is below the threshold for reproductive effects based on the above review. This level is very close to what was measured in eggs from Anacapa in 1992.

Reports of DDE residues in the northern anchovy diet of brown pelicans were not found in published literature after 1975. Therefore, a confirmation of the biomagnification from fish diet to eggs of approximately 11, estimated from the Anderson et al. (1975) data, is not available. There does not appear to be a way to confirm with any certainty that 150 ppb DDE in fish is a no-effect level in brown pelicans.

However, one can conclude that the Anacapa breeding colony most likely represents a worst case for all other regions that are not directly influenced by DDT production wastes. That is, if reproduction in the Anacapa population is no longer affected by DDT, then one should expect that aquatic environments contaminated from agricultural use, a much lower level of contamination than that on the Palos Verdes shelf, should also no longer be at a level of DDE that would affect reproduction in brown pelicans. In fact, the margin of safety for agricultural

residues should be greater than that for the industrial wastes contaminating the food supply of the Anacapa colony.

CONCLUSIONS

- The National criterion and CTR standard for DDT in the water column is based on a fish residue of 150 ppb and reduced hatching success in a sensitive avian species, the brown pelican.

- Reproduction was inhibited in brown pelican populations in Louisiana, Florida, South Carolina, Texas and California by residues of DDE during the DDT use era.

- Since the ban of DDT in 1972, residue levels have declined, brown pelican eggshells have become thicker, and reproduction has slowly returned to normal.

- By the mid 1990s, reproduction was no longer inhibited by DDE in all populations of brown pelicans in the United States. Residues are below levels associated with reduced hatching success.

- A 27-fold decline in DDE in northern anchovies would result in a proportionate decline in egg residues to 1.7 ppm. This egg residue level is below the NOEL (no-observable-effect-level) of 2.5 ppm for reduced hatching success. Assuming a 27-fold reduction in egg residues, the CTR of 1 pptr total DDT in water and 150 ppb in fish is based on a NOEL and not a LOEL (low-observable-effect-level).

- The recovery of brown pelican breeding on Anacapa Island represents a worst case because of the very high concentrations of DDE in sediments and fish on the nearby Palos Verdes Shelf.

- The much higher level of DDE from manufacturing wastes on the Palos Verdes Shelf along with the recovery of the nearby Anacapa colony suggests that lower residues from agricultural uses should have no measurable effect on reproduction in brown pelicans.

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Appendix C: Dr. James L. Byard, "Scientific Commentary on the 1972 National Academy of Sciences DDT Guidance in Fish for the Protection of Wildlife"

SCIENTIFIC COMMENTARY ON THE 1972 NATIONAL ACADEMY OF SCIENCES DDT GUIDANCE IN FISH FOR THE PROTECTION OF WILDLIFE

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SUMMARY

State and Regional Water Quality Control Boards are planning to use the 1972 National Academy of Sciences recommendations for DDT residue guidance in fresh water and marine fish to protect wildlife. The recommendations were made by different NAS panels. Using essentially the same information, the two panels recommended DDT residue guidance in fish that differed by 20-fold. A review of the recommendations, in comparison to what was known in 1972, found major oversights of information that could have resulted in a higher marine and lower fresh water fish recommendation. The marine fish recommendation was studied in detail. Since the marine fish recommendation is based primarily on protecting the osprey, a detailed scientific review was done to evaluate the effects of DDT on reproduction in ospreys. An analysis of the data discussed in the review resulted in a new recommendation of 150 ppb DDT (DDE plus DDT) in marine fish to protect wildlife. The new recommendation considers information overlooked by the panel in 1972 as well as the extensive research done from 1972 to the present. The 150 ppb recommendation is consistent with the National criterion and State CTR standard for DDT in the water column. Both the criterion and standard are based on 150 ppb in fish to protect the brown pelican, a species with sensitivity to DDT similar to the osprey.

INTRODUCTION

A National debate over the impact of DDT on wildlife culminated in the cancellation of DDT in 1972. In the same year, the National Academy of Sciences made recommendations for DDT residue levels in fish for the protection of wildlife. One panel made a recommendation of 1,000 ppb in fresh water fish and another panel made a recommendation of 50 ppb in marine fish. The two panels cited essentially the same scientific studies of eggshell thinning and reproductive failure in sensitive avian species. Why then are the recommendations so different and which panel, if either, is right?

DDT and Seals

The Harbor Seal (Phoca vitulina)

DDT has been detected in harbor seals (*P. vitulina*) throughout the world for several decades. A series of early studies centered on the North Sea coastline documented the ΣDDT concentrations, with tissue type, commonly encountered when the insecticide was in widespread use (Koeman and van Genderen, 1966; Koeman *et al.*, 1972; Drescher *et al.*, 1977; Duinker *et al.*, 1979). ΣDDT concentrations (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE); in ppm) ranged as follows: blubber, 0.51 to 25.4; liver, 0.06 to 1.3; kidney, 0.05 to 0.76; brain, 0.038 to 3.1; spleen, 0.029 to 0.18; and heart, 0.25 to 0.60. It was obvious from an early date that the fat-soluble DDT and its associated degradation products selectively partitioned to relatively inactive adipose tissue. Thus, while tissue-borne residues could be significant, the potential for toxic effects as a result would be both low and difficult to assess.

In response to declining harbor seal populations in Dutch Wadden Sea (the southern coastal North Sea), Reijnders (1980) measured ΣDDT concentrations (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE); in ppm) in kidney, liver, and blubber (on a lipid weight basis) from harbor seals of the Wadden Sea. In adult seals, mean ΣDDT concentrations varied as follows: kidney, 0.2 to 0.9; liver, 0.4 to 2.1; and blubber, 8.5 to 47.3. He also determined that the decreased reproductive success reported for the Dutch Wadden Sea (versus the German Wadden Sea) was strongly correlated to the ten-fold higher PCB concentrations of the region; ΣDDT was not strongly correlated with reproductive success.

In 1990, Luckas *et al.* reported mean ΣDDT concentrations (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE); in ppm) in harbor seals from a number of diverse geographic locations: Norway, 1.226; Sweden, 22.498; Iceland, 1.546; Germany, 3.903, and Antarctica, 0.105. Not surprisingly, higher concentrations were associated with regions of greater agricultural activity.

In 1992 Hall *et al.* compared ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) concentrations in both victims (34) and survivors (54) of a phocine distemper epizootic to determine if a correlation with the disease may exist, indicating a possible immunosuppressive role for DDT – one has been suspected for some chlorinated biphenyls. As ΣDDT concentrations ranged from 0.13 to 12.1 ppm for live animals and 0.71 to 7.17 ppm for dead animals, no significant correlation could be made to indicate that DDT residues may have increased seal susceptibility to the disease.

Vetter *et al.* (1996) reported the mean ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE); in ppm) concentrations for 32 harbor seals collected from the North Sea between 1988 and 1995 to be 3.903 ppm (range, 1.501 to 11.475). They also found no significant difference in the ΣDDT concentrations between seal adults and pups collected prior to (1987) and during (1988) a major seal die-off, which indicated DDT was probably not the cause.

In 1997, Hayteas and Duffield reported the *p,p'*-DDE concentrations from the blubber of some 10 harbor seals collected off the Oregon coast to have a geometric mean of 1.9 ppm (range, 0.4 to 12.5 ppm); *p,p'*-DDT levels were not reported as they were negligible in all samples. They concluded that DDT contamination along the Oregon coast was relatively low, and that animals with higher residue levels may have migrated from California. Also in 1997, Mossner and Ballschmiter reported a mean ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD + *o,p'*-DDE) concentrations from two harbor seals collected from the North Atlantic Ocean to be 18.99 ppm (on a lipid weight basis).

More recently, Kajiwahara *et al.* (2001) reported the ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) concentrations (based on lipid weight) from the livers of some 10 stranded harbor seals collected between 1991 and 1997; they possessed a geometric mean of 12 ppm (range, 2.8 to 85 ppm).

In recent years, DDT contamination of harbor seals in the U.S. has been re-evaluated in light of the fact the use ban has been in place for well over 30 years. Shaw *et al.* (2005) sampled the blubber of 30 stranded harbor seals from the northwestern Atlantic coast of the U.S.; ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD + *o,p'*-DDE) concentrations ranged from 1.4 to 57.5 ppm (lipid weight). Also of note was substantial variation between adult males (12.40 ± 6.65 ppm), adult females (4.60 ± 2.56 ppm), yearlings (13.00 ± 14.40 ppm), pups (21.10 ± 19.70 ppm), and fetuses (2.21 ± 0.62 ppm).

Summary

To date, a number of investigations have confirmed the presence of DDT in harbor seals throughout the world, and thus their ability to accumulate DDT primarily via biomagnification. Concentrations vary, reflecting the varied length of use of the insecticide (banned in 1972 in the U.S., but used much more recently in other parts of the world), as well as the harbor seal's habit of feeding high on the marine food web (primarily fishes), but have been generally reported in the parts-per-million range. Toxic effects in harbor seals from DDT have yet to be conclusively demonstrated via controlled studies.

DDT and Sea Lions

The California Sea Lion (Zalophus californianus)

There are a number of reports of DDT in sea lions (*Z. californianus*) residing along the California coast. In 1971, Le Boeuf and Bonnell published a seminal report of blubber concentrations in California sea lions collected in 1970 (n = 25), a full two years prior to the banning of the use of DDT in the U.S. In it, they reported high ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) concentrations (wet weight basis: arithmetic mean, 911 ± 582 ppm and range, 41 to 2678 ppm; lipid weight basis: arithmetic mean 1452 ± 1104 ppm and range, 47 to 5077 ppm). Geometric mean values were ΣDDT (*p,p'*-DDT + *o,p'*-DDT,

17 ppm; range, 8.8 to 34 ppm) and ΣDDE (*p,p'*-DDE + *o,p'*-DDE, 740 ppm; range, 370 to 1500 ppm).

In 1992, Bacon *et al.* surveyed milk samples from a number of pinniped species, including one lactating California sea lion resident to the central coast – geometric mean values ranged from 3.3 ppb for *o,p'*-DDT to 1.4 ppm for *p,p'*-DDE. This was not considered unusual, as the area is one of intense agricultural activity and has a history of DDT use.

In 1995, Lieberg-Clark *et al.* followed up on the above 1971 report of Le Boeuf and Bonnell by measuring ΣDDT (*p,p'*-DDT + *o,p'*-DDT; in ppm) and ΣDDE (*p,p'*-DDE + *o,p'*-DDE; in ppm) concentrations in blubber from seven California sea lions sampled between 1988 and 1992. Their numbers clearly indicated a significant decline (greater than 99%) in residues over the 30-year time span for both ΣDDT (geometric mean, 0.16 ppm; range, 0.07 to 0.35 ppm) and ΣDDE (geometric mean, 5.0 ppm; range, 2.5 to 10 ppm). Therefore, they concluded the following:

1. The decline in the residue levels in California sea lions over this period was accompanied by a significant increase in their population during the same time period.
2. The high ΣDDT concentrations reported in the 1970s may have been associated with reproductive problems in California Sea Lions.
3. The decline in ΣDDT residues in California sea lions was so dramatic because their breeding area in southern California was much less contaminated with DDT residues than in 1970.

However, O'Shea and Brownell (1996) took issue with the latter statement, which they considered to be based primarily upon circumstantial evidence. For instance, they suggested that the original sample sizes (7 and 12) were too limited to draw such sweeping conclusions. In addition, they noted a paucity of experimental evidence demonstrating an impact of DDT and/or it metabolites on sea lion reproduction. In addition, O'Shea and Brownell (1996) noted that California sea lion populations have historically fluctuated, declining in the late 1800s and early 1900s, and increasing in the 1960s. Therefore, while they do not necessarily discount the observations of Lieberg-Clark *et al.* (1995), their overall contention was that to-date there was insufficient evidence to draw such conclusions.

In 1997, Hayteas and Duffield reported the *p,p'*-DDE concentrations from the blubber of some five California sea lions (in addition to harbor seals, above) collected off the Oregon coast to have a geometric mean of 8.1 ppm (range, 3.2 to 15.4 ppm); *p,p'*-DDT levels were again not reported as they were negligible in all samples. They again concluded that animals with higher residue levels may have migrated from California. Also, and most importantly, their *p,p'*-DDE value was similar to the ΣDDE value reported by the Lieberg-Clark *et al.* (1995) study, providing further confirmation of the dramatic decline in residues reported by them.

More recently, Kajiwahara *et al.* (2001) reported the concentrations of organochlorine insecticides (based on lipid weight) in some 15 stranded California sea lions collected between 1991 and 1997; in blubber, the geometric mean ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) concentration was 209 ppm (range, 13 to 2,900 ppm), while in liver it averaged 142 ppm (range, 12 to 970 ppm). Their results contrast with those of Lieberg-Clark *et al.* (1995) for animals collected during an overlapping time period; however, the Lieberg-Clark *et al.* (1995) data were reported on a wet sample weight basis.

Connolly and Glaser (2002) reported the accumulation of *p,p'*-DDE in female California sea lions resident to the California Channel Islands. Due to the high concentrations of DDT and its degradation products emanating from the Whites Point outfall, which contaminated the sediments of the Palos Verdes shelf and Santa Monica Bay, contaminated fish were suspected of serving as a vector in the transfer of such residues to the sea lion population. However, they determined that *p,p'*-DDE residues in the blubber of female premature parturient sea lions from San Miguel Island declined from a mean of 944 ppm in 1970 to 40 in 1991, while those from full-term parturient females also declined during the same time period (from 109 to 10 ppm). Both declines, approximately a full order of magnitude, were similar to that reported by Lieberg-Clark *et al.* (1995) and mirrors the declines observed in sediments and mussels. In addition, Connolly and Glaser (2002) noted that concentrations were also reduced in full-term parturient females were most likely also influenced by lactation.

As a follow up to the 1971 study, Le Boeuf *et al.* (2002) revisited the topic of organochlorine pesticides in marine mammals. They collected blubber samples from some 36 stranded animals along the coast of California in 2000, and determined mean ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) concentrations of 37 ± 27 ppm (wet weight basis) and 150 ± 257 ppm (lipid weight basis). They found no significant differences in concentrations with differences in age or sex, but did conclude that ΣDDT levels decreased by over an order of magnitude between 1970 and 2000. Kannan *et al.* (2004) also reported the results of DDT analysis performed on the blubber of some 36 stranded California sea lions collected in 2000. As Kannan is a co-author of the Le Boeuf *et al.* (2002) study, it is unclear if the animals used were the same in both studies. However, he reports a mean ΣDDT concentration of 143 ± 253 ppm, with a geometric mean of 69 ppm.

While toxicity endpoint and threshold studies involving marine mammals have been virtually impossible to conduct, two studies designed to correlate toxic effects with DDT in California sea lions have recently been published. Debier *et al.* (2005) investigated a possible relationship between ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD) concentrations in the serum of 12 healthy California sea lions and circulating levels of vitamins A and E and the thyroid hormones thyroxine (T4) and triiodothyronine (T3). While a number of negative correlations were reported for ΣPCB, only vitamin A was significantly correlated with ΣDDT concentrations, but only when they were reported on a lipid weight basis.

Also in 2005, Ylitalo *et al.* used a logistic regression model with California sea lions to attempt to correlate the unusually high prevalence of neoplasms (carcinomas – found in 18% of stranded adults) with blubber ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD + *o,p'*-DDE) concentrations. While concentrations were significantly higher in animals that died from carcinomas versus those that did not, after controlling for other confounding factors only blubber thickness proved to be a reliable predictor of death via carcinoma – ultimately ΣDDT was proven not significant.

Summary

A number of studies have confirmed the presence of DDT in California sea lions, thus their ability to accumulate it primarily via biomagnification (similar to seals, they also primarily feed on fishes). DDT concentrations have generally been reported in the parts-per-million range but have been on the decline in recent years due to the discontinuation of its use. Similar to harbor seals, toxic effects from DDT in California sea lions have yet to be conclusively demonstrated.

DDT and Dolphins (including porpoises)

While dolphins and porpoises are not likely to spend much time (if any) in the bay, to be conservative they have been included in this literature review. There are relatively few published reports of DDT in dolphins and porpoises relevant to Newport Bay.

The Pacific Bottlenose Dolphin (Tursiops gilli)

In 1980, O'Shea *et al.* reported the ΣDDT in the blubber, brain and muscle tissues of 69 small cetaceans, including one Pacific bottlenose dolphin (*T. gilli*) with an excessively high blubber DDT concentration of 2,695 ppm.

The Common Dolphin (Delphinus delphis)

Smyth *et al.* (2000) reported concentration ranges of ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD + *o,p'*-DDE) in the blubber and liver of six common dolphins (*D. delphis*) accidentally caught in fishing nets off the coast of Ireland to range from 3,998 to 9,444 ppb and 2,293 to 4,528 ppb, respectively. In 2001, Borrell *et al.* reported the ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT) concentrations measured in the blubber of common dolphins accidentally caught in fishing nets along both the Atlantic and Mediterranean coasts of Spain during a 12-year time span. In dolphins from the Atlantic mean ΣDDT concentrations did not significantly differ between 1984 and 1996 (1984: 15.54 ± 8.82 ppm; 1996: 59.55 ± 9.04 ppm). In dolphins from the Mediterranean mean ΣDDT concentrations of animals sampled in 1992 through 1994 was 33.40 ± 38.64. Of note was the fact that males in both regions accumulated significantly higher concentrations than females.

The Rough-Toothed Dolphin (Steno bredanensis)

No published papers were found describing any aspect of DDT or its degradation products with rough-toothed dolphins.

Summary

There are few reports of DDT concentrations in dolphins or porpoises important to the Newport Bay region. Those above are for animals sampled elsewhere in the world – while they demonstrate the ability of both common and bottlenose dolphins to accumulate DDT and its degradation products, the actual concentrations probably do not reflect animals residing on the California coast. Similar to harbor seals and California sea lions, toxic effects from DDT in the subject dolphins have yet to be conclusively demonstrated via controlled studies.

DDT and Whales

Although whales (baleen or toothed) are not likely to spend time in Newport Bay, again to be conservative a summary of pertinent publications involving DDT and the whale species most likely to at least briefly visit the area is presented below.

The Gray Whale (Eschrichtius gibbosus)

Over the years a number of studies have reported on the contaminants present in the blubber of baleen whales, including gray and minke whales. For instance, in gray whales (*E. gibbosus*) Wolman and Wilson (1970) measured ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) concentrations as high as 680 ppb in some 23 animals collected between 1968 and 1969, while Schaffer *et al.* (1984) reported a concentration of 470 ppb in a single animal sampled in 1976. In 1994, Varanasi *et al.* reported the concentrations of ΣDDE (*p,p'*-DDE + *o,p'*-DDE) in the tissues and stomach contents from 22 gray whales stranded between 1988 and 1991 along the coast from Kodiak Island, Alaska, to San Francisco, California. Gray whales have the unique habit of filter feeding along benthic sediments. Therefore, they are potentially capable of ingesting sediment-sorbed organic contaminants. Mean concentrations, and the ranges, measured in blubber were: ΣDDT (*p,p'*-DDT + *o,p'*-DDT), 68 ± 22 ppb (1 to 370 ppb); ΣDDD (*p,p'*-DDD + *o,p'*-DDD), 76 ± 24 ppb (1 to 470 ppb); and ΣDDE (*p,p'*-DDE + *o,p'*-DDE), 310 ± 96 ppb (9 to 2,100 ppb). In liver they were predictably reduced: ΣDDT, 1 ± 0.4 ppb (0.4 to 3 ppb); ΣDDD, 23 ± 5 ppb (0.6 to 52 ppb); and ΣDDE, 100 ± 28 ppb (7 to 280 ppb). Most interestingly, they found no significant differences in the concentrations from whales collected in the more pristine Kodiak Island/Washington outer coastal areas versus those collected in the more impacted areas of Puget Sound, Washington, and San Francisco.

Tilbury *et al.* (2002) sampled gray whales from a subsistence harvest in the Arctic during the fall of 1994 and compared their ΣDDT concentrations (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD + *o,p'*-DDE; per lipid weight) with those of stranded gray whales from the same general collection area. They discovered significant differences in

the harvested versus stranded whale blubber concentrations of males (200 ± 38 ppb versus 39,000 ± 23,000 ppb), females (360 ± 66 ppb versus 2,8000 ± 1,000 ppb) and juveniles (330 ± 53 ppb versus 11,000 ± 4,300 ppb), respectively. The consistently higher concentrations in stranded animals may indicate their possible cause of death. However, tissue degradation of dead and potentially decaying animals limits the usefulness of such a comparison.

The Minke Whale (Balaenoptera acutorostrata)

In minke whales, Schafer *et al.* (1984) reported a ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) concentration of 587 ppm from a single animal stranded off southern California. However, this high concentration appears to be linked to an urbanized area, as 29 minke whales sampled off the South African coast ranged only as high as 820 ppb (Henry and Best, 1983), while another 37 sampled in Antarctica ranged from 10 to 140 ppb (Tanabe *et al.*, 1986).

In 1998, Klevaine and Skaare published their findings on the chemical concentrations in some 72 minke whales stranded along the northeastern Atlantic seaboard (coastal Norway, West Spitsbergen Island, and Bear Island) in 1992. While they found no significant differences in mean ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD) concentrations between juvenile males versus females (1.94 versus 2.77 ppm lipid weight, respectively), they did conclude differences existed between adult males and females (3.86 versus 1.51 ppm, respectively), as well as between juveniles and adults (both males and females).

The ΣDDT (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD + *o,p'*-DDE) concentrations were also determined for some 155 minke whales harvested in 1998 from the North Atlantic and European Arctic Oceans (Hobbs *et al.*, 2003). Concentrations ranged from 65.3 to 6,280 ppb (lipid weight basis), a range that encompasses the concentrations measured in whales taken six years earlier by Klevaine and Skaare (1998).

Finally, in one of the few mechanistically-oriented papers involving any cetacean, Niimi *et al.* (2005) reported the full-length cDNA sequences of two cytochrome P450 (CYP) isozymes, from minke whale liver, responsible for either the bioactivation or detoxication of xenobiotic chemicals. While CYP1A1 consisted of 516 amino acid residues and was deemed most closely related to that from sheep and pigs, CYP1A2, also consisting of 516 residues, was deemed most closely analogous to that from humans, indicating that the enzyme's function in minke whales may be similar to that of humans. However, Niimi *et al.* (2005) found no significant correlation between hepatic DDT levels and mRNA expression levels of CYP1A1 and CYP1A, indicating that DDT may not be responsible for their induction in minke whales.

Summary

The few studies reporting DDT in gray and minke whales indicate that they are also capable of accumulating residues in their blubber and other tissues. However, since they

feed fairly low on the marine food web (invertebrates), their residue levels tend to be relatively low when compared to those of fish-eating marine mammals (seals, sea lions, and dolphins). Similar to the other marine mammals discussed above, toxic effects from DDT in gray and minke whales have yet to be conclusively demonstrated.

ANALYSIS

Residues of the fat-soluble DDT and/or its degradation products have been detected in a number of marine mammalian species worldwide since the mid-1960s. In general, during the DDT-use era blubber concentrations in the parts-per-million range were not uncommon, particularly for the species that feed primarily on fishes, and are thus higher on the marine food web. Of importance to the Newport Bay region are the harbor seal, California sea lion, and the Pacific bottlenose and common dolphins. Clearly, marine mammals are capable of accumulating residues as long as they are also accumulating in the environment. However, over the years since the ban on DDT in the U.S. tissue concentrations have decreased in tandem with the decline in environmental concentrations. A similar trend has been observed for gray and minke whales. However, since, as baleen whales, they tend to feed at lower levels of the marine food web, blubber concentrations have tended to be an order of magnitude lower in those species – in the parts-per-billion range.

One area of focus of this paper was to be on the role of DDT in possible embryo deformities and/or other measurable health effects. However, marine mammals are a unique class or animals in that published reports on controlled studies documenting such toxic effects were not encountered. There appear to be two reasons for this. First, they are too large and heavy to be easily housed, handled and utilized in controlled experiments with sample sizes sufficient to provide for statistical analysis. Second, they have been strictly protected by the federal government for many years, which has severely limited access to them. As a result, and as can be deduced from the chronology above, nearly all studies involving marine mammals and toxicants have been limited to residue analyses involving either dead/decaying animals or live, captive animals sampled via blubber biopsies.

These restrictions have limited the field to speculation on the effects of DDT residues in marine mammals based upon measured residues and, in some cases weak, correlations. However, since blubber is metabolically a relatively inactive tissue, it is assumed that large concentrations would need to be attained before measurable effects would be observed. Thus, to date few if any toxic impacts have been clearly delineated for DDT in the marine mammals that constitute the focus of this report, and with tissue residues clearly on the decline, the likelihood that such impacts might be identified in the future is also declining.

CONCLUSIONS

The following points can be made regarding DDT in the marine mammals of importance to Newport Bay:

- Concentrations of DDT and/or its degradation products have been reported in various marine mammals since the 1960s, which indicates their ability to accumulate the highly fat-soluble compounds.

- Via biomagnification, blubber concentrations in fish-eating harbor seals, California sea lions, and Pacific bottlenose and common dolphins have typically been in the parts-per-million range. Since they filter feed at lower levels of the marine food web, blubber concentrations in baleen whales such as gray and minke whales have tended to be in the parts-per-billion range.

- In general, the ΣDDT concentrations in all marine mammalian species of importance to Newport Bay are in decline, which reflects currently declining environmental concentrations worldwide as well as in the region.

- Due to strictly limited access to these marine mammals, no published reports describing the toxic effects of DDT and/or its degradation products deduced from controlled potency or mechanistic studies utilizing statistically-relevant population sizes were encountered in the relevant literature.

- With continued species access limitations and housing/handling difficulties, the potential toxic actions of DDT and/or its residues are not likely to be delineated in the near future.

- Since DDT concentrations are on the decline in Newport Bay (i.e sediments and shellfish), and with the ephemeral nature of marine mammal visitation to the region, it is unlikely that sufficient concentrations will be accumulated in the region to cause toxic consequences, particularly given that accumulations tend to occur in the metabolically relatively inactive blubber tissue of the subject mammals, suggesting that concentrations would have to be quite high to precipitate measurable toxic effects.

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Appendix I

MARINE MAMMALS OF THE PACIFIC COAST

The information presented below was derived from Ingles (1965) and Burt (1976). Species of importance to Newport Bay, California, are denoted with an asterisk (*).

ORDER ODONTOCETI (toothed whales)

Family Ziphiidae (beaked whales)

Baird beaked whale (*Berardius bairdii*) – Marine; rare along the California coast.

Pacific (Stejneger) beaked whale (*Mesoplodon stejnegeri*) – Marine; rare along the California coast.

Archbeak whale (*Mesoplodon carlhubbsi*) – Marine; rare along the California coast.

Goosebeak whale (*Ziphius cavirostris*) – Marine; rare along the California coast.

Japanese (ginkgo) beaked whale (*Mesoplodon ginkgodens*) – Marine; rare along the California coast.

Family Physeteridae (sperm whales)

Sperm whale (*Physeter catodon*) – Rare along the California coast.

Pygmy sperm whale (*Kogia breviceps*) – Rare along the California coast.

Dwarf sperm whale (*Kogia simus*) – Marine; rare along the California coast.

Family Delphinidae (porpoises and dolphins)

*Pacific bottlenose dolphin (*Tursiops gilli*) – Frequents the southern California coast north to SF Bay; generally offshore.

Graffman dolphin (*Stenella graffmani*) – ??

Striped (longsnout) dolphin (*Stellena caeruleoalba;* formerly *S. styx*) – Columbia river to the Bering Sea.

*Rough-toothed dolphin (*Steno bredanensis;* formerly *S. rostratus*) – Coastal waters; California and southward.

*Common dolphin (*Delphinus delphis*) – Found offshore along the entire Pacific coast, including California.

Northern right whale dolphin (*Lissodelphis borealis*) – Bering Sea south; rare along the California coast.

Pacific white-sided dolphin (*Lagenorhynchus obliquidens*) – Nearshore waters; commonly in schools on the open sea along entire Pacific coast.

Common (harbor) porpoise (*Phocaena phocoena*) – Offshore north of Pismo Beach and in SF Bay.

Dall porpoise (*Phocoenoides dalli*) – Usually well offshore; Pacific coast south, rarely to Long Beach.

Killer whale (*Orcinus orca*) – Entire Pacific coast.

Grampus (*Grampus griseus*) – Entire Pacific coast; rare along the California coast.

False killer whale (*Pseudorca crassidens*) – Pacific coast from Washington south.

Short-finned (Pacific) blackfish or pilot whale (*Globicephala macrorhyncha;* formerly *G. scammonii*) – Pacific coast; well offshore in schools.

ORDER MYSTICETI (baleen whales)

Family Eschrichtidae (gray whales)

*Gray whale (*Eschrichtius gibbosus; formerly E. glaucus, R. glaucus, E. robustus*) – Coastal waters; migratory from Baja California to the Arctic Ocean.

Family Balaenopteridae

Blue whale (*Balaenoptera musculus;* formerly *Sibbaldus musculus*) – North and South Poles, but rare along the California coast.

Finback whale (*Balaenoptera physalus*) – Most common along Pacific coast in summer.

Rorqual (sei) whale (*Balaenoptera borealis*) – Along entire Pacific coast.

*Minke (piked) whale (*Balaenoptera acutorostrata*) – Near shore waters; along entire Pacific coast.

Humpback whale (*Megaptera novaeangliae*) – Entire Pacific coast; common off Washington coast.

Family Balaenidae (right whales)

Right whale (*Balaena glacialis;* formerly *Eubalaena sieboldi*) – Entire Pacific coast; rare along the California coast.

ORDER CARNIVORA

Family Mustelidae (weasels, minks, martens, skunks, badgers, fishers, wolverines)

River otter (*Lutra Canadensis*) – Central California north to Washington; along rivers, streams, marshes, lakes and estuaries.

Sea otter (*Enhydra lutris*) – Kelp beds and rocky shores; mainly from the Channel Islands north to San Francisco, then north to Alaska.

ORDER PINNIPEDIA

Family Otariidae (eared seals and sea lions)

Alaska (northern) fur seal (*Callorhinus ursinus*) – Marine; principally on Pribilof Islands, but in California to Washington, 10-50 mi offshore; can winter as far south as San Diego.

Guadalupe (southern) fur seal (*Arctocephalus philippi; formerly A. townsendi*) – Marine; rare, but on San Nicholas Island and southward on offshore islands.

Northern (steller) sea lion (*Eumetopias jubatus*) – Marine, but occasionally goes up rivers; Channel Islands (Santa Rosa Island) and north, but mainly north of SF.

*California sea lion (*Zalophus californianus*) – Baja California to British Columbia; rocky shoreline and islands.

Family Phocidae (hair seals and earless seals)

*Harbor seal (*Phoca vitulina*) – Coastal waters; mouths of rivers, shallow harbors, inland lakes. Found from the Arctic south along the Pacific.

Ribbon seal (*Histriophoca fasciata*) – Northwestern Bearing Sea; rare along the California coast.

Northern elephant seal (*Mirounga angustirostris*) – Coastal waters and sandy beaches; from British Columbia and along California coast, mainly on the islands off Southern California. Co., Boston, 289 pp.

Appendix F: Dr. James L. Byard, "Scientific Commentary on Sediment TELs for Total DDT"

SCIENTIFIC COMMENTARY ON SEDIMENT TELs FOR TOTAL DDT

James L. Byard, Ph.D., D.A.B.T.

OCTOBER 5, 2006

SUMMARY

The data points underlying the threshold effects levels (TELs) for total DDT in sediments were analyzed to determine the ability of TELs to predict thresholds for toxicity. The data sets for freshwater and marine TELs were found to be erroneous due to many problems with individual data points. Errors in interpretation of data points, repeated use of the same data points, use of outdated values for Koc and Kow, arbitrary selection of data points, inconsistent correction for organic carbon, use of parent DDT data points for the total DDT TELs, and the use of low residue effect data points when higher levels were without effect, all contributed to flawed data sets. If these flaws had been corrected, the TEL values would be much higher. However, the corrected TELs would still rely primarily on the co-occurrence of toxicity and DDT in sediments, and not on a true dose-response. Many of the toxic sediments used to derive TELs are contaminated by other pollutants, often at levels that could account for the observed toxicity. Spiked sediment bioassays and studies of benthic communities in sediments highly contaminated by DDTs indicate that the toxicity threshold for total DDT to benthic organisms is more than two orders of magnitude higher than the TELs proposed for use in Newport Bay and San Diego Creek.

INTRODUCTION

On June 14, 2002, the U.S. EPA, Region IX (EPA), promulgated total maximum daily loads (TMDLs) for total DDT (sum of DDT, DDD and DDE) in the San Diego Creek and Newport Bay in a document titled: Total Maximum Daily Loads for Toxic Pollutants, San Diego Creek and Newport Bay, California (U.S. EPA, 2002). The final EPA DDT TMDL went largely unreviewed because it was so different from the draft that went through internal and external review. In 2005, the Santa Ana Regional Water Quality Control Board (SARWQCB) and stakeholders took a closer look at the derivation of the DDT TMDL and found it difficult to understand (Rose, 2005a; Rose, 2005b; Byard, 2005a; Byard, 2005b). There were many errors, wrong assumptions and contradictions. The use of threshold effects levels (TELs) as sediment targets was based largely on the occurrence of DDT and toxicity in the same sediments and not on a true dose-response. In their most recent report on the organochlorine TMDLs, staff at the

SARWQCB (Rose, 2006) have decided to use TELs to achieve DDT residue targets in fish. This report will take a detailed look at the scientific basis for the sediment TELs for total DDT in marine and fresh waters. A good starting point is a conceptual model that explains how sediment targets achieve protection of beneficial uses.

CONCEPTUAL MODEL

A TMDL should achieve levels in water and sediment that will not bioaccumulate in aquatic life to levels that are harmful to wildlife or human health. The TMDL should be based on a conceptual model that is consistent with the fate and toxicity of DDT and is applicable to San Diego Creek and Newport Bay. A conceptual model is also helpful in understanding the derivation of a TMDL. Figure 1 portrays a conceptual model for the fate of DDT in the environment relevant to a TMDL for San Diego Creek and Newport Bay.

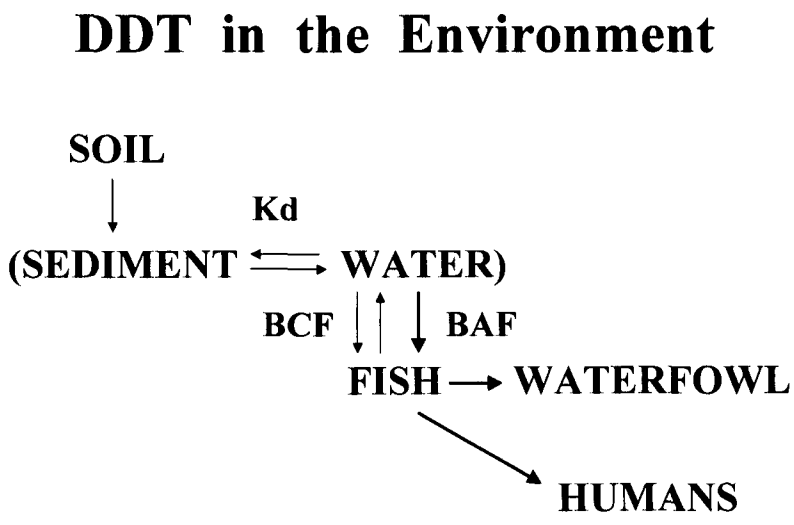

Figure 1. Conceptual model of the fate of DDT in the environment.

Soil residues are eroded into channels in the San Diego Creek Watershed and carried as sediment to Newport Bay. A distribution constant, Kd, describes the equilibrium between DDT in sediment and DDT in water. Bioconcentration factors (BCF) describe the equilibrium between water and highly perfused fish tissues. Bioaccumulation factors (BAF) describe the accumulation of DDT up the aquatic food chain to fish and top-of-the-food chain feeders like humans and waterfowl. The one directional arrows reflect the very slowly reversed storage of DDT in poorly perfused adipose tissue of fish, birds and humans.

The EPA also used the same conceptual model to determine the loading capacity and existing loads. Figure 2 illustrates the derivation of the loading concentration for San Diego Creek.

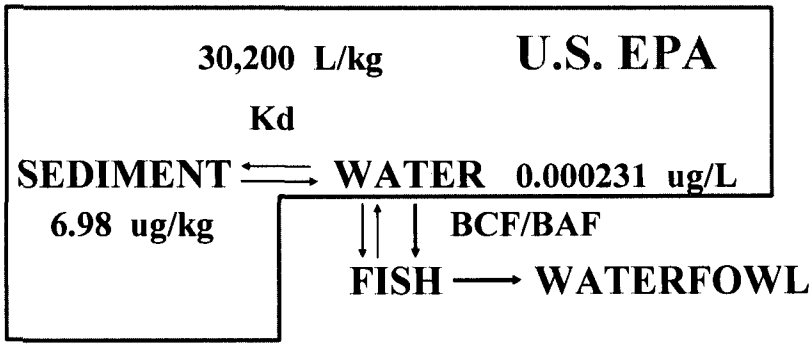

Figure 2. DDT loading concentration determined from a sediment TEL.

Because most of the DDT in the aquatic environment is bound to sediment, the EPA promulgated DDT sediment targets (Buchman, 1999) they said were necessary to achieve beneficial uses. Sediment targets of 6.98/3.89 ppb for Creek/Bay are based largely on a statistical association of DDT levels and degree of toxicity to benthic organisms. The derivation of the TEL is explained in the following excerpt reproduced from Macdonald et al. (1996).

> **For each analyte, a TEL was derived by calculating the geometric mean of the 15th percentile of the effects data set and the 50th percentile of the no effects data set.**

A theoretical plot of the data used to derive a TEL is shown in Figure 1 from MacDonald et al. (1996).

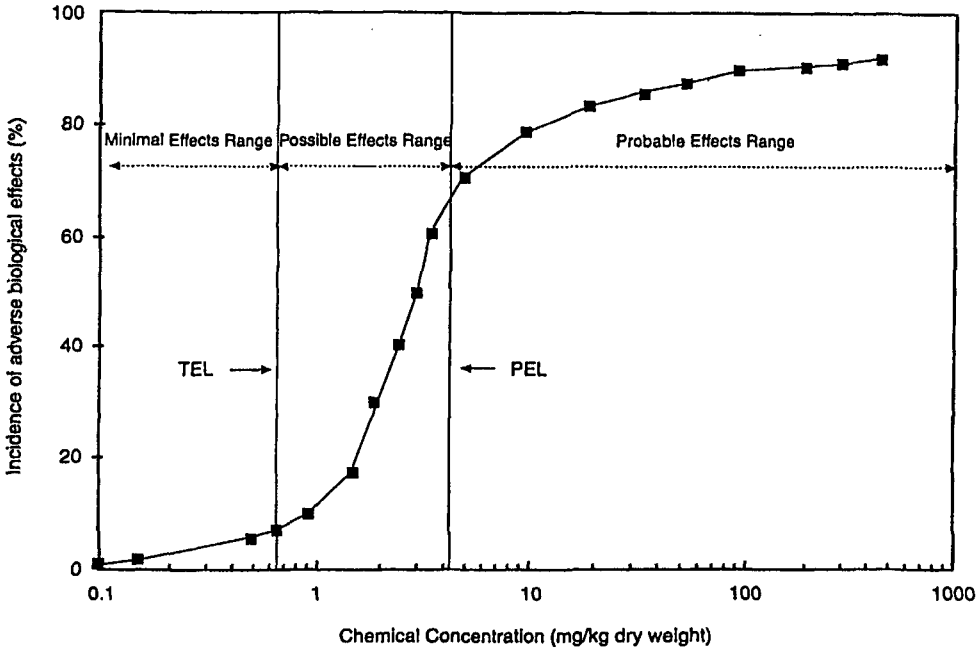

Fig. 1. Conceptual representation of the ranges of contaminant concentrations defined by SQGs and *the potential for observing adverse effects within these ranges.*

From the figure above, one might get the impression of a dose-response. However, most of the data points in the plot are based on an association of effect and concentration in the sediment. For these data points, any one or more of hundreds of chemicals in a sediment could be causing the effect. Here is what several key authors have to say about TELs for DDT:

Peter Kozelka and David Smith (EPA, 2002) at Region IX, authors of the 2002 TMDL document, said:

> "We recognize these NOAA values have been derived by associating nationwide sediment chemistry data sets with benthic toxicity results and there is no direct cause and effect relationship."

Buchman (1999), author of the table listing the sediment targets, said :

> "These tables are intended for preliminary screening purposes only: they do not represent official NOAA policy and do not constitute criteria or clean-up levels."

MacDonald, et al. (MacDonald et al. 1996; Smith et al. 1996) authors of the primary reference cited by Buchman said:

> "Low reliability (TS = 0) was indicated for only one substance (total DDT)."

MacDonald et al. (1996) also stated:

> "...the guidelines developed in this study do not address either the potential for bioaccumulation or the associated adverse effects of bioaccumulation on higher trophic levels."

Sediment residues of DDTs in Newport Bay also appear not to account for toxicity seen in a recent study of toxicity to a benthic organism. A SCCWRP scientist, Steven Bay, stated in his report on the toxicity of sediments to a benthic organism in Newport Bay (Bay, et al, 2004):

> "Relatively low correlations were present between sediment toxicity and the concentration of trace organics (PCBs, PAHs, or DDTs)."

To gain a full understanding of the individual data points from which the fresh water and marine TELs for total DDT were derived, each data point in the TELs reported by Buchman (1999) as cited by the EPA (2002) was reviewed. The data points and reference citations were obtained directly from the author of the TELs (MacDonald, 2005). The data sets are slightly different than the ones used to derive the published TELs (MacDonald, 2005). The original data sets were not memorialized and are, therefore, unavailable. Definitions of the abbreviations and notations used to describe the data sets can be found in MacDonald, 2005 (Appendix I). Each data point was reproduced below from MacDonald (2005) in the order of increasing DDT. Contiguous data points from the same study are reproduced together. Relevant comments follow each individual or group of data points.

FRESH WATER SEDIMENT TEL

Table FW-1. A summary of the available data on the biological effects associated with sediment-sorbed TOTAL DDT (ppb) used to support the derivation of sediment quality guidelines for freshwater ecosystems.

Total DDT Conc. +/- SD	Bit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
0.384 +/-0.469	NE	Upper Mississippi River, MS	COA	4-d	Not significantly toxic (8+/-6.3% mortality)	Gammarus pseudolimnaeus (Scud)	VAR			Marking et al. 1981
0.384 +/-0.469	NE	Upper Mississippi River, MS	COA	4-d	Not significantly toxic (2+/-4.8% mortality)	Procambarus sp. (crayfish)	VAR			Marking et al. 1981
0.384 +/-0.469	NE	Upper Mississippi River, MS	COA	4-d	Not significantly toxic (16.5+/-21.7% mortality)	Hexagenia sp. (mayfly)	VAR			Marking et al. 1981
0.384 +/-0.469	NE	Upper Mississippi River, MS	COA	4-d	Not significantly toxic (13.5+/-13.8% mortality)	Physa gyrina (snail)	VAR			Marking et al. 1981
0.384 +/-0.469	NE	Upper Mississippi River, MS	COA	4-d	Not significantly toxic (0% mortality)	Truncilla donaciformis (fawnfoot clam)	VAR			Marking et al. 1981
0.384 +/-0.469	NE	Upper Mississippi River, MS	COA	4-d	Not significantly toxic (11+/-8.4% mortality)	Sphaerium sp. (fingernail clam)	VAR			Marking et al. 1981

Toxicity was observed in sediments from two of the three locations with detectable DDT. The DDT analytical data is inconsistent with the major degradate being DDE. For example, Red Wing Commercial Harbor sediments were toxic in three species and contained 5.28 ppb DDD, 0.56 ppb DDT and only 0.28 ppb DDE. The high proportion of DDD is contrary to the general finding that old residues of DDT are predominantly DDE. DDD is less stable in the environment than DDE.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1.5	*	United States	EqPA		Chronic Marine EqP Threshold	Aquatic biota		1		Bolton et al. 1985

Table 2.1 lists threshold contamination concentrations for sediments based on 4 % organic carbon and the equilibrium between organic carbon and water. The threshold in water is the chronic National criterion. The organic carbon is corrected to 1 %. Only DDT is included. Total DDT from this study is not determined and used in the TEL data set. For example, the threshold concentration for DDE (the predominant form of DDT in the environment) in this study is 28 ppm! The problem with these data is the apparent use of old Koc values (Koc is the the equilibrium constant between water and the organic carbon in sediment) that give inaccurate estimates of the partition of DDTs between sediment organic carbon and water. The likely threshold concentration for DDT is higher than 1.5 ppb at 1 % organic carbon, and lower for DDE and DDD at 7,000 ppb and 3,250 ppb, respectively.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1.9	*	United States	SLCA		National Screening Level Criteria - Freshwater			1		Neff et al. 1986

The fresh water SLCA is normalized to 1 % organic carbon. The salt water SLCA is 428 ppb and is based on sediments from the Southern California Bight. Neff et al. suggest that the difference is due to low DDT levels in the fresh water sediment data base and much higher DDT levels in the salt water sediment data base. Therefore, the difference appears to be an artifact of the method by which SLCA values are derived. The salt water SLCA is not used to derive the marine sediment TEL.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5	*	Canada	EqPA		Sediment Quality Guidelines			1		Hart et al. 1988

This fish tissue-based guidance is derived from the equilibrium between water and sediment organic carbon, using a logKoc of 5.92. The logKoc of 5.92 is a geometric mean of values ranging from 5.26 to 6.58. The 6.58 value is closer to values obtained from the superior slow-stir method. The EqPA value becomes 23 ppb with the higher Koc. The value has been normalized to 1 % organic carbon. If one assumes a proportion of 80 % DDE, 10 % DDD and 10 % DDT as an example of the residues typically found in sediments, the logKoc would be 6.77, using the Kocs selected by the EPA in their 2002 DDT TMDL for Newport Bay and San Diego Creek. The higher Koc would result in a EqPA value of 36 ppb.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5	NC	DuPage River Basin, IL	COA		Low biotic integrity (7.9; MBI)	Macroinvertebrates				IEPA 1988b

The one station with an MBI of 7.9 had no detectable total DDT, with a detection limit of 10 ppb. Presumably, the TEL data point is one-half the detection limit. Organic carbon in sediments was not reported.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5	NC	Kishwaukee River Basin, IL	COA		Low biotic integrity (7.7; MBI)	Macroinvertebrates				IEPA 1988a
5	NE	Kishwaukee River Basin, IL	COA		High taxa (16.3+/-4.61 S)	Benthic species				IEPA 1988a
5	NE	Kishwaukee River Basin, IL	COA		High biotic integrity (47.9+/-4.36;	Freshwater fish				IEPA 1988a

The report describes one sediment that had 15 ppb total DDT (Mokeler Creek). This sampling site was described as: "Mokeler Creek station (PQEA-01) had the maximum mean values for ammonia nitrogen, un-ionized ammonia, dissolved phosphorus, oil and grease, fluoride, and boron and the second highest WQI value (56.2)." The WQI value and the author's statement indicates that the low biotic integrity at Mokeler Creek is due to pollutants other than DDT. The remaining 24 sediment sampling sites all had nondectable total DDT with a detection limit of 10 ppb. Organic carbon levels in the sediments were not reported.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5	NE	Galveston Bay, TX	COA		Low species (11.2+/-1.94 S/0.00203	Benthic species		0.642+/-0.356	13.4+/-8.65	Carr 1992
5	NE	Galveston Bay, TX	COA		High abundance (154+/-30.2 N/0.00203 sq.m.)	Benthic invertebrates		0.47+/-0.27	9.07+/-2.94	Carr 1992
5	NG	Galveston Bay, TX	COA		Low abundance (53+/-33.9 N/0.00203 sq.m.)	Benthic invertebrates		0.985+/-0.247	22+/-11.2	Carr 1992

The DDT analyses for this study were all nondetectable with a detection limit of 10 ppb. Toxicity varied by location. Presumably the 3 values listed above are one-half the detection limit. Sediment samples from 35 locations were analyzed. Total organic carbon was measured, but the DDT values were apparently not normalized by organic carbon.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5.42 +/-2.04	NE	Kishwaukee River Basin, IL	COA		High biotic integrity (5.18+/-0.713;	Macroinvertebrates				IEPA 1988a

If one averages the one dectect of 15 ppb DDT and one-half of the 24 nondetects at the 10 ppb detection limit, one gets 5.4 ppb. The 5.18 biotic integrity index is presumably the average of the 25 stations.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
6	NE	St. Lawrence River	SBA		Sediment Quality Criteria - No Effects Threshold					Environment Canada 1992

The value of 6 ppb for DDT is for the parent compound and not total DDT. The value is considered background in sediments from the Saint Lawrence River at relatively unpolluted sites where no effects were observed on benthic organisms. The number is based on professional judgement. Actual data and calculations are not presented in the reference. The SBA is not corrected for organic carbon.

Total DDT Conc. +/- SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
7	*	Ontario	SLCA		OMOE Provincial SQGs - Lowest Effect Level			1		Persaud et al. 1991

The SLC method is the same as used by Neff et al. (1986) to derive the value of 1.9 ppb above. The method is described, but the actual data used to determine the 7 ppb value are not presented. The SLCA value is normalized to 1 % organic carbon.

Total DDT Conc. +/- SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
7 +/-4.47	SG	Kishwaukee River Basin, IL	COA		Low taxa (8.4+/-0.55 S)	Benthic species				IEPA 1988a
7.5 +/-5	SG	Kishwaukee River Basin, IL	COA		Low biotic integrity (37+/-2.45; AIBI)	Freshwater fish				IEPA 1988a

The first data point is the mean of one sediment with 15 ppb total DDT and four sediments with no detectable DDTs (one-half of detection limit of 10 ppb gives 5 ppb). The second data point is the same 15 ppb and three other nondetects.

Total DDT Conc. +/- SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
9	*	St. Lawrence River	SLCA		Sediment Quality Criteria - Minimal Effects Threshold					Environment Canada 1992

The value of 9 ppb for DDT is for the parent compound and not total DDT. The value is derived by the screening level concentration (SLC) method. Actual data and calculations are not presented in the reference. The SLCA is not corrected for organic carbon. This data point, the 1.9 ppb data point (Neff et al., 1986) and the 7 ppb data point (Persaud et al., 1991) are all derived by the SLC method, and are essentially the same, except for regional differences in sediment residue levels and biota. All three of these data points rely on mutual occurrence. None of them identify causality or represent a measure of dose-response to DDT.

Total DDT Conc. +/- SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
10	*	Great Lakes	SBA		WDNR Interim Criteria for In-Water Disposal of Dredged Sediments					Sullivan et al. 1985 (As cited in Fitchko 1989)

This value is an interim guidance developed by Wisconsin for dredge materials. The number is derived from background sediments and bluff soils from the Great Lakes. No data or calculations are presented. No indication is given whether DDT represents total DDT or just the parent compound. The guidance calls for measure of TOC, but there is no mention as to whether the guidance is to be normalized to 1 % OC.

Total DDT Conc. +/- SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
10	*	United States			Texas Water Quality Board Average Historical Concentrations					TWQB 1977 (As cited in Dickson et al. 1989)

The value of 10 ppb is for the parent compound, DDT. No gradient of DDT concentration and no bioassays are associated with this data point. So, it is unclear why the 10 ppb is included in the effects data base.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
19.6 +/-18.4	NE	DuPage River Basin, IL	COA		High taxa (15.8+/-2 S)	Benthic species				IEPA 1988b

The value of 15.8 for the total number of taxa appears to be the correct average of the last nine stations. However, the average total DDT for those nine stations (using one-half of the detection limit of 10 ppb when the result was nondetectable) was only 9.7 ppb and not the figure of 19.6 ppb shown. One must assume some other subset of the 21 stations were used for the TEL. Which stations and by what criteria are not known.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
50	*	St. Lawrence River	SLCA		Sediment Quality Criteria - Toxic Effects Threshold					Environment Canada 1992

This value of 50 ppb is supposed to be the 90 % effect level for the parent DDT according to the SLC method. That is, 50 ppb of total DDT in sediments is associated with an effect on biota in 90 % of those sediments. Any one or more of many hundreds of chemicals potentially present in those same toxic sediments could have accounted for the measured toxicity.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
50.7 +/-119	NE	DuPage River Basin, IL	COA		High biotic integrity (6.02 +/-0.47;	Macroinvertebrates				IEPA 1988b

There are 21 stations with a mean total DDT of 48.5 ppb (using one-half of the detection limit of 10 ppb for stations with nondetectable DDT). For all 21 stations, the average MBI was 6.1. Since the numbers are slightly different, one assumes a subset of the 21 stations were used for the TEL. Which stations and by what criteria are not specified. Of interest is that the station with 540 ppb total DDT had an MBI of 6.3 and the next highest station at 120 ppb DDT had an MBI of 6.1. Obviously, DDT at these levels in sediments is not impacting the MBI.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
120	*	Ontario	SLCA		OMOE Provincial SQGs - Severe effect level			1		Persaud et al. 1991

This value of 120 ppb is supposed to be the 95 % effect level for the parent DDT according to the SLC method. Sediment residues of DDT are normalized to 1 % organic carbon. The severe effect level is defined as that level "...that could potentially eliminate most of the benthic organisms." Any one or more of many hundreds of chemicals potentially present in those same toxic sediments could have accounted for the measured toxicity. The observation of apparently healthy benthic communities at sediment residue levels in excess of 120 ppb certainly puts in question the concept of severe effect level for DDT by the SLC method.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
200	NE	Laboratory	SSBA	10-d	Not toxic (15% mortality)	Hyalella azteca (amphipod)	JUV/ADT	3		Schuytema et al. 1989

This value is one-half the detection limit of the unspiked control sediment used to determine the LC-50 of DDT in the amphipod *Hyalella azteca.* Organic carbon was measured at 3 %.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
222 +/-282	*	DuPage River Basin, IL	COA		Low taxa (6.67+/-2.5 S)	Benthic species				IEPA 1988b

This DDT level is the average of three stations with the lowest taxa. These three stations were also polluted by several other contaminants other than DDT. For example, Station GBL-08 sediments contained 270 ppm lead and 3.9 ppm mercury. This station also contained the highest sediment concentration of DDT at 540 ppb.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1300	NE	Laboratory	SSBA	10-d	Not toxic (2.5% mortality)	Hyalella azteca (amphipod)	JUV/ADT	11		Schuytema et al. 1989
1800	NE	Laboratory	SSBA	10-d	Not toxic (2.5% mortality)	Hyalella azteca (amphipod)	JUV/ADT	3		Schuytema et al. 1989
3783		PEL								
4200 +/-125	*	Laboratory	SSBA	10-d	LC50	Hyalella azteca (amphipod)	JUV/ADT	3		Schuytema et al. 1989
4800	*	Laboratory	SSBA	10-d	LC50	Hyalella azteca (amphipod)	JUV/ADT	3		Schuytema et al. 1989

The 1,300 ppb and 1,800 ppb values are spiked sediments used to determine the LC-50 of DDT in *Hyalella azteca.* These levels did not measurably affect the survival of this amphipod crustacean. The value of 4,200 ppb is the calculated LC-50 from the dose-response data. The value of 4,800 ppb is one half of the lowest dose in the first trial. This dose level killed 39/40 (97.5 %) of the test organisms.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5300	NE	Laboratory	SSBA	10-d	Not toxic (6.67% mortality)	Hyalella azteca (amphipod)	2-mo	3		Nebecker et al. 1989
5800	NE	Laboratory	SSBA	10-d	Not toxic (10% mortality)	Hyalella azteca (amphipod)	2-mo	7.2		Nebecker et al. 1989
11000 +/-650	*	Laboratory	SSBA	10-d	LC50	Hyalella azteca (amphipod)	2-mo	3		Nebecker et al. 1989

These data points are from a companion study to the Schuytema, et al (1989) study. The same sediments were spiked with DDT. LC-50 was determined in *Hyalella azteca.* The 11,000 value is the 10 day LC-50 at 3 % organic carbon.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
11100 +/-190	*	Laboratory	SSBA	10-d	LC50	Hyalella azteca (amphipod)	JUV/ADT	11		Schuytema et al. 1989
16100	*	Laboratory	SSBA	10-d	LC100	Hyalella azteca (amphipod)	JUV/ADT	3		Schuytema et al. 1989
16100	*	Laboratory	SSBA	10-d	LC100	Hyalella azteca (amphipod)	JUV/ADT	3		Schuytema et al. 1989

The 11,100 ppb value is the 10 day LC-50 at 11 % organic carbon.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
19600 +/-2180	*	Laboratory	SSBA	10-d	LC50	Hyalella azteca (amphipod)	2-mo	7.2		Nebecker et al. 1989
21100	*	Laboratory	SSBA	10-d	Toxic (86.7% mortality)	Hyalella azteca (amphipod)	2-mo	3		Nebecker et al. 1989
22100	NE	Laboratory	SSBA	10-d	Not toxic (5% mortality)	Hyalella azteca (amphipod)	2-mo	10.5		Nebecker et al. 1989
30600	*	Laboratory	SSBA	10-d	Toxic (65% mortality)	Hyalella azteca (amphipod)	2-mo	7.2		Nebecker et al. 1989
46300	*	Laboratory	SSBA	10-d	Toxic (48.3% mortality)	Hyalella azteca (amphipod)	2-mo	10.5		Nebecker et al. 1989
49600	*	Laboratory	SSBA	10-d	LC100	Hyalella azteca (amphipod)	2-mo	3		Nebecker et al. 1989
49700 +/-3030	*	Laboratory	SSBA	10-d	LC50	Hyalella azteca (amphipod)	2-mo	10.5		Nebecker et al. 1989

The study found that organic carbon was inversely related to the LC-50. The 22,100 ppb value at 10.5 % organic carbon did not produce significant mortality.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
71300	*	Laboratory	SSBA	10-d	LC100	Hyalella azteca (amphipod)	JUV/ADT	11		Schuytema et al. 1989

This level represents a lethal concentration of DDT in sediment.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
88400	*	Laboratory	SSBA	10-d	LC100	Hyalella azteca (amphipod)	2-mo	7.2		Nebecker et al. 1989
198000	*	Laboratory	SSBA	10-d	LC100	Hyalella azteca (amphipod)	2-mo	10.5		Nebecker et al. 1989

These levels represent lethal concentrations of DDT in sediment.

ANALYSIS OF THE FRESH WATER SEDIMENT TEL

A variety of data types are listed in the data set from which the TEL is calculated. Some sediment residue levels are considered to be background levels found in relatively unpolluted and nontoxic sediments; some are levels associated with toxic sediments; some are calculated from water column criteria and equilibrium constants; some represent true dose-response from bioassays of spiked sediments. All of these data types should be considered in the determination of a sediment threshold for DDT toxicity. However, the TEL does not appropriately weigh the quality of the various data points. Outdated equilibrium constants are included and should be removed or replaced with more accurate constants based on the slow-stir methodology (deBruijn et al., 1989). Effects associated with relatively low concentrations of DDT are included even though orders of magnitude higher concentrations of DDT in sediments are without effect for the same biological endpoint. Bioassay data are given the same weight as all other data even though bioassay data are the only data type representing true dose-response. Probably the most relevant data points of all, toxicity thresholds from bioassay data using spiked sediments, are under-weighted in the determination of TELs. Other troubling observations are the omission of data (even within the same studies), repeated use of the same data in different data points, the inconsistent correction for organic carbon, and the use of data for just the parent compound in the determination of the TEL for total DDT. The only data points that address the issue of bioaccumulation beyond benthic organisms are the equilibrium derived data points, but these appear to all have used older Kocs that underestimate sediment thresholds.

Based on the toxicity threshold of several thousand ppb in amphipod toxicity assays, the freshwater TEL of 6.98 for total DDT is likely to be more than two orders of magnitude below the threshold for benthic organisms. Even if one were to use the TEL methodology and throw out the outdated and illogical data points (e.g., where known toxic levels of other chemicals are present, where higher concentrations were without effect, and where outdated equilibrium constants were used), the TEL for total DDT in fresh water sediments would be an order of magnitude higher than 6.98 ppb. Use of the freshwater TEL for DDT by the SARWQCB represents bad science that greatly underestimates a scientifically appropriate sediment target. The consequence of this erroneous and unjustified sediment target is the waste of resources appied to a nonproblem when those resources could be used to address known toxicity in the Watershed.

MARINE SEDIMENT TEL

Table SW-1. A summary of the available data on the biological effects associated with sediment-sorbed TOTAL DDT (ppb) used to support the derivation of sediment quality guidelines for marine and estuarine ecosystems.

Total DDT Conc.+/-SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
0.4	*	United States	EqPA		99% Chronic Marine Criteria	Aquatic organisms		1		Pavlou et al. 1987
0.7	*	United States	EqPA		95% Chronic Marine Criteria	Aquatic organisms		1		Pavlou et al. 1987

Permissible sediment contaminant concentrations were derived from the equilibrium between water and sediment organic carbon using a Koc and the National criterion in water. The logKoc for parent DDT is a mean of 5.52 derived from several values. The logKoc for DDE is a mean of 5.17. The 95th percentile of the distribution of Kocs is two orders of magnitude (a 100-fold) lower than values obtained from the superior slow-stir method. The sediment value has been normalized to 1 % organic carbon. These data points have been superceded by superior methods for determining Koc.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
0.8	NE	San Francisco Bay, CA	COA	48-h	Least toxic (17.3% mortality)	Mussel	LAR	1.25		Chapman et al. 1987a
0.9 +/-0.42	SG	San Francisco Bay, CA	COA	48-h	Moderately toxic (57.1+/-13.6% mortality)	Mussel	LAR	1.14+/-0.33		Chapman et al. 1987a
1.04 +/-0.35	NE	San Francisco Bay, CA	COA	4-wk	Least toxic (116+/-4.3 young produced)	Tigriopus californicus (copepod)	ADT	1.23+/-0.09		Chapman et al. 1987a
1.08 +/-0.618	NE	San Francisco Bay, CA	COA	48-h	Least toxic (18+/-8.01% abnormal)	Mussel	LAR	1.2+/-0.38		Chapman et al. 1987a
1.27 +/-1.08	NE	San Francisco Bay, CA	COA	10-d	Least toxic (13.6+/-7.76% mortality)	Amphipod	ADT	1.4+/-0.79		Chapman et al. 1987a
1.36 +/-0.77	SG	San Francisco Bay, CA	COA	48-h	Moderately toxic (25.1+/-6.61% abnormal)	Mussel	LAR	1.26+/-0.17		Chapman et al. 1987a
1.39 +/-1.06	NE	San Francisco Bay, CA	COA	10-d	Least Toxic (4.63+/-2.91% avoidance)	Amphipod	ADT	1.44+/-0.74		Chapman et al. 1987a

Copper, lead, mercury and hydrocarbon contamination of these sediments is a more plausible cause of the observed toxicity than DDT.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1.5	*	United States	EqPA		Chronic Marine EqP Threshold	Aquatic biota		1		Bolton et al. 1985

Table 2.1 in Bolton et al. lists threshold contamination concentrations for sediments based on 4 % organic carbon and the equilibrium between organic carbon and water. The threshold in water is the chronic National criterion. The organic carbon was corrected to 1 % for this data point. Only DDT was included. Total DDT from this study was not determined and used for this data point. The threshold concentration for DDE (the predominant form of DDT in the environment) in this study was 28 ppm! The problem with this data set is the apparent use of old Koc values that give inaccurate estimates of the partition of DDTs between sediment organic carbon and water. The likely threshold concentration for DDT is higher than 1.5 ppb at 1 % organic carbon, and lower for DDE and DDD at 7,000 ppb and 3,250 ppb, respectively.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1.5 +/-0.408	NE	Puget Sound, WA	COA	15-min	Not significantly toxic (EC50; 0.283+/- 0.168% extract)	Microtox (Photobacterium phosphoreum)		1.39 +/-0.37		Pastorok & Becker 1990

This data point is the mean DDT level in sediments from less polluted reference sites in Puget Sound. These sediments were used as the controls in the microtox bioassay.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1.58	*	United States	EqPA		Marine Chronic Sediment Criteria	Aquatic biota		1		JRB Associates 1984

The data point refers to parent DDT and not total DDT. The sediment equilibrium concentration is derived from a logKow of 5.98. The slow-stir logKow for DDT reported by deBruijn et al. (1989) is 6.914. The Kow derived by the superior slow-stir method gives a sediment criteria almost an order of magnitude higher, using the formula in JRB Associates (1984).

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1.6	*	United States	EqPA		EPA Chronic Marine EP Threshold	Aquatic biota		1		Lyman et al. 1987

This value is cited as coming from the JRB Associates (1984) reference just above. Apparently, Lyman et al. have rounded the JRB Associates value of 1.58 ppb to 1.6 ppb. These two values are essentially the same. The Kow derived by the superior slow-stir method gives a sediment criterion almost an order of magnitude higher, using the formula in JRB Associates (1984).

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
2	NC	Puget Sound, WA	COA	2-d	Significantly toxic (3.8% abnormal chromosome)	Dendraster excentricus (echinoderm)	EMB	1.5		Pastorok & Becker 1990

This data point was measured in sediment from Commencement Bay diluted 10-fold with reference sediments from relatively unpolluted areas of Puget Sound. The value of 2 ppb represents one-half the detection limit for total DDT in a sediment sample in which total DDT was not detected. The undiluted sediment did not produce a significant increase in abnormal chromosomes. The 10-fold diluted sediment data point should not be used since the undiluted sediment is nontoxic in the same bioassay. Numerous other contaminants were present in these

sediments and are more likely to have caused toxicity than any DDT that may have been present below the detection limit. In addition, DDT is not known to cause chromosomal abnormalities.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
2.18 +/-1.45	SG	San Francisco Bay, CA	COA	10-d	Moderately toxic (28.3+/-7.51% mortality)	Amphipod	ADT	2.01+/-0.98		Chapman et al. 1987a
2.92 +/-0.68	*	San Francisco Bay, CA	COA	48-h	Highly toxic (92.3+/-5.5% mortality)	Mussel	LAR	2.87+/-1.32		Chapman et al. 1987a
2.92 +/-0.68	*	San Francisco Bay, CA	COA	4-wk	Moderately toxic (94.9+/-10.1 young produced)	Tigriopus californicus (copepod)	ADT	2.87+/-1.07		Chapman et al. 1987a
2.93	*	San Francisco Bay, CA	COA	10-d	Most toxic (95% mortality)	Amphipod	ADT	4.03		Chapman et al. 1987a
2.93	*	San Francisco Bay, CA	COA	10-d	Highly toxic (37% avoidance)	Amphipod	ADT	4.03		Chapman et al. 1987a
3.27	*	San Francisco Bay, CA	COA	48-h	Highly toxic (66.8% abnormal)	Mussel	LAR	3.59		Chapman et al. 1987a

Copper, lead, mercury and hydrocarbon contamination of these sediments is a more plausible cause of the observed toxicity than DDT.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
3.42 +/-2.87	NE	Puget Sound, WA	COA	2-d	Not significantly toxic (6.67+/-8.07% abnormal development)	Dendraster excentricus (echinoderm)	EMB	1.51+/-0.330		Pastorok & Becker 1990
3.89		TEL								

The 3.42 ppb data point is the average of three reference sediments, one sediment from Commencement Bay, and two dilutions of the Commencement Bay sediment. None of the six sediment samples caused abnormal development in *Dendraster excentricus*.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
4.6 +/-3.39	NE	Tampa Bay, FL	COA	1-h	Least toxic (79.4+/-9.9% fertilization)	Arbacia punctulata (sea urchin)	GAM	1.45+/-0.587		Long 1993

For the 11 stations without a significant effect on fertilization of sea urchin eggs ($p<0.1$), the mean total DDT was 4.44 ppb.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
4.94 +/-3.56	NE	Puget Sound, WA	COA	10-d	Not significantly toxic (13.8+/-4.09% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.47+/-0.306		Pastorok & Becker 1990

The data point is the average of six dilutions of sediments from two polluted locations and three undiluted reference sediments from Puget Sound. All nine sediment samples were nontoxic in the amphipod mortality bioassay.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5	NC	Galveston Bay, TX	COA	1-h	Toxic (20.4+/-18.9% fertilization)	Arbacia punctulata (sea urchin)	EMB	1.26+/-0.47	14.6+/-10.1	Carr 1992
5	NC	Galveston Bay, TX	COA	48-h	Toxic (4.65+/-16.1% normal development)	Arbacia punctulata (sea urchin)	EMB	1.06+/-0.449	14.5+/-9.2	Carr 1992
5	NC	Galveston Bay, TX	COA		Low abundance (2.05+/-1.58 N 0.00203 sq.m.)	Copepoda		0.879+/-0.47	9.63+/-9.65	Carr 1992

The values of 5 ppb are one-half the detection limit for sediments in which DDT was not detected. The major contaminants in these sediments were polycyclic aromatic hydrocarbons from the oil production and refining in Galveston Bay.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5	NE	Puget Sound, WA	SBA		EPA/ACOE Puget Sound Interim	Aquatic biota				USACOE 1988

The value of 5 ppb is the analytical method limit of quantitation or five times the detection limit. Para, para isomers of DDD, DDE and DDT make up the total DDT.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5	NE	Galveston Bay, TX	COA		High species richness (24.5 +/-3.7 S/0.00203 sq.m.)	Benthic species		1.36 +/-0.66	1.2 +/-0.39	Carr 1992

5	NE	Galveston Bay, TX	COA		High abundance (359+/-92.8 N/0.00203 sq.m.)	Benthic species		1.36+/-0.66	1.2+/-0.39	Carr 1992
5	NE	Galveston Bay, TX	COA		High abundance (156+/-22.9 N/0.00203 sq.m.)	Polychaeta		0.916+/-0.661	2.94+/-2.3	Carr 1992
5	NE	Galveston Bay, TX	COA		High abundance (155+/-49.5 N/0.00203 sq.m.)	Oligochaeta		1.2+/-1.27	4.27+/-3.85	Carr 1992
5	NE	Galveston Bay, TX	COA		High abundance (27.3+/-10 N/0.00203 sq.m.)	Mollusca		1.64+/-0.4	1.39+/-0.17	Carr 1992
5	NE	Galveston Bay, TX	COA		High Abundance (6+/-1.41 N/0.00203 sq.m.)	Amphipoda		0.955+/-0.629	7.21+/-8.2	Carr 1992
5	NE	Galveston Bay, TX	COA		Low species (11.2+/-1.94 S/0.00203	Benthic species		0.642+/-0.356	13.4+/-8.65	Carr 1992
5	NE	Galveston Bay, TX	COA		High abundance (154+/-30.2 N/0.00203 sq.m.)	Benthic invertebrates		0.47+/-0.27	9.07+/-2.94	Carr 1992
5	NG	Galveston Bay, TX	COA		Low abundance (4.21+/-5.66 N/0.00203 sq.m.)	Oligochaeta		0.895+/-0.453	7.85+/-8.8	Carr 1992
5	NG	Galveston Bay, TX	COA		Low abundance (53+/-33.9 N/0.00203 sq.m.)	Benthic invertebrates		0.985+/-0.247	22+/-11.2	Carr 1992
5.17 +/-0.93	SG	Galveston Bay, TX	COA		Low abundance (1.86+/-2.59 N/0.00203 sq.m.)	Mollusca		0.784+/-0.421	8.29+/-8.13	Carr 1992
5.17 +/-0.913	SG	Galveston Bay, TX	COA		Low abundance (0.3+/-0.651 N/0.00203 sq.m.)	Amphipoda		0.859+/-0.487	7.67+/-8.12	Carr 1992
5.18 +/-0.945	NE	Galveston Bay, TX	COA	1-h	Not toxic (92.5+/-6.9% fertilization)	Arbacia punctulata (sea urchin)	EMB	0.77+/-0.448	6.37+/-6.58	Carr 1992
5.18 +/-0.945	SG	Galveston Bay, TX	COA		Low species richness (10+/-3.73 S/0.00203 sq.m.)	Benthic species		0.795+/-0.425	8.56+/-8.14	Carr 1992

Total DDT Conc.+/-SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
5.21 +/-1.02	SG	Galveston Bay, TX	COA		Low abundance (41.7+/-21.8 N/0.00203 sq.m.)	Polychaeta		0.848+/-0.427	9.21+/-8.61	Carr 1992
5.23 +/-1.07	NE	Galveston Bay, TX	COA	48-h	Not toxic (98.1+/-1.79% normal development)	Arbacia punctulata (sea urchin)	EMB	0.748+/-0.476	4.16+/-3.45	Carr 1992
5.38 +/-1.39	NE	Galveston Bay, TX	COA		High abundance (16.2+/-6.19 N/0.00203 sq.m.)	Copepoda		0.844+/-0.524	4.73+/-3.1	Carr 1992
5.83 +/-2.04	SG	Galveston Bay, TX	COA		Moderate abundance (58.3+/-16.2 N/0.00203 sq.m.)	Oligochaeta		0.632+/-0.274	7.93+/-5.6	Carr 1992

The values of 5 ppb are one-half the detection limit for sediments in which DDT was not detected. The values of 5.17 to 5.83 ppb are means of mostly one-half the detection limit for sediments in which DDT was not detected; one sample was reported to contain 10 ppb DDT. The major contaminants in these sediments were polycyclic aromatic hydrocarbons from oil production and refining in Galveston Bay.

Total DDT Conc.+/-SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
6.9	NE	Puget Sound, WA	AETA		PSDDA Screening level concentration	Aquatic biota				USACOE 1988

The value of 6.9 ppb is 10 % of the highest apparent effects threshold (HAET) or highest threshold levels for a range of biological indicators. The value is for 4,4` isomers of DDT, DDD and DDE.

Total DDT Conc.+/-SD)	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
8.38 +/-11.9	NE	Puget Sound, WA	COA	20-d	Not significantly toxic (5+/-3.86% mortality)	Neanthes arenaceodentata (polychaete)	EMB	1.46+/-0.26		Pastorok & Becker 1990

The data point is the average of nine dilutions of sediments from three polluted locations and three undiluted reference sediments from Puget Sound. All 12 sediment samples were nontoxic in the polychaete mortality bioassay, including one sediment containing 45 ppb total DDT.

Total DDT Conc.+/-SD)	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
11 +/-7.89	NE	Tampa Bay, FL	COA	10-d	Not significantly toxic (14.6+/-7.67% mortality)	Ampelisca abdita (amphipod)	SUBADT	1.63+/-0.596		Long 1993

This data point is the mean of a subset of sediments from Tampa Bay that were not significantly toxic to *Ampelisca abdita*. The subset is unknown. Fifty three out of 61 sediments that were analyzed for total DDT were not significantly toxic. In a second study, none of the sediments were found to be significantly toxic to *Ampelisca abdita,* including one sediment with 3,802 ppb total DDT.

Total DDT Conc.+/-SD)	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
11.6 +/-16.4	NE	Puget Sound, WA	COA	10-d	Not significantly toxic (4.43+/-2.1% mortality)	Panope generosa (geoduck)	JUV	1.51+/-0.261		Pastorok & Becker 1990
14.1 +/-18	NE	Puget Sound, WA	COA	2-d	Not significantly toxic (2.09+/-1.73% abnormal chromosome)	Dendraster excentricus (echinoderm)	EMB	1.56+/-0.329		Pastorok & Becker 1990
15.4 +/-17.2	*	Puget Sound, WA	COA	15-min	Significantly toxic (EC50; 0.065+/-0.043% extract)	Microtox (Photobacterium phosphoreum)		1.58+/-0.255		Pastorok & Becker 1990
18.8 +/-18.8	*	Puget Sound, WA	COA	2-d	Significantly toxic (60.4+/-46.5% abnormal development)	Dendraster excentricus (echinoderm)	EMB	1.57+/-0.287		Pastorok & Becker 1990

These data points represent various dilutions of sediments from polluted areas of Puget Sound. The 15.4 ppb and 18.8 ppb data points were the average total DDT residues in toxic sediments. These sediments were highly contaminated with metals and hydrocarbons that could well have accounted for the observed toxicity.

Total DDT Conc.+/-SD)	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
22.3	*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	1.92+/-0.085		Word et al. 1987

The value of 22.3 ppb is in error. The author was determining the LC-50 based on DDT concentrations in pore water and not DDT concentrations in sediment. The pore-water concentrations were normalized by the organic carbon content of the sediment. The unnormalized LC-50 in pore water was 4.28 ppb. The normalized LC-50 in pore water was 2.23 ppb. If one were to calculate the LC-50 on a sediment basis, one would have to multiply the LC-50s in pore water by a distribution coefficient for the equilibrium between pore water and sediment. The result would be a much higher LC-50 for sediment than for pore water. For example, a Kd of 30,000 would give a sediment LC-50 of 66,900 ppb. Only parent DDT was studied.

Total DDT Conc.+/-SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
22.5 +/-25.9	NE	Tampa Bay, FL	COA		Not significantly toxic (EC50, 0.066+/-0.033 mg dry wt/ml)	Microtox (Photobacterium phosphoreum)		1.89+/-0.902		Long 1993

This data point is the mean of a subset of sediments from Tampa Bay that were not significantly toxic in the Microtox bioassay. The subset is unknown. One of the nontoxic sediments contained 131 ppb total DDT.

Total DDT Conc.+/-SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
24	*	Northern California	AETA		Northern California AET Values	Benthic species				Becker et al. 1990

This value is the highest toxicity threshold for benthic species for total DDT in sediments from Northern California. The comparable value for Southern California (where sediments have much higher levels of total DDT) is 3,000 ppb! It would seem that the 3,000 ppb value is more relevant.

Total DDT Conc.+/-SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
24.2 +/-21.4	*	Puget Sound, WA	COA	10-d	Significantly toxic (80.8+/-30.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.65+/-0.266		Pastorok & Becker 1990

This data point represents the mean of various dilutions of sediments from two polluted areas of Puget Sound. The 24.2 ppb data point was the average total DDT residues in sediments that were toxic to amphipods. These sediments were highly contaminated with metals and hydrocarbons that could well have accounted for the observed toxicity.

Total DDT Conc.+/-SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
27	*	Northern California	AETA	10-d	Northern California AET Values	Rhepoxynius abronius (amphipod)	ADT			Becker et al. 1990
27	*	California	AETA	48-h	California AET Values	Mytilus edulis (bivalve)	LAR			Becker et al. 1990

The first 27 ppb value is the highest threshold for *Rhepoxynius abronius* toxicity for total DDT in sediments from Northern California. The comparable value for Southern California (where sediments have much higher levels of total DDT) is > 9,300 ppb! The second 27 ppb value is the highest threshold for bivalve toxicity for total DDT in sediments from Northern California. A similar threshold was not determined for Southern California. The AET values for Northern California appear to be artifacts of the method (most likely determined by the presence of toxic levels of other contaminants), since sediments from Southern California with high residues of total DDT were not toxic in the selected bioassays.

Total DDT Conc.+/-SD		Hit Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
27	*	Puget Sound, WA	COA	10-d	Significantly toxic (86% mortality)	Panope generosa (geoduck)	JUV	2.1		Pastorok & Becker 1990

This data point is from a toxic sediment collected in Eagle Harbor in Puget Sound. This site is highly contaminated with metals and hydrocarbons that could well have accounted for the observed toxicity.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
29.7 +/-23.1	*	Puget Sound, WA	COA	20-d	Significantly toxic (37.3+/-22% mortality)	Neanthes arenaceodentata (polychaete)	EMB	1.87+/-0.208		Pastorok & Becker 1990

The data point is the average of three undiluted sediments from three polluted sites in Puget Sound. Numerous other contaminants were present in these sediments and are more likely to have caused toxicity than this level of total DDT.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
33.2 +/-43.5	*	Tampa Bay, FL	COA	1-h	Moderately toxic (15.3+/-10.6% fertilization)	Arbacia punctulata (sea urchin)	GAM	1.94+/-0.908		Long 1993

Moderately toxic is not defined herein, so the subset of sites for this value is unkown. However, if 33.2 ppb total DDT inhibits fertilization of sea urchin eggs 84.7 % (only 15.3 % of the eggs were fertilized), one should take note that sediment from station 18A contained 116 ppb total DDT and was associated with a minimal inhibition of fertilization of 26 % (76 % of the eggs were fertilized). The conclusion that 33.2 ppb total DDT is inhibiting fertilization in these sediments is not toxicologically plausible. Other chemicals are likely causing the toxicity.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
46.1	*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	0.581+/-0.121		Word et al. 1987

The value of 46.1 ppb is in error. The author was determining the LC-50 based on DDT concentrations in pore water and not DDT concentrations in sediment. The pore-water concentrations were normalized by the organic carbon content of the sediment. The unnormalized LC-50 in pore water was 2.67 ppb. The normalized LC-50 in pore water was 4.61 ppb. If one were to calculate the LC-50 on a sediment basis, one would have to multiply the LC-50s in pore water by a distribution coefficient for the equilibrium between pore water and sediment. The result would be a much higher LC-50 for sediment than for pore water. For example, a Kd of 30,000 would give a sediment LC-50 of 138,300 ppb. Only parent DDT was studied.

Total DDT Conc. +/- SD		Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
50 +/-60		NE	Southern California	COA		High abundance (191+/-70.1 N:0.1	Echinoderm				Word & Mearns 1979
51.7			PEL								

The value is the total DDT in superficial sediments taken at 60 meters depth off the coast of Southern California. The value is the mean of a subset that is not specified. Forty two of seventy one stations, from Point Conception to the Mexican border, were sampled for total DDT analysis.

Total DDT Conc. +/- SD		Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
54.5		*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	0.6 +/-0.031		Word et al. 1987
55.2		*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	0.12 +/-0.006		Word et al. 1987

The values of 54.5 and 55.2 ppb are in error. The author was determining amphipod LC-50s based on DDT concentrations in pore water and not DDT concentrations in sediment. The pore-water concentrations were normalized by the organic carbon content of the sediment. The unnormalized LC-50s in pore water were 3.27 and 0.69 ppb. The normalized LC-50s in pore water were 5.45 and 5.52 ppb, respectively. If one were to calculate the LC-50s on a sediment basis, one would have to multiply the LC-50s in pore water by a distribution coefficient for the equilibrium between pore water and sediment. The result would be much higher LC-50s for these sediments than for their pore waters. For example, a Kd of 30,000 would give sediment LC-50s of 163,500 ppb and 165,600 ppb, respectively. Only parent DDT was studied.

Total DDT Conc. +/- SD		Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
68 +/-71.7		NC	Southern California	COA	10-d	Significantly toxicity (51.7% mortality)	Grandidierella japonica	JUV			Anderson et al. 1988

This association between sediment residue and sediment toxicity makes no sense when one considers that in the same study, 1,018 ppb total DDT in sediment was not associated with significant sediment toxicity to the same amphipod species. The authors stated: "Most notably, DDT concentration did not correlate with short-term toxicity or macrofaunal patterns."

Total DDT Conc. +/- SD		Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
69		*	Puget Sound, WA	AETA		PSDDA Maximum Level Criteria	Aquatic biota				USACOE 1988

The value of 69 ppb represents the HAET for a range of biological indicators. That is, 69 ppb is the highest residue of total DDT in sediments that were also found not to be toxic to benthic organisms.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
90 +-130	SG	Southern California	COA		Moderate abundance (56.2 +-23 N 0.1 sq.m.)	Echinoderm				Word & Mearns 1979
100 +-150	NE	Southern California	COA		High abundance (148 +-58 N 0.1	Arthropods				Word & Mearns 1979

The values are the total DDT in superficial sediments taken at 60 meters depth off the coast of Southern California. The values are the means of a subset that is not specified. Forty two of seventy one stations, from Point Conception to the Mexican border, were sampled for DDT analysis. The high abundance of benthic species at 35,300 ppb total DDT suggests that lower concentrations are unlikely to have an effect on abundance of benthic species.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
125	*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	0.25 +-0.01		Word at al. 1987

The value of 125 ppb is in error. The author was determining the LC-50 based on DDT concentrations in pore water and not DDT concentrations in sediment. The pore-water concentrations were normalized by the organic carbon content of the sediment. The unnormalized LC-50 in pore water was 3.13 ppb. The normalized LC-50 in pore water was 12.51 ppb. If one were to calculate the LC-50 on a sediment basis, one would have to multiply the LC-50s in pore water by a distribution coefficient for the equilibrium between pore water and sediment. The result would be a much higher LC-50 for sediment than for pore water. For example, a Kd of 30,000 would give a sediment LC-50 of 375,300 ppb. Only parent DDT was studied.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
210	*	United States	EqPA		EPA Acute Marine EP Threshold	Aquatic biota		1		Lyman et al. 1987

This value is derived in the same way as the 1.58 ppb value by JRB Associates (1984) and the 1.6 ppb value by Lyman et al. (1987). The only difference is the use of the National acute marine criterion instead of the chronic marine criterion. The sediment equilibrium concentration is derived from a logKow of 5.98. The slow-stir logKow reported by deBruijn et al. (1989) is 6.914. The Kow derived by the superior slow-stir method gives a sediment acute marine threshold nearly an order of magnitude higher at 1 % organic carbon, using the formula in JRB Associates (1984).

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
210 +/-490	NC	Southern California	COA		Moderate abundance (75.6+/-12.7 N-0.1 sq.m.)	Benthic species				Word & Mearns 1979
250 +/-620	NC	Southern California	COA		Moderate species richness (72+/-3.3 S-0.1 sq.m.)	Benthic species				Word & Mearns 1979
350 +/-710	*	Southern California	COA		Moderate abundance (72.6 +/-6.8 N-0.1 sq.m.)	Arthropods				Word & Mearns 1979

The values are the total DDT in superficial sediments taken at 60 meters depth off the coast of Southern California. The values are the mean of a subset that is not specified. Forty two of seventy one stations, from Point Conception to the Mexican border, were sampled for DDT analysis. The data set is dramatically influenced by the very high level of pollutants coming out of the Los Angeles County outfall off the Palos Verdes Penninsula. In addition to high levels of DDT, high levels of metals and other contamininants were measured in these particular sediments. Contaminants other than DDT may well be affecting the abundance of benthic species. This point is further supported by the finding of high abundance of benthic species at 35,300 ppb total DDT, a finding that suggests that lower concentrations are unlikely to have an effect on abundance of benthic species.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
432 +/-1125	*	Tampa Bay, FL	COA	1-h	Most toxic (0.091+/-0.187*s	Arbacia punctulata (sea urchin)	GAM	2.96+/-1.49		Long 1993

This data point is not in the Long et al. (1993) reference. The subset of data used to obtain this value is not given. Seven sites were described as most toxic to fertilization of sea urchin eggs. Of these, 9 samples were analyzed for total DDT. For one of these sites, toxicity was attributed to ammonia. The mean of the remaining 8 samples was 588 ppb total DDT. Within these 8 samples, a dose-response for total DDT is not apparent. For example, at a four-fold dilution of pore water, the highest total DDT level of 3,800 ppb was associated with 45 % fertilization and a sample with 134.2 ppb total DDT was associtated with 2.4 % fertilization.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
505	*	United States	SLCA		National Screening Level Concentration Marine	Benthic species		1		Neff et al. 1987

Using a method similar to the one estimating this data point, Neff et al. (1986) derived a screening level concentration for fresh water of 1.9 ppb. How can fresh and salt water screening levels differ by 265-fold when the toxicity of DDT to fresh and marine benthic organisms is similar? One or both of the screeing levels are most likely in error. Based on bioassay results, the freshwater screening level is too low.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
596 +/-1302	*	Tampa Bay, FL	COA	10-d	Significantly toxic (35.2+/-17.5% mortality)	Ampelisca abdita (amphipod)	SUBADT	3.53+/-1.35		Long 1993
665 +/-1391	*	Tampa Bay, FL	COA		Significantly toxic (EC50; 0.017+/-	Microtox (Photobacterium		3.47+/-1.49		Long 1993

The 596 ppb value is listed in Table 30. The footnote to Table 30 references sediment LC-50s of 2,500 ppb and 1,040 ppb for two amphipods, *Eohaustorius estuarius* and *Rhepoxynius abronius*, respectively. The 665 ppb value is listed in Table 34. The footnote to Table 34 references a sediment LC-50 of 2,500 ppb in the amphipod, *Eohaustorius estuarius*.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1018 +/-2424	NE	Southern California	COA	10-d	Not significantly toxic (23.2%	Grandidierella japonica	JUV			Anderson et al. 1988

Reburial and survival of amphipods was not significantly affected by sediments from the Palos Verdes site. Total DDT concentration in the Palos Verdes sediment sample was 5,966 ppb.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
1410 +/-5440	NC	Southern California	COA		Low abundance (57.6+/-13.6 N/0.1	Benthic species				Word & Mearns 1979
2170 +/-7190	NE	Southern California	COA		High species richness (96.3+/-22.3 S/0.1 sq.m.)	Benthic species				Word & Mearns 1979

The values are the total DDT in superficial sediments taken at 60 meters depth off the coast of Southern California. The values are the mean of a subset that is not specified. Forty two of seventy one stations, from Point Conception to the Mexican border, were sampled for DDT analysis. The data set is dramatically influenced by the very high level of pollutants coming out of the Los Angeles County outfall off the Palos Verdes Penninsula. In addition to high levels of DDT, high levels of metals and other contamininants were measured in these particular sediments. Contaminants other than DDT may well be affecting the abundance of benthic species. The high abundance of benthic species at 35,300 ppb total DDT suggests that lower concentrations are unlikely to have an effect on abundance of benthic species.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
3000	*	California	AETA		California AET Values	Benthic species				Becker et al. 1990
3000	*	Southern California	AETA		Southern California AET Values	Benthic species				Becker et al. 1990
>9300	-	California	AETA	10-d	California AET Values	Rhepoxynius abronius (amphipod)	ADT			Becker et al. 1990
>9300	-	Southern California	AETA	10-d	Southern California AET Values	Rhepoxynius abronius (amphipod)	ADT			Becker et al. 1990

An AET of 27 ppb total DDT was determined for mortality in amphipods from Northern California. A bivalve AET of 27 ppb for total DDT was determined for Southern California. An AET of 24 ppb total DDT was determined for benthic species from Northern California. The inconsistent values suggests that the AET approach is misleading and inappropriate. None of the AET values were corrected for organic carbon.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
13420 +/-37670	*	Southern California	COA		Low abundance (35.3+/-15.8 N/0.1 sq.m.)	Arthropods				Word & Mearns 1979
14190 +/-40200	*	Southern California	COA		Low species richness (51.2+/-8.6 S/0.1 sq.m.)	Benthic species				Word & Mearns 1979

The values are the total DDT in superficial sediments taken at 60 meters depth off the coast of Southern California. The values are the mean of a subset that is not specified. Forty two of seventy one stations, from Point Conception to the Mexican border, were sampled for DDT analysis. The data set is dramatically influenced by the very high level of pollutants coming out of the Los Angeles County outfall off the Palos Verdes Penninsula. In addition to high levels of total DDT, high levels of metals and other contamininants were measured in these particular sediments. Contaminants other than DDT may well be affecting the abundance of benthic species. The high abundance of benthic species at 35,300 ppb total DDT suggests that lower concentrations are unlikely to have an effect on abundance of benthic species.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
16500	*	Laboratory	SSBA	288-h	LC0	Nereis virens (sandworm)		2		McLeese et al. 1982

Marine worms that live in sediment appeared to be in excellent condition with normal burrowing behavior after 288 hours of exposure to sediments containing 16,500 ppb DDT.

Total DDT Conc. +/- SD	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	AVS (umol/g)	Reference
18260 +/-43080	*	Southern California	COA		Low abundance (6.1+/-7.2 N/0.1 sq.m.)	Echinoderm				Word & Mearns 1979
35300 +/-59540	NE	Southern California	COA		High abundance (88.9+/-35.4 N/0.1	Benthic species				Word & Mearns 1979

The values are the total DDT in superficial sediments taken at 60 meters depth off the coast of Southern California. The values are the mean of a subset that is not specified. Forty two of seventy one stations, from Point Conception to the Mexican border, were sampled for DDT analysis. The data set is dramatically influenced by the very high level of pollutants coming out of the Los Angeles County outfall off the Palos Verdes Penninsula. In addition to high levels of DDT, high levels of metals and other contamininants were measured in these particular sediments. Contaminants other than DDT may well be affecting the abundance of benthic species. The high abundance of benthic species at 35,300 ppb total DDT suggests that lower concentrations are unlikely to have an effect on abundance of benthic species.

ANALYSIS OF THE MARINE SEDIMENT TEL

A variety of data types are listed in the data set from which the TEL is calculated. Some sediment residue levels are considered to be background levels found in relatively unpolluted and nontoxic sediments; some are levels associated with toxic sediments; some are calculated from water column criteria and equilibrium constants; some represent true dose-response from bioassays of spiked sediments. All of these data types should be considered in the determination of a sediment threshold for DDT toxicity. However, the TEL does not appropriately weigh the quality of the various data points. Outdated equilibrium constants are included and should be removed. Effects associated with relatively low concentrations of DDT are included even though several orders of magnitude higher concentrations of DDT in sediments are without effect for the same biological endpoint. Bioassay data using spiked sediments is given the same weight as all other data even though this type of bioassay data is the only data type representing true dose-response. Probably the most relevant data points of all, toxicity thresholds from bioassay data using spiked sediments, are under-weighted in the determination of TELs. Other troubling observation are the omission of data (even within the same studies), repeated use of the same data in different data sets, the inconsistent correction for organic carbon, and the use of data for just the parent compound in the determination of the TEL for total DDT. The only data points that address the issue of bioaccumulation to trophic levels higher than benthic organisms are the equilibrium derived data points, but these appear to all have used older Kocs that underestimate sediment thresholds. The misinterpretation of pore water LC50s as sediment LC50s has created very large errors in the effects data set.

Based on the lack of toxicity of sandworms to 16,500 ppb total DDT in sediment, the lack of amphipod toxicity at 5,960 ppb and high benthic species abundance at 35,300 ppb, the marine TEL of 3.89 for total DDT is likely to be several orders of magnitude below the toxicity threshold for benthic organisms. Even if one were to use the TEL methodology and throw out the outdated and illogical data points (e.g., where errors in interpretation occurred, where known toxic levels of other chemicals are present, where higher concentrations were without effect, and where outdated equilibrium constants were used), the TEL would be an order of magnitude higher than 3.89 ppb. Use of the marine TEL for DDT by the SARWQCB represents bad science that greatly underestimates a scientifically appropriate sediment target. The consequence of this erroneous and unjustified sediment target is the waste of resources applied to a nonproblem when those resources could be used to address known toxicity in the Watershed.

CONCLUSIONS

- The TEL determination relies primarily on the association of DDT and toxicity in in the same sediments, rather than a true dose-response.

- In many of the toxic sediments containing DDT, the toxicity can be explained by the presence of other contaminants.

- The data sets used to derive the TELs are flawed due to errors in interpretation of data, use of outdated Kocs and Kows, arbitrary selection of data, repeated use of the same data points, use of parent DDT instead of total DDT, inconsistent correction for organic carbon, and use of low residue level effects where much higher levels are without effect.

- If the flaws in the data sets were corrected, the TELs would be much higher.

- Sediments spiked with DDT have toxicity thresholds in benthic organisms in excess of 1,000 ppb.

- The toxicity threshold for total DDT in freshwater and marine sediments to benthic organisms appears to be more than two orders of magnitude higher than the TELs proposed by EPA and SARWQCB for Newport Bay and San Diego Creek.

- Use of TELs for DDT by the SARWQCB represents bad science that greatly underestimates a scientifically appropriate sediment target. The consequence of these erroneous and unjustified sediment targets is the waste of resources applied to a nonproblem when those resources could be used to address known toxicity in the Watershed.

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Appendix G: Dr. James L. Byard, "Scientific Commentary on the Canadian Tissue Residue Guideline for DDT"

SCIENTIFIC COMMENTARY ON THE CANADIAN TISSUE RESIDUE GUIDELINE FOR DDT

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August 21, 2006

SUMMARY

Environment Canada has developed a fish tissue residue guideline (fish TRG) for total DDT for the protection of sensitive fish-eating avian species. Canadian environmental agencies have also published a Protocol document that was used in developing the DDT fish TRG. Canada ignored dose-response studies in a raptor, the sparrow hawk, and chose less sensitive ducks and shell thinning instead of hatching failure as the basis for the TRG. Canada also chose Wilson's storm petrel to achieve the highest estimate of food intake rate. Petrels are much less sensitive to DDE than sensitive species such as the osprey and, therefore, are inappropriate for estimating the maximum rate of food intake of sensitive species. Using the Canadian protocol procedures, the dose-response in the sparrow hawk, the threshold for hatching failure, and the rate of food intake of the osprey, the fish TRG calculates to 250 ppb total DDT, a value 18 times greater than the 14 ppb recommended by Environment Canada.

INTRODUCTION

In 2000, Environment Canada published Environmental Quality Assessments for PCBs, DDT and Toxaphene. The Assessment document contains the derivation of a Canadian tissue residue guideline (TRG) for total DDT. The TRG for fish was intended to protect avian species from the reproductive effects of DDE. The TRG is based on low-observed-effect-levels (LOELs) for shell thinning in mallard and black ducks. Several generic assumptions were made to arrive at the TRG of 14 ppb in fish as shown in the text of the Assessments document as follows.

> For birds exposed to DDT, the most sensitive endpoint appears to be eggshell thinning and associated reproductive impairment. The most sensitive LOAEL determined from the avian dataset was 0.3 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$. The same LOAEL was determined from several studies on mallard ducks and black ducks. Eggshell thinning occurred when mallard ducks were fed 0.3 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$ of *p,p'*-DDT for 30 days (Kolaja 1977), 0.3 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$ of *p,p'*-DDE for 105 days (Vangilder and Peterle 1980), for 30 days (Kolaja 1977), and for 365 days (Heath

et al. 1969). Black ducks showed a reduction in eggshell thickness when administered 0.3 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$ of *p,p'*-DDE for 136 days (Loncore *et al.* 1971). The NOAEL was assumed to be 0 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$. For the purpose of calculating the TDI, the LOAEL was divided by 5.6 (according to CCME 1993) to estimate a NOAEL of 0.054 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$.

According to Sample *et al.* (1996), avian studies where exposure duration is 10 weeks or less are considered to be sub-chronic, and those where the exposure duration is greater than 10 weeks are considered chronic studies. Several studies on the reproductive effects of DDT in birds were carried out for longer than 10 weeks, therefore these studies were considered to be chronic. Although no data were located on the carcinogenic or mutagenic effects of DDT in avian species, a large quantity of data exists on the effects of DDT to several avian species, including those known to be sensitive to the reproductive effects of DDT such as raptors. Therefore, an UF of 10 (CCME 1997) was used to account for differences in interspecies sensitivities. The LOAEL of 0.30 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$ was used in conjunction with the NOAEL of 0.054 $mg \cdot kg^{-1}$ $bw \cdot day^{-1}$ to calculate an avian TDI of 13.0 $\mu g \cdot kg^{-1}$ $bw \cdot day^{-1}$ for total DDT.

$$TDI = (0.30\ mg \cdot kg^{-1}\ bw \cdot day^{-1} \bullet 0.054\ mg \cdot kg^{-1}\ bw \cdot day^{-1})^{0.5} \div 10$$
$$TDI = 0.013\ mg \cdot kg^{-1}\ bw \cdot day^{-1} = 13.0\ \mu g \cdot kg^{-1}\ bw \cdot day^{-1}$$

The mammalian and avian TDIs were then used in conjunction with the body weights (BW) and daily food intake rates (FI) of the wildlife species with the highest FI:BW ratios to calculate reference concentrations (RCs) of total DDT, using the following equation:

$$RC = TDI \bullet (BW \div FI)$$

where:
RC = Reference concentration ($mg \cdot kg^{-1}$ ww);
TDI = Tolerable daily intake ($mg \cdot kg^{-1}$ $bw \cdot day^{-1}$);
BW = Body weight (kg ww);
FI = Food intake rate (kg $ww \cdot day^{-1}$)

Among mammalian and avian wildlife species, female mink (*Mustela vison*) and Wilson's storm-petrel (*Oceanites oceanicus*) have the highest potential exposure to DDT due to their high FI:BW ratios (0.24 and 0.94, respectively) (CCME 1997). Therefore, these species were used to calculate the RCs for total DDT.

Similarly, a RC of 14.0 $\mu g \cdot kg^{-1}$ was calculated for Wilson's storm-petrel, assuming a body weight of 0.032 kg, an average daily food intake rate of 0.03 kg $ww \cdot day^{-1}$, and a TDI of 13.0 $\mu g \cdot kg^{-1}$ $bw \cdot day^{-1}$ for birds (Dunning 1993).

$$RC = 13.0\ \mu g \cdot kg^{-1}\ bw \cdot day^{-1} \bullet (0.032\ kg \div 0.030\ kg\ ww \cdot day^{-1})$$
$$RC = 14.0\ \mu g \cdot kg^{-1}$$

The lower of the mammalian and avian RCs, 14.0 $\mu g \cdot kg^{-1}$ was recommended as the Canadian TRG for total DDT for the protection of freshwater, marine, and estuarine wildlife that consume aquatic biota.

These assumptions were based on a Protocol document developed by Canadian environmental agencies.

PROTOCOL DOCUMENT

The procedures for deriving the TRG for DDT in fish were from a report published by the Canadian Council of Ministers of the Environment (1999). The Protocol document calls for the use of: "...sensitive endpoints, such as embryonic development, early survival, growth, reproduction, adult survival, and other ecologically relevant responses." This Protocol document states that an uncertainty factor of at least 10 is to be used to account for variability in species, gender, life stage, and duration of exposure. The Protocol document also recommends the use of a factor of 5.6 to extrapolate from a LOEL to a no-observable-effect-level (NOEL), if a NOEL cannot be estimated directly from dose-response data. Finally, TRGs are to be corrected for the species with the highest food consumption per body mass.

SELECTION OF TEST SPECIES

Environment Canada chose to use ducks as the test species and egg shell thinning as the toxic endpoint for assessing the reproductive effect of DDT on fish-eating avian species. Mallard and black ducks are not fish-eating. They are primarily herbivores. They are also not particularly sensitive to the reproductive effects of DDE (Peakall et al., 1973; Peakall, 1975). Eggshell thinning below the threshold for hatching failure has been shown in numerous studies not to be detrimental to avian wildlife. Environment Canada cites, but does not use, studies done with American kestrels (sparrow hawks). This hawk species is not fish-eating, but does feed on insects and small mammals. Laboratory and field studies have established a dose-response in eggshell thinning, DDE residues in eggs, and hatching failure (Porter and Wiemeyer, 1969; Wiemeyer and Porter, 1970; Peakall et al., 1973). Studies reported by Lincer (1975) contain concurrent laboratory and field studies. Residues in diet, eggs and eggshell thinning were used

to correlate the field and laboratory studies. In figure 3 below from Lincer (1975), one can see a clear dose-response between shell thickness and DDE egg residue level (dry weight basis) using the combined laboratory and field data.

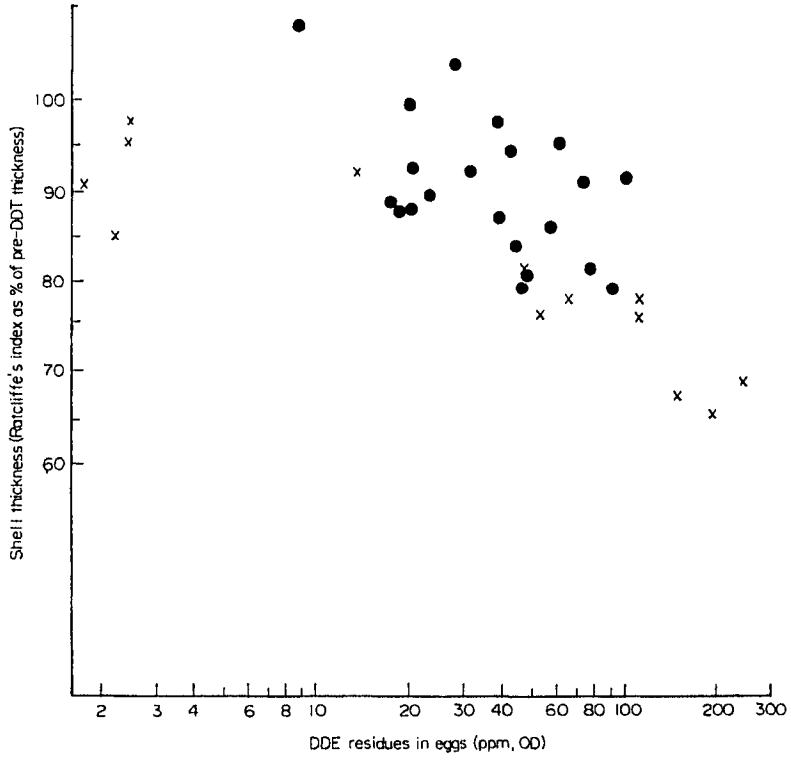

FIG. 3. Relationship between mean clutch shell-thickness and DDE residue of kestrel eggs collected in Ithaca, New York during 1970 (●) and same relationship experimentally induced with dietary DDE (×).

The same data are summarized in Appendix 16 of the Assessment document (Environment Canada, 2000) as shown below.

Appendix 16. Summary of data on the reproductive effects of orally-administered DDT and its metabolites on birds.

Species	Life Stage	Sex	Daily Dose (mg/kg BW/day)	Duration of Exposure (d)	Total Dose (mg/kg BW)	Endpoint Measured	Reference
p,p'-DDE (cont.)							
Black ducks	adult	F	0	136	0	Eggshell thickness (0.34 mm) - control	Longcore et al. 1971
Black ducks	adult	F	0.3	136	41	Eggshell thickness (0.28 mm) - S	Longcore et al. 1971
Black ducks	adult	F	0.9	136	122	Eggshell thickness (0.26 mm) - S	Longcore et al. 1971
Indian runner ducks	1 year	F	0	45	0	Eggshell index (2.2) - control	Lundholm 1984
Indian runner ducks	1 year	F	4	45	180	Eggshell index (1.6) - S	Lundholm 1984
Indian runner ducks	1 year	F	0	45	0	Calcium secretion (39.4 µg/duck) - control	Lundholm 1984
Indian runner ducks	1 year	F	4	45	180	Calcium secretion (28.0 µg/duck) - S	Lundholm 1984
American kestrels	adult	F	0	168	0	Eggshell thickness (0.171 mm) - control	Lincer 1975
American kestrels	adult	F	0.05	168	8	Eggshell thickness (0.175 mm) - NS	Lincer 1975
American kestrels	adult	F	0.5	168	84	Eggshell thickness (0.145 mm) - S	Lincer 1975
American kestrels	adult	F	1	168	168	Eggshell thickness (0.135 mm) - S	Lincer 1975
American kestrels	adult	F	1.7	168	286	Eggshell thickness (0.126 mm) - S	Lincer 1975

The 0.5 mg/kg-day level (3 ppm in the diet) produced 15 % eggshell thinning, corresponding to a level just below the threshold for hatching failure, the most sensitive toxic endpoint of chronic DDE exposure in birds. The near threshold dietary intake of 0.5 mg/kg-day in a sensitive carnivorous species is a more appropriate basis for a maximum tolerable daily intake (TDI) than the square root of the product of the shell thinning LOEL in ducks and an estimated (5.6 times less) shell thinning NOEL. The TDI should be based on 0.5 mg/kg-day and not 0.13 mg/kg-day as used by Environment Canada.

UNCERTAINTY FACTOR

Environment Canada used an uncertainty factor of 10 to account for interspecies variability. A factor of 10 from ducks to sensitive fish-eating raptors is certainly less protective than a factor of 10 from sparrowhawks to sensitive fish-eating raptors. The Lincer (1975) study evaluated the most sensitive chronic endpoint, gender and life stage in a sensitive species. For example, Newton and Bogan (1978) in their report on the DDE-eggshell thinning dose-response, stated: "The regression of shell index on log DDE content in the sparrow hawk was similar to those found by other workers for *Falco peregrinus, F. mexicanus* and *Pelecanus occidentalis.*" In Chapter 3 of this report, the dietary threshold for DDE reproductive effects in osprey was estimated to be 0.3 ppm in fish. This level would correspond to exactly one-tenth of the 0.5 mg/kg-day threshold in the sparrowhawk, which is calculated from a dietary level of 3 ppm. If one accepts the 10-fold uncertainty factor for variability in species susceptibility, the one remaining variable to consider is the rate of dietary intake.

FOOD INTAKE RATE - WILSON'S STORM PETREL

Environment Canada applied an additional uncertainty factor to the TDI to account for the species with the maximum food intake per day. They chose Wilson's storm petrel, with a food intake of 0.94 kg food/kg body weight per day. The choice of the species with the highest rate of food intake should be limited to species as sensitive or nearly as sensitive as the most sensitive species. The choice of Wilson's storm petrel is inappropriate, because petrels have not been shown to be anywhere near as sensitive as the osprey, brown pelican, peregrine falcon, or other sensitive species. In addition, Wilson's storm petrel eats fish only as a minor part of its diet. Most of the petrels diet is at lower trophic levels, explaining, at least in part, the lower sensitivity of this species to the reproductive effects of DDE.

For example, Coulter and Risebrough (1973) measured 43 ppm DDE in ashy petrel eggs that were thinned only 8-9 %. The authors concluded: "The magnitude of shell-thinning is apparently less than a critical level that would affect reproductive success." Henny et al. (1982) measured DDE residues in eggs from Leach's storm petrel collected in 1979 along the Oregon coast. DDE residue levels averaged 2.5 ppm. Eggshell thinning in Leach's storm petrel measured in eggs collected from 1946 to 1979 did not exceed 8 %. Pearce et al. (1979) reported residues of DDE in Leach's storm petrel eggs of 0.75 to 6.81 ppm. The eggs were collected in 1972 and 1976 off the east coast of Canada. The authors report measuring shell thickness, but no data were reported. The authors claim that 12 ppm DDE in eggs produces 20 % shell thinning. This conclusion was based on an extrapolation of the residue - shell thinning data. Again, no data or regression plots were reported in the article. Elliot et al. (1989) reported DDE residues in Leach's storm petrel eggs collected off the Pacific coast of Canada in 1970-1985. Residue levels ranged from 0.601 to 2.16 ppm. Residues in eggs of fork-tailed storm petrel eggs ranged from 1.68 to 2.62 ppm. The authors cite the 12 ppm DDE critical level reported by Pearce et al. (1979). Elliot et al. (1989) concluded that DDE levels were well below concentrations known to reduce reproductive rates or survival in related species elsewhere.

With critical egg residue levels for hatching failure in the range of 3-4 ppm for sensitive species, Wilson's storm petrel appears to be an inappropriate choice for a protective rate of food intake. The Protocol document lists many species that are consumers of aquatic biota (Table 1). In this list, the osprey appears to be the most sensitive species. The daily food intake rate for the osprey is listed as 0.2 kg/kg body weight-day. If one considers both the rate of food intake and reproductive effect threshold to DDE as a measure of sensitivity to DDE, the osprey appears to be the most sensitive species listed in Table 1. The peregrine falcon is not listed in Table 1. The peregrine falcon is less sensitive than the osprey when comparing egg residues of DDE, eggshell thinning, and threshold for hatching failure. However, the peregrine is at least a fraction of a trophic level higher than the osprey, because the peregrine preys, at least in part, on birds that consume aquatic biota. The comparison between the osprey and peregrines is difficult without knowing the prey of the peregrine. Coastal peregrines tend to have higher residue levels than interior peregrines, because their diet reflects bird species that feed on small fish and lower trophic level aquatic organisms.

REFERENCE CONCENTRATION

The reference concentration can be most simply calculated directly from the ppm DDE in the sparrow hawk diet. If one divides the 3 ppm dietary level, a level that produced 15 % shell thinning, by an uncertainty factor of 10, the maximum NOEL for reproduction in the most sensitive species is 0.3 ppm or 300 ppb in the diet. Assuming the osprey is the most sensitive species with a food consumption rate of 0.2 kg/kg (Table 1 in the Protocol document) and the sparrow hawk with a food consumption rate of 0.167 kg/kg (calculated from data in Appendix 16 of the Assessment document), the reference concentration in fish is 300 ppb x 0.167/0.2 = 250 ppb.

ANALYSIS

The reference concentration (which becomes the tissue reference guideline or TRG) calculated above is 18 times higher than that recommended by Environment Canada. Environment Canada's 18-fold lower TRG is due to the use of inappropriate species for establishing the TDI, shell thinning instead of hatching failure as the toxic endpoint, and an inappropriate species for estimating the maximum food intake rate.

CONCLUSIONS

- Environment Canada has developed a TRG for the protection of fish-eating birds that did not consider the best science.
- A TDI was calculated from shell thinning dose-response studies in ducks, when a combined field and laboratory study in raptors was available.
- Use of the more sensitive raptor study and a hatching failure endpoint resulted in a four-fold greater TDI.
- A relatively insensitive species, Wilson's storm petrel, was used to estimate a maximum food intake rate.
- Considering sensitivity to DDE and food intake rate, the most sensitive species in Environment Canada's list of species ingesting aquatic biota was the osprey.
- Use of the food intake rate of the osprey increased the tissue reference concentration by more than four-fold.

- Using Environment Canada's methodology, but with more appropriate species and toxic endpoint, increased the TRG in fish 18-fold. The TRG is more appropriately 250 ppb rather than 14 ppb.

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Appendix H: Dr. James L. Byard, "Scientific Commentary on the California OEHHA Sport Fish Guidance for DDT"

SCIENTIFIC COMMENTARY ON CALIFORNIA OEHHA SPORT FISH GUIDANCE FOR DDT

James L. Byard, Ph.D., D.A.B.T.

August 28, 2006

SUMMARY

The U.S. EPA and SARWQCB have misinterpreted the OEHHA fish guidance for DDTs to claim impairment of sport fishing in Newport Bay. The OEHHA guidance cautions against using the 100 ppb target as a standard. The objective of the OEHHA guidance was to achieve a potential cancer risk of less than 1/10,000 at each site. This objective is met in Newport Bay. The guidance states that the linear dose extrapolation procedure used to estimate cancer risk likely overestimates the actual risk. Studies confirm that DDTS are not genotoxic and produce cancer in rodent livers by a threshold promoting activity. This understanding was part of the original FDA action level of 5,000 ppb in commercial fish. OEHHA has recently issued new draft guidance that raises the fish fillet screening level to 560 ppb total DDT. The new guidance uses the 1/10,000 cancer risk level and considers the decay of DDTs in the environment. This new guidance is also met in Newport Bay. DDTs are not impairing sport fishing in Newport Bay.

INTRODUCTION

On June 14, 2002, the U.S. EPA, Region IX (EPA), promulgated total maximum daily loads (TMDLs) for total DDT (sum of DDT, DDD and DDE) in the San Diego Creek and Newport Bay (U.S. EPA, 2002). Staff at the SARWQCB (Rose, 2006) have concurred with U.S. EPA in the use of 100 ppb total DDT in fish fillets as a TMDL target to protect human health. The 100 ppb target was adopted from guidance issued by the Office of Environmental Health Hazard Assessment (OEHHA) of the California EPA. The guidance was developed to protect sport fishermen. The guidance is explained in a report published by OEHHA scientists in 1991 (Pollock et al., 1991). The following is a scientific commentary on the sport fish guidance developed by OEHHA.

OEHHA 1991 REPORT ON DDT IN FISH

The guidance was based on fish caught in Southern California in 1987. The focus was the high concentrations of total DDT in fish in the area of the Palos Verdes Shelf. Fish there were contaminated from DDT wastes from the Montrose Chemical Company that were released by

way of the Los Angeles County outfall. The intent was to limit the potential cancer risks of ingestion of a variety of fish species at the more highly contaminated sites.

A trigger level, set at a lifetime cancer risk of 1/100,000, was developed for each chemical based on cancer potency in rodents and assuming a linear dose-response. The following statements concerning the trigger levels were copied from the OEHHA report.

The trigger levels for total DDTs and chlordanes are based on excess cancer risks of about 1 in 100,000 (1×10^{-5}).

Recommendations are provided for species and sites which exceeded 100 ppb of either total DDTs or PCBs or 23 ppb of total chlordane.

The trigger levels were not intended to be used as standards as stated in the report as follows.

The trigger levels were developed specific to this study, therefore, and should not be used in deriving standards.

Although the trigger levels were developed for each species and chemical, the overall objective was to achieve a potential cancer risk of less than 1/10,000 as noted in the following statement from the report.

The specific recommendations for each site and species attempt to reduce exposures to levels that result in overall risks of less than 1×10^{-4} (risk for PCBs at the MDL) or lower depending on the site.

This latter objective was overlooked by both U.S. EPA and the SARWQCB in deciding to use the 100 ppb guidance as a TMDL target for total DDT. OEHHA's objective was to have the total cancer risk for a site, considering multiple species and chemicals, below a potential lifetime cancer risk of 1/10,000, not necessarily below a risk of 1/100,000. The 1/100,000 objective was an operational goal by species and chemical and was clearly not intended for adoption as a TMDL target. Considering the levels of chlordane, PCBs and total DDT in fish fillets from Newport Bay (Allen et al., 2004) recent estimates of potential cancer risks are below 1/10,000, meeting the site objective in the OEHHA guidance. In fact, OEHHA has not issued a fish consumption warning for Newport Bay.

Furthermore, OEHHA is in the process of revising the fish advisory for DDT. The draft guidance lists the screening value for total DDT at 560 ppb (Klasing and Brodberg, 2006).

Table 2. Screening Values[1] for Selected Fish Contaminants (ppb, wet weight)

Contaminant	Screening Value
Chlordane	200
DDTs	560
Dieldrin	16
Methylmercury	80
Selenium	1,940
PCBs	20
Toxaphene	220

[1] Screening values are specific guidance tissue levels used to identify situations where contaminant concentrations in fish are of potential health concern and further action (e.g., additional sampling or developing consumption advice) is recommended.

The value of 560 ppb is based on a 1/10,000 potential lifetime cancer risk. The value also incorporates a factor for the ongoing decay of DDTs (DDTs include DDT, DDE and DDD) in the environment as explained in the OEHHA draft guidance as follows.

> For carcinogenic chemicals, the exposure duration is assumed to be 30 years over a 70 year lifespan ("averaging time"). Thirty years is considered a high-end estimate of residence time for U.S. citizens (U. S. EPA, 1997; OEHHA, 2000). More importantly, levels of legacy sport fish contaminants such as PCBs, DDTs and dieldrin are declining in the environment (see for example, ATSDR, 1996; Bentzen et al., 1999; Huestis et al., 1997; Kannan et al., 1997). The average PCB half-life for Lake Ontario biota is reported to be 12 years (Bentzen et al., 1999). Even if fishers fish the same location for 70 years, their exposure to such chemicals will undoubtedly decline significantly over this period.

The risk of cancer from exposure to DDTs is inappropriately estimated by extrapolation of rodent tumor dose-response with the linearized multi-stage model. This model is intended for genotoxic carcinogens. The weight of evidence indicates that DDTs are not genotoxic. This point is made for DDE in the most widely used text in toxicology (Pitot and Dragan, 1996).

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Table 8–17
Some Nonmutagenic Chemical Carcinogens

COMPOUND	SPECIES/TARGET ORGAN	PROMOTING ACTION
p,p'-Dichlorodiphenyl-dichloroethylene	Rat/liver	+

The authors indicate that DDE is nonmutagenic (one measure of genotoxicity) and acts as a promoter. This conclusion is further explained in a recent publication from the the Pitot laboratory (Holsapple et al., 2006).

> ***Mode of action and human relevance of phenobarbital-like rodent liver carcinogens.*** Phenobarbital is the prototype of several rodent hepatocarcinogens (*e.g.*, oxazepam, DDT) that induce tumors by a non-genotoxic mechanism involving liver hyperplasia (Williams and Whysner, 1996).

The threshold for promotion is orders of magnitude higher than that for a significant carcinogenesis risk estimated by the linearized multistage model. Hence, the linear extrapolation risk numbers in the OEHHA guidance overestimate the actual cancer risk. The potential for overestimating the cancer risks is acknowledged in the OEHHA guidance.

> **V.A.5.a.(1). DDTs, Chlordane, and PCBs. The classification of DDTs, chlordane, and PCBs as potential (probable) human carcinogens is based on animal studies conducted using high doses of the chemicals. Some scientists may argue that DDTs and PCBs are not tumor initiators but rather, promoters. Resolution of this debate is beyond the scope of this report. We also recognize that the derivation of the carcinogenic potency factors (CPF or Q_1^*) are based on numerous assumptions.**

> **Overall, the assumptions used to derive the CPF are weighted such that the estimated cancer risk at a given dose is unlikely to be higher than estimated, but most likely will be lower (maybe by orders of magnitude) and perhaps may even be zero.**

These concepts were known as early as the late 1960s, explaining, in part, why the U. S. Food and Drug Administration set the action level for DDTs in commercial fish at 5,000 ppb. That action level is still in effect today as shown below.

U.S. Food & Drug Administration
Center for Food Safety & Applied Nutrition

FISH AND FISHERIES PRODUCTS HAZARDS AND CONTROLS GUIDANCE:

Third Edition June 2001

APPENDIX 5

FDA & EPA Safety Levels in Regulations and Guidance

(Return to table of contents.)

This appendix contains a listing of FDA and EPA levels relating to safety attributes of fish and fishery products published in regulations and guidance. In many cases, these levels represent the point at or above which the agency will take legal action to remove products from the market. Consequently, the levels contained in this table may not always be suitable for critical limits.

Table A-5

FDA & EPA Safety Levels in Regulations and Guidance

Product	*Level*	*Reference*
All fish	DDT, TDE and DDE - 5.0 ppm (edible portion).	Sec 575.100 Compliance Policy Guide

ANALYSIS

The OEHHA guidance dealing with the risk of human cancer from ingestion of fish fillets has been misinterpreted to claim impairment of beneficial uses of Newport Bay. However, even the 1/100,000 potential risk level is met by those ingesting sport fish from Newport Bay. As reported in the Allen et al. (2004) study, a survey among local anglers identified the most sought after species of fish. Four of the top five were analyzed for DDTs. Total DDT residues in these four species by preference rank were 69, 68, 64 and 84(68, 101) ppb. The average DDT residue in 14 species of sport fish was 79 ppb. These fish were captured in 2000 and 2001. The levels today are almost certainly lower. Considering these residue levels in sport fish fillets, even the 100 ppb target is met. There is no impairment of sport fishing in Newport Bay.

CONCLUSIONS

- U.S. EPA and the SARWQCB staff have misinterpreted the guidance for DDTs in the OEHHA base document.

- The OEHHA guidance cautions against using the 100 ppb target in fish fillets as a standard. The guidance uses the operational target of 100 ppb to achieve a risk objective of less than 1/10,000 at each site. There is no guidance issued for Newport Bay.

- The OEHHA guidance warns the reader that the linearized multi-stage extrapolation of cancer risk is conservative and may greatly overestimate actual risk. Recent publications confirm the nongenotoxic promoting action of DDTs.

- New draft OEHHA guidance considers the decay of DDTs in the environment and uses the 1/10,000 risk level. The result is new guidance of 560 ppb total DDT in fish fillets.

- The FDA action level for DDTs in commercial fish is 5,000 ppb.

- Preferred species of sport fish in Newport Bay meet all of the guidance issued by OEHHA as does the overall average residue in 14 species of sport fish. There is no impairment of sport fishing in Newport Bay.

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Pollock, Gerald A., Uhas, Iyorlumun J., Fan, Anna M., Wisniewski, Joy A. and Ingrid Witherell, A study of chemical contamination of marine fish from Southern California. II. Comprehensive study. Office of Environmental Health Hazard Assessment, Califonia Environmental Protection Agency, Sacramento, 1991.

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Appendix I: Consultant Qualifications

ERICSON JOHN LIST

Principal Consultant, Flow Science Incorporated and *Emeritus* Professor of Environmental Engineering Science, California Institute of Technology, Pasadena, California.

Years of Experience

44

Education

Ph.D. Applied Mechanics and Mathematics - California Institute of Technology, 1965
M.E. (Civil Engineering) - University of Auckland, New Zealand, 1962
B.Sc. (Mathematics) - University of Auckland, New Zealand, 1962
B.E. (First Class Honors) – University of Auckland, New Zealand, 1961

Professional Affiliations

Professional Civil Engineer in the States of California (C 36791), South Carolina (20646)
Florida (57786), North Carolina (027270), Nevada (015627), Georgia (028604)
Life Member and Fellow of American Society of Civil Engineers
Consulting Engineers and Land Surveyors of California and ACEC South Carolina
U.S. National Science Foundation Award for Special Creativity, 1982
Who's Who in America and Who's Who in Engineering

Key Qualifications

Dr. List was Professor of Environmental Engineering Science at the California Institute of Technology between 1969 and 1997. He joined the faculty at Caltech in 1969 as an Assistant Professor, after spending three years as a lecturer and senior lecturer at the University of Auckland. For the period of 1980-1985, he was Executive Officer of Environmental Engineering Science at Caltech. He also held the position of editor of the *Journal of Hydraulic Engineering*, American Society of Civil Engineers, from 1984 to 1989. Since 1997 he has been Principal Consultant at Flow Science Incorporated.

Related Experience

Professor List has consulted with more than 800 industrial organizations, consulting engineers and governmental agencies, including Southern California Edison, Chevron, IBM, Exxon, AstraZeneca, City and County of San Francisco, City of Los Angeles, City of Seattle, City of San Diego, City and County of Honolulu, Southern California Metropolitan Water District, Southern Nevada Water Authority, Los Angeles, Orange County and Sacramento Sanitation Districts. He has authored reports in the following areas of work: brine disposal, coastal ocean mixing, ICP-MS tracer analysis, power plant cooling systems, wastewater diffusers, dredge spoil disposal, river dispersion, reservoir modeling, reservoir destratification and mixing, well testing, renovation and failure analysis, pulsation control and waterhammer protection, pipeline failure, groundwater mass balance, pump wetwell design, acoustic resonance in piping systems, particle coagulation and sedimentation, fate and transport of DDT, arsenic, chromium and perchlorate.

Professor List is co-author of the texts *Mixing in Inland and Coastal Waters* (Academic Press, 1979), *Turbulent Buoyant Jets and Plumes* (Pergamon Press, 1983), and the award-winning *Handbook of Ground Water Development* (Wiley, 1990). He is the author or co-author of 40 scientific publications. Since its establishment in 1983 by Dr. List, Flow Science Incorporated has successfully completed more than 1,000 contracts.

SUSAN C. PAULSEN

Vice President and Senior Scientist, Flow Science Incorporated

Years of Experience

14

Education

Ph.D. Environmental Engineering Science, California Institute of Technology, 1997
M.S. Civil Engineering, California Institute of Technology, 1993
B.S. Civil Engineering (with honors), Stanford University, 1990

Professional Affiliations

Registered Professional Engineer in California (C66554)

Key Qualifications

Dr. Paulsen has been employed at Flow Science since 1997, where she has project responsibility for work involving environmental fate and transport. Dr. Paulsen has particular expertise in the analysis of fate, transport, and water quality in estuarine systems, including the San Francisco Bay-Delta system, where she developed a unique fingerprinting method for the analysis of mixing patterns and the sources of salinity in the Delta. At Flow Science she has been involved in projects combining hydrodynamics, aquatic chemistry, and the environmental fate of various constituents. Dr. Paulsen also oversees water quality regulatory and policy analysis for Flow Science.

Experience

Dr. Paulsen has designed and implemented field studies in reservoir, river, estuarine, and ocean environments using both dye and elemental tracers to evaluate the impact of treated wastewater, thermal, and agricultural discharges on receiving waters and drinking water intakes. Dr. Paulsen has expertise designing and managing modeling studies to evaluate transport and mixing, including the siting and design of diffusers, and she has conducted water quality analyses for storm water runoff, NPDES permitting, irrigation, and wastewater and industrial process water treatment facilities.

Dr. Paulsen has designed studies utilizing the Fischer Delta Model (FDM), three-dimensional CFD modeling, longitudinal dispersion modeling, and Monte Carlo modeling to evaluate water quality impacts and to develop NPDES permit limits for a major treated wastewater discharge to a tidally-driven river. She has designed and implemented tracer and/or modeling studies for a number of agencies including Contra Costa Water District, CALFED, DWR, Irvine Ranch Water District, and the Sacramento Regional County Sanitation District. Dr. Paulsen has also managed and designed studies to investigate the disposal of brines from salt production and reverse osmosis (RO) facilities, and she has participated in several intensive multi-disciplinary studies of the fate and transport of both organic and inorganic pollutants, including DDT, copper, and selenium, in surface and ground waters and sediments.

Dr. Paulsen has extensive expertise with water quality regulation in California and served as primary author for a comprehensive review of the administrative record of the Los Angeles Basin Plan. She has worked on temperature compliance models, NPDES permitting, permit compliance, master planning and EIR/EIS processes, and TMDL development. She has expertise regarding the importance of atmospheric deposition, soil erosion, and wildfires on storm water quality, the development of numeric limits for storm flows, and the use of indicator bacteria as a measure of water quality. Dr. Paulsen has also provided testimony to the California State Water Resources Control Board and Regional Boards in water rights and permitting issues, has spoken extensively on regulatory issues, and currently serves on the State Board's Sediment Quality Objective Advisory Committee.

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EDUCATION

B.S., Biochemistry, Cornell University, 1960-1964
Ph.D., Biochemistry, University of Wisconsin, 1964-1968
Postdoctorate, Biological Chemistry, Harvard Medical School, 1968-1970

HONORS

Babcock Fellow, University of Wisconsin, 1967-1968
Arthritis Fellow, Harvard Medical School, 1968-1970

CERTIFICATIONS

Diplomate of the American Board of Toxicology, 1980-present

PROFESSIONAL SOCIETIES

Society of Environmental Toxicology and Chemistry
Society of Toxicology
Society for Risk Analysis

EMPLOYMENT

Sole Proprietor of James L. Byard, Toxicology Consultant, 1984 - present. Consulting in basic and applied research in toxicology, risk assessment, auditing toxicity studies, environmental fate of chemicals, and testimony as an expert witness.

Adjunct Associate Professor, Distinguished Visiting Scholar, and Lecturer, Department of Environmental Toxicology, Univeristy of California, Davis, California 95616 (1984-1995). Teaching University courses in toxicology.

Assistant and Associate Professor of Environmental Toxicology, Department of Environmental Toxicology, University of California, Davis (1974-1984). Teaching, research, and public service in toxicology. Research in chemical carcinogenesis, metabolism, mechanism-of-action, and primary liver cell cultures.

Research Assistant Professor of Toxicology, Center of Experimental Pathology and Toxicology, Albany Medical College of Union University, Albany, New York 12208 (1970-1974). Teaching in toxicology and biochemistry. Research in metabolism and mechanism-of-action of saccharin, carrageenan, dieldrin, mirex, PCBs, hexachlorobenzene, methyl mercury, and freons.

CONSULTING EXPERIENCE

Reviewed NIOSH criteria document for benzylchloride.

Reviewed EPA drinking water criteria document for dibromochloropropane.

Participated in the laetrile hearings in the California Governor's Office.

Gave written and oral testimony to Proposition 65 Scientific Advisory Panels, State and Regional Water Boards, and District Air Pollution Boards.

Consulted with the California Department of Pesticide Regulation, Office of Environmental Health Hazard Assessment, U. S. Environmental Protection Agency, and the U. S. Food and Drug Administration

Toxicology consultant to the Health Effects Study of the Replenishment of Ground water with Treated Waste Water, County Sanitation Districts of Los Angeles County.

Member of the California Department of Health Service's Water Reuse Health Effects Panel.

Developed a surface and ground water monitoring program for Alpine County, California.

Chaired a two-day conference on chemical carcinogenesis and teratology for the California Air Resources Board.

Toxicology consultant to several engineering firms dealing with cleanup of hazardous wastes (e.g., Rocky Mountain Arsenal, Brio Refining, THAN- Fresno, BKK Landfill, Concord Naval Weapons Station; Operating Industries Landfill, Kopper's Oroville site, Silicon Valley groundwater contamination, Lincoln Village, etc.).

Consultant to several chemical companies (e. g., Monsanto, Syntex, IBM, U.

S. Borax, Du Pont, TH Agriculture and Nutrition, etc.). Assignments include risk assessment, audits of toxicology studies, human exposure studies, and genetic toxicology studies.

Consultant to the California Rice Industry Association (risk assessment of rice pesticides and rice smoke).

Consultant to The Irvine Company (predevelopment hazard assessments, Proposition 65 compliance, pesticides and metals in aquatic environments).

Evaluation of the hazards of consumer products to meet regulations of the Consumer Product Safety Commission.

Consultant/expert witness for numerous legal cases involving human exposure to aldrin, ammonia, asbestos, benzene, brodifacoum, cadmium, carbon monoxide, chlordane, chlorine, chloroform, chlorpyrifos, chromium, creosote, 2,4-D, DBCP, DDT, diazinon, dieldrin, diesel fuel, dioxin, endrin, ethyl ether, formaldehyde, freon 113, gasoline, heptachlor, hexane. isopropyl alcohol, lead, marijuana, mercury, methyl bromide, methylene chloride, methyl ethyl ketone, methyl isobutyl ketone, mixed hydrocarbon solvents, paraquat, parathion, PAHs, PCBs, pentachlorophenol, perchlorate, perchloroethylene, phosdrin, selenium, silica, silvex, sulfur oxides, 2,4,5-T, toluene, trichloroethane, trichloroethylene, vinyl chloride, vinylidene chloride, xylene, etc.

EXAMPLES OF TECHNICAL REPORTS

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2. **Risk assessment of the Denver Rail Yard, site of the Coors Baseball Field.**
3. **Risk assessment of vehicle emissions contaminating the Sweetwater Reservoir.**
4. **Comparison of hazardous materials in household wastes and industrial liquid wastes.**
5. **Annotated bibliography of industrial vitiligo.**
6. **Annotated bibliography of carbon monoxide poisoning.**
7. **Report on monitoring of rice pesticide residues in the United States and Japan.**
8. **Hazard assessment of amorphous silica in rice straw smoke.**
9. **Annotated bibliography of 2,4-D, 2,4,5-T, and 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin.**

10. **Annotated bibliography of the acute dose-response of ammonia in humans.**

11. **Annotated bibliography of the acute dose-response of sulfur dioxide in humans.**

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3. **Byard, J. L., The Toxicological Significance of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Related Compounds in Human Adipose Tissue. Journal of Toxicology and Environmental Health 22: 381-403, 1987.**

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8. **Knadle, S. A., The Kinetics of Benzene Metabolism in Rhesus Monkey Hepatocytes Cultured in Glass T-flasks, Ph.D. Thesis, University of California, Davis, 1982 (Chairperson of thesis committee).**

9. **Salocks, C. B., Hsieh, D. P. H. and Byard, J. L., Effects of Butylated Hydroxy-toluene Pretreatment on the Metabolism and Genotoxicity of Aflatoxin B_1 in Primary Cultures of Adult Rat Hepatocytes: Selective Reduction of Nucleic Acid Binding. Toxicol. Appl. Pharmacol. 76: 498-509, 1984.**

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24. Dougherty, K. K., Spilman, S. D., Green, C. E., Steward, A. R. and Byard, J. L., Primary Cultures of Adult Mouse and Rat Hepatocytes for Studying the Metabolism of Foreign Chemicals. Biochemical Pharmacology 29: 2117- 2124, 1980.

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27. Wong, Z. A., Decad, G. M., Byard, J. L. and Hsieh, D. P. H., Conversion of Aflatoxicol to Aflatoxin B_1 in Rats in vivoand in Primary Hepatocyte Culture. Food Cosmetics Toxicology 17: 481-486, 1979.

28. Decad, G. M., Dougherty, K. K., Hsieh, D. P. H. and Byard, J. L., Metabolism of Aflatoxin B_1 in Cultured Mouse Hepatocytes: Comparison with Rat and Effects of Cyclohexene Oxide and Diethyl Maleate. Toxicol. Appl. Pharmacol. 50: 429-436, 1979.

29. Decad, G. M., Hsieh, D. P. H. and Byard, J. L., Maintenance of Cytochrome P-450 and Metabolism of Aflatoxin B_1 in Primary Hepatocyte Cultures. Biochem. Biophys. Res. Comm. 78: 279-287, 1977.

30. Byard, J. L., Koepke, U. Ch., Abraham, R., Golberg, L. and Coulston, F., Biochemical Changes in the Liver of Mice Fed Mirex. Toxicol. Appl. Pharmacol. 33: 70-77, 1975.

31. Byard, J. L., McChesney, E. W., Golberg, L. and Coulston, F., Excretion and Metabolism of Saccharin in Man. II. Studies With ^{14}C-Labelled and Unlabelled Saccharin. Food Cosmetics Toxicology 12: 175-184, 1974.

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33. Griffin, T., Byard, J. L. and Coulston, F., Toxicological Responses to

Halogenated Hydrocarbons, In An Appraisal of Halogenated Fire Extinguishing Agents. Christian, W. J. and Wands, R. C., eds., pp. 136-145, National Academy of Sciences, Washington, 1972.

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PUBLISHED ABSTRACTS OF PRESENTATIONS GIVEN AT NATIONAL MEETINGS

1. Loury, D. J. and Byard, J. L., Subchronic Ethanol Administration Enhances the Hepatocellular Genotoxicity of Several Pyrolysate Mutagens. Toxicologist 4: 33, 1984.

2. DiRenzo, A.B., Gandolfi, A. J., Brendel, K., Sipes, I. G. and Byard, J. L., Effect of Hypoxia on the Bioactivation and Toxicity of CCl_4 and Halothane in Primary Rat Hepatocyte Cultures. Pharmacologist 25: 170, 1983.

3. Salocks, C. B., Hsieh, D. P. H. and Byard, J. L., Butylated Hydroxytoluene Pretreatment Selectively Reduces Covalent Binding of Aflatoxin B_1 to DNA and RNA in Primary Cultures of Rat Hepatocytes. Proceedings of the American Association for Cancer Research 24: 87, 1983.

4. Loury, D. J. and Byard, J. L., A Rapid and Sensitive Technique for Measuring Unscheduled DNA Synthesis in Primary Hepatocyte Cultures. Toxicologist 3: 38, 1983.

5. Knadle, S. A. and Byard, J. L., The Kinetics of Benzene Metabolism in Primary Hepatocyte Cultures of Guinea Pig and Rat Compared to Inhalation Uptake Kinetics. Toxicologist 3: 87, 1983.

6. Knadle, S. A., Salocks, C. B., Nakashima, J. and Byard, J. L., Comparative Rates of Benzene Metabolism in Primary Hepatocyte Cultures. Toxicologist 2: 22-23, 1982.

7. Steward, A. R. and Byard, J. L., Effect of Vitamin A on the Induction of Benzopyrene Metabolism by 2,3,7,8-Tetra- chlorodibenzodioxin in Primary Hepatocyte Cultures. Pharmacologist 23: 179, 1981.

8. Gill, S. S., Hammock, B. D. and Byard, J. L., Comparative Metabolism of Stilbene Oxides by Primary Hepatocyte Cultures. Toxicologist 1: 141-142, 1981.

9. Spilman, S. D. and Byard, J. L., Sulfate-Dependent Metabolic Activation of 2-Acetylaminofluorene by Primary Cultures of Adult Rat Hepatocytes. Toxicologist 1: 35, 1981.

10. Green, C. E., Rice, D. W., Hsieh, D. P. H. and Byard, J. L., Potency of Aflatoxin B_1 and Aflatoxin M_1 in Cytotoxicity and DNA Repair Assays. Toxicologist 1: 42, 1981.

11. Salocks, C. B., Hsieh, D. P. H. and Byard, J. L., Butylated Hydroxytoluene Pretreatment Reduces Cytotoxicity and Covalent Binding of Aflatoxin B_1 in Primary Hepatocyte Cultures. Toxicologist 1: 108-109, 1981.

12. Green, C. E., Segall, H. J. and Byard, J. L., Metabolic Fate and Toxicity of Senecionine in Primary Hepatocyte Cultures. Abstracts of the Nineteenth Annual Meeting of the Society of Toxicology, A44, 1980.

13. Steward, A. R. and Byard, J. L., Induction of Benzopyrene Metabolism by 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Primary Cultures of Adult Rat Hepatocytes. Abstracts of the Nineteenth Annual Meeting of the Society of Toxicology, A85, 1980.

14. Dougherty, K. K. and Byard, J. L., Induction of Mixed Function Oxidase by Phenobarbital, Hormones and Serum in Primary Cultures of Mouse Hepatocytes. Fed. Proc. 38: 846, 1979.

15. Spilman, S. D. and Byard, J. L., Metabolism of 2-Acetylaminofluorene in Primary Cultures of Rat Hepatocytes. Pharmacologist 20: 175, 1978.

16. Decad, G. M., Dougherty, K. K., Hsieh, D. P. H. and Byard, J. L., Comparative Metabolism of Aflatoxin B_1 in Mouse and Rat Primary Hepatocyte Cultures. Toxicol. Appl. Pharmacol. 45: 274, 1978.

17. Wei, C. I., Decad, G. M., Wong, Z. A., Byard, J. L. and Hsieh, D. P. H., Characterization and Mutagenicity of Water-Soluble Conjugates of Aflatoxin B_1. Toxicol. Appl. Pharmacol. 45: 274, 1978.

18. Dougherty, K. K. and Byard, J. L., Induction of Mixed- Function Oxidase in Primary Cultures of Mouse Hepatocytes. Toxicol. Appl. Pharmacol. 45: 261, 1978.

19. Dougherty, K. K., Spilman, S. D., Green, C. E., Steward, A. R. and Byard, J. L., Primary Hepatocyte Cultures for the Investigation of the Fate and Mechanism of Action of Environmental Chemicals. Toxicol. Appl. Pharmacol. 41: 190, 1977.

20. Byard, J. L. and Pittman, K. A., Early Liver Changes Produced by Mirex and Their Reversibility. Toxicol. Appl. Pharmacol. 33: 130, 1975.

21. Griffin, T.B., Byard, J. L. and Coulston, F., Golberg, L. and Harris, E.S. Continuous Exposure of Rats to Hexafluoroethane. Toxicol. Appl. Pharmacol. 29: 82, 1974.

22. Byard, J. L., Koepke, U. Ch., Abraham, R., Golberg, L. and Coulston, F., Biochemical Changes Produced in the Liver by Mirex. Toxicol. Appl. Pharmacol. 29: 126-127, 1974.

23. Byard, J. L., McChesney, E., Golberg, L. and Coulston, F., Further Observations on the Metabolism of Saccharin in Man. Toxicol. Appl. Pharmacol. 29: 154-155, 1974.

24. Byard, J. L., Observations on the Metabolism of Saccharin. Toxicol. Appl. Pharmacol. 22: 291-292, 1972.

25. Byard, J. L. and Bauman, C. A., Protein-Bound Selenium in Rats Given Sodium Selenite. Fed. Proc. 27: 417, 1968.

26. Byard, J. L. and Bauman, C. A., Selenium Metabolites in the Urine of Rats Given a Subacute Dose of Selenite. Fed. Proc. 26: 476, 1967.

September, 2006

RON TJEERDEMA

POSITION TITLE

Professor/Chair of Environmental Toxicology, University of California, Davis

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	YEAR	FIELD OF STUDY
Humboldt State University, Arcata, CA	BS	1980	Wildlife Mgmt.
Humboldt State University, Arcata, CA	BS	1980	Natural Resources Mgmt.
University of California, Santa Barbara	MA	1983	Pharmacology/Toxicology
University of California, Davis	PhD	1987	Pharmacology/Toxicology

A. Positions and Honors.

Professional Experience

2003–present Chair, Department of Environmental Toxicology, UC Davis
1999–present Professor, Department of Environmental Toxicology, UC Davis
1998–99 Professor, Department of Chemistry & Biochemistry, UC Santa Cruz
1994–98 Associate Professor, Department of Chemistry & Biochemistry, UC Santa Cruz
1992–94 Assistant Professor, Department of Chemistry & Biochemistry, UC Santa Cruz
1987–92 Assistant Research Toxicologist (research faculty), Institute of Marine Sciences, UC Santa Cruz

Professional Certification

1994–present Diplomate in General Toxicology, American Board of Toxicology (DABT)

Honors

1997 Distinguished Alumnus Award, Department of Environmental Toxicology, UC Davis
1983–87 NIEHS Predoctoral Fellowship in Toxicology, UC Davis

B. Selected peer-reviewed publications (in chronological order

1. Martello, L. B. and R. S. Tjeerdema, 2001. Combined effects of pentachlorophenol and salinity stress on chemiluminescence activity in two species of abalone. *Aquat. Toxicol.* 51, 351–362.
2. Wolfe, M. F., G. J. B. Schwartz, S. Singaram, E. E. Mielbrecht, R. S. Tjeerdema and M. L. Sowby, 2001. Influence of dispersants on the bioavailability and trophic transfer of petroleum hydrocarbons to larval topsmelt (*Atherinops affinis*). *Aquat. Toxicol.* 52, 49–60.
3. Viant, M. R., J. H. Walton, and R. S. Tjeerdema, 2001. Comparative toxic actions of 3-trifluoro-4-nitrophenol (TFM) in marine mollsucs as characterized by in vivo ^{31}P-NMR. *Pestic. Biochem. Physiol.* 71, 40–47.
4. Viant, M. R., J. H. Walton, P. L. TenBrook and R. S. Tjeerdema, 2002. Sublethal actions of copper in abalone (*Haliotis rufescens*) as characterized by in vivo ^{31}P-NMR. *Aquat. Toxicol.* 57, 139–151.
5. Viant, M. R., C. A. Pincetich, J. H. Walton, R. S. Tjeerdema and D. E. Hinton, 2002. Utilizing in vivo NMR to study sublethal stress in aquatic organisms. *Mar. Environ. Res.* 54, 553–557.
6. Shofer, S. L. and R. S. Tjeerdema, 2002. Sublethal actions of pentachlorophenol in abalone (*Haliotis rufescens*) veliger larvae as measured by ^{31}P NMR. *Ecotoxicol. Environ. Saf.* 51, 155–160.

7. TenBrook, P. L., S. M. Kendall and R. S. Tjeerdema, 2003. Toxicokinetics and biotransformation of *p*-nitrophenol in the red abalone (*Haliotis rufescens*). *Aquat. Toxicol.* 62, 329–336.
8. Neale, J. C. C., J. A. Van de Water, J. T. Harvey, R. S. Tjeerdema and M. E. Gershwin, 2002. Proliferative responses of harbor seal (*Phoca vitulina*) T lymphocytes to model marine pollutants. *Develop. Immunol.* 9, 215–221.
9. Viant, M. R., E. R. Rosenblum and R. S. Tjeerdema, 2003. NMR-based metabolomics: A powerful tool for characterizing the effects of environmental stressors on organism health. *Environ. Sci. Technol.* 37, 4982–4989.
10. Viant, M. R., I. Werner, E. R. Rosenblum, A. S. Gantner, R. S. Tjeerdema and M. L. Johnson, 2004. Correlation between heat-shock protein induction and reduced metabolic condition in juvenile steelhead trout (*Oncorhynchus mykiss*) chronically exposed to elevated temperature. *Fish Physiol. Biochem.* 29, 159–171.
11. Neale, J. C., F. M. D. Gulland, K. R. Schmelzer, J. T. Harvey, E. A. Berg, S. G. Allen, D. J. Greig, E. K. Grigg and R. S. Tjeerdema, 2005. Contaminant loads and hematological correlates in the harbor seal (*Phoca vitulina*) of San Francisco Bay, California. *J. Toxicol. Environ. Health.* 68: 617–633.
12. Mielbrecht, E. E., M. F. Wolfe, R. S. Tjeerdema and M. L. Sowby, 2005. Influence of a dispersant on the bioaccumulation of phenanthrene by topsmelt (*Atherinops affinis*). *Ecotoxicol. Environ. Saf.* 61, 44–52.
13. Donham, R. T., D. Morin, W. T. Jewell, M. W. Lame, H. J. Segall and R. S. Tjeerdema, 2005. Characterization of glutathione *S*-transferases in juvenile white sturgeon (*Acipenser transmontanus*). *Aquat. Toxicol.* 71, 203–214.
14. Braid, B. A., J. D. Moore, T. T. Robbins, R. P. Hedrick, R. S. Tjeerdema, and C. S. Friedman, 2005. Health and survival of red abalone, *Haliotis rufescens,* under varying temperature, food supply, and exposure to the agent of withering syndrome. *J. Invert. Pathol.* 89, 219–231.
15. Donham, R. T., D. Morin, W. T. Jewell, M. W. Lame, H. J. Segall and R. S. Tjeerdema, 2005. Characterization of cytosolic glutathione *S*-transferases in juvenile Chinook salmon (*Oncorhynchus tshawytscha*). *Aquat. Toxicol.* 73, 221–229.
16. Johnson, C. S., S. E. Schwarzbach, J. D. Henderson, B. W. Wilson and R. S. Tjeerdema, 2005. Effects of temperature on cholinesterase activity in frogs. *Environ. Toxicol. Chem.* 24, 2074–2077.
17. Neale, J. C. C., T. P. Kenny, R. S. Tjeerdema and M. E. Gershwin, 2005. PAH- and PCB-induced alterations of protein tyrosine kinase and cytokine gene transcription in harbor seal (*Phoca vitulina*) peripheral blood mononuclear cells. *Clin. Develop. Immunol.* 12: 91–97.
18. Neale, J. C., K. R. Schmelzer, F. M. D. Gulland, E. A. Berg and R. S. Tjeerdema, 2005. Organohalogen levels in harbor seal (*Phoca vitulina*) pups increase with duration of nursing. *J. Toxicol. Environ. Health.* 68, 687–691.
19. Pincetich, C. A., M. R. Viant, D. E. Hinton and R. S. Tjeerdema, 2005. Metabolic changes in Japanese medaka (*Oryzias latipes*) during embryogenesis and hypoxia determined by in vivo ^{31}P NMR. *Comp. Biochem. Physiol.* 140, 103–113.
20. Rosenblum, E. S., M. R. Viant, B. M. Braid, J. D. Moore, C. S. Friedman and R. S. Tjeerdema, 2005. Investigating the effects of pathogen, elevated temperature and starvation on the metabolic profiles of California red abalone, *Haliotis rufescens. Metabolomics* 1, 199–209.
21. Viant, M. R., J. G. Bundy, C. A. Pincetich, J. de Ropp and R. S. Tjeerdema, 2005. NMR-derived developmental metabolic trajectories: An approach for visualizing the toxic actions of trichloroethylene during embryogenesis. *Metabolomics* 1, 149–158.
22. Donham, R. T., D. Morin and R. S. Tjeerdema, 2006. Influence of salinity on activity and expression of glutathione *S*-transferases in juvenile sturgeon (*Acipenser transmontanus*) and salmon (*Oncorhynchus tshawytscha*). *Ecotoxicol. Environ. Saf.* 63, 293–298.
23. Viant, M. R., C. A. Pincetich, D. E. Hinton and R. S. Tjeerdema, 2006. Toxic effects of dinoseb in medaka (*Oryzias latipes*) embryos as determined by *in vivo* ^{31}P NMR, HPLC, and ^{1}H NMR metabolomics. *Aquat. Toxicol. Aquat. Toxicol.* 76, 329–342.

24. Viant, M. R., C. A. Pincetich and R. S. Tjeerdema, 2006. Metabolic effects of dinoseb, diazinon, and esfenvalerate in eyed eggs and alevins of Chinook salmon (*Oncorhynchus tshawytscha*) as determined by ^{1}H NMR metabolomics. *Aquat. Toxicol.* 77, 359–371.
25. Wheelock, C. E., J. L. Miller, M. J. Miller, B. M. Phillips, S. A. Huntley, S. J. Gee, R. S. Tjeerdema and B. D. Hammock, 2006. Use of carboxylesterase activity to remove pyrethroid-associated toxicity to *Ceriodaphnia dubia* and *Hyalella azteca* in toxicity identification evaluations. *Environ. Toxicol. Chem.* 25, 973–984.
26. Dixon, R. A., D. R. Gang, A. J. Charlton, O. Fiehn, H. A. Kuiper, T. L. Reynolds, R. S. Tjeerdema, E. H. Jeffery, J. B. German, W. P. Ridley and J. N. Seiber. Applications of metabolomics in agriculture. *J. Agric. Food Sci.* (invited; in press)
27. Donham, R. T., S. Chang, A. D. Luna, D. Morin and R. S. Tjeerdema. Characterization of cytosolic glutathione *S*-transferases in juvenile California halibut (*Paralichthys californicus*). *Ecotoxicol. Environ. Saf.* (in press)
28. Palumbo, A. J., J. Linares-Casenave, W. Jewell, S. I. Doroshov and R. S. Tjeerdema. Induction, purification, and partial characterization of California halibut (*Paralichthys californicus*) vitellogenin. *Comp. Biochem. Physiol.* (in press)
29. Rosenblum, E. S., M. R. Viant and R. S. Tjeerdema. Effects of the local environment on host-pathogen-drug interactions in red abalone determined by ^{1}H NMR metabolomics. *Environ. Sci. Technol.* (in press)
30. Werner, I., M. R. Viant, E. S. Rosenblum, A. S. Gantner, R. S. Tjeerdema and M. L. Johnson. Cellular responses to temperature stress in steelhead trout (*Onchorynchus mykiss*) parr with different rearing histories. *Fish Physiol. Biochem.* (in press)

C. Current Research Support

Acute and Chronic Effects of Crude Oil and Dispersed Oil on Chinook Salmon Smolts
NOAA – University of New Hampshire Cooperative Institute for Coastal and Estuarine Environmental Technology
PI – Tjeerdema
2004–06
$150,000

Influence of Temperature on the Pharmacokinetics and Efficacy of Oxytetracycline in RLP-Infected Abalone
NOAA, U.S. Department of Commerce, National and California Sea Grant College Programs
PI – Tjeerdema
2004–06
$102,282

Surface Water Ambient Monitoring Program (SWAMP)
California Department of Fish and Game
PI – Tjeerdema
2004–07
$241,610

Acute and Chronic Effects of Crude Oil and Dispersed Oil on Chinook Salmon Smolts
California Department of Fish and Game, Office of Spill Prevention and Response
PI – Tjeerdema
2004–07
$194,999

Toxic Pesticides, Location of Sources, and Evaluation of Mitigations Effectiveness in the Gabilan Watershed, Resource Conservation District of Monterey Bay
PI – Tjeerdema
2005–07
$174,790

Eradication of Exotic Fishes from Lake Davis, CA
California Department of Fish and Game
PI – Tjeerdema
2006–07
$26,500

Acute and Chronic Effects of Crude Versus Dispersed Oil on Pre-Smolt Chinook Salmon
California Department of Fish and Game, Office of Spill Prevention and Response
PI – Tjeerdema
2006–08
$84,573

Acute and Chronic Effects of Crude Oil and Dispersed Oil on Chinook Salmon Smolts
UC Wildlife Health Center, Oiled Wildlife Care Network
PI – Tjeerdema
2003–06
$106,111.

Acute and Chronic Effects of Crude Versus Dispersed Oil on Pre-Smolt Stage Chinook Salmon, UC Wildlife Health Center, Oiled Wildlife Care Network
PI – Tjeerdema
2005–07
$78,425

Sediment Toxicity Identification to Support the TMDL Process
Water Environment Research Foundation (WERF)
PI – Tjeerdema
2003–06
$497,425

Sources and Effects of Pyrethroid Pesticides in Watersheds of the San Francisco Estuary
San Francisco Estuary Institute
PI – Tjeerdema
2004–06
$111,990

Sediment Toxicity Testing: Dose-Response Sensitivity Evaluations
San Francisco Estuary Institute
PI – Tjeerdema
2004–06
$80,867

Analysis of Environmental Samples for Toxicity in the Bay Area
San Francisco Estuary Institute
PI – Tjeerdema
2006–07
$37,800

The Environmental Fate of Pesticides Important to Rice Culture
California Rice Research Board
PI – Tjeerdema
2006–07
$49,712

Pending Research Support

Watershed-Scale Effectiveness Evaluation for Pesticide Loadings to Critical Coastal Habitats
Consolidated Grants Program, California State Water Resources Control Board
PI – Tjeerdema
2007–10
$885,000

Pesticide Water Quality Criteria Development
California Regional Water Quality Control Board (Central Valley Region)
PI – Tjeerdema
2005–07
$156,000

Binary Mixtures of Endocrine Disrupting Contaminants in White Sturgeon (A*cipenser transmontanus*), UC Center for Water Resources
PI – Tjeerdema
2006–08
$52,174

Deborah A. Chiavelli, Ph.D.

CONTACT INFORMATION

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PROFESSIONAL HISTORY

Quantitative Environmental Analysis, LLC, Senior Project Scientist, January 2006 to present.
Dartmouth Medical School, Co-Principal Investigator; Post-Doctoral Research Associate, 2002 to 2005.
Dartmouth College, Grant Proposal Developer and Writer, 2000 to 2001.
Dartmouth College, Teaching Assistant and Guest Lecturer, 1996 to 2001.
University of Mississippi, Teaching Assistant, 1992 to 1995.
University of Mississippi, Research Assistant, 1992 to 1993.
Cornell University, Research Assistant, 1990 to 1992.

EDUCATION

Dartmouth College, Ph.D., Biology, 2003
University of Mississippi, M.S., Biology, 1995
Cornell University, B.S., Natural Resources, 1990

EXPERIENCE SUMMARY

Dr. Chiavelli is an aquatic ecologist with broad expertise in ecology of human pathogens in aquatic environments, host-parasite population dynamics, aquatic food web and nutrient dynamics and plankton biology. She has experience in designing and supervising logistically complex sampling programs, in developing, managing and analyzing large environmental data sets, in advanced statistical analysis and experimental design, and in modeling species interactions in aquatic communities.

Dr. Chiavelli's work as a co-Principal Investigator at Dartmouth Medical School is representative of her interdisciplinary approach to research. The project combined genomic, genetic, microbiological and ecological approaches to study the biodynamics of *Vibrio cholerae*, the causative agent of cholera, in response to changing aquatic conditions. She is familiar with current ecological and public heath issues associated with aquatic pathogens and with the latest molecular genetic and genomic techniques for monitoring aquatic pathogens as well as the more traditional microbiological monitoring methods.

Dr. Chiavelli has been a manuscript reviewer for Limnology and Oceanography, Ecology, Oecologia, Archiv fur Hydrobiologie, and Estuaries.

PROFESSIONAL ACTIVITIES

Affiliations

American Society of Limnology and Oceanography
Ecological Society of America
Sigma Xi

PRESENTATIONS

Links between the ecology, epidemiology and pathogenicity of *Vibrio cholerae*: a molecular genetic approach. Chiavelli, D.A., K.L. Cottingham and R.K. Taylor. CIESM workshop for Novel Contaminants and Pathogens in Coastal Waters, Neuchatel, Switzerland. May 12-15, 2004. Also presented at EAWAG, Department of Limnology, Dubendorf, Switzerland, and May 2004.

Host effects on transmission and growth of epibionts of *Daphnia*. Chiavelli, D.A. EAWAG (Swiss Federal Institute for Environmental Science and Technology), Department of Limnology, Dubendorf, Switzerland, May 2004.

Genomic response of *Vibrio cholerae* to changes in the aquatic environment. Chiavelli, D.A. International Centre for Diarrhoeal Disease Research, Dhaka, Bangladesh, November 2003.

Linking the ecology, epidemiology and pathogenicity of *Vibrio cholerae*: a genomic approach. Chiavelli, D.A., K.L. Cottingham and R.K. Taylor. Marine Sciences Research Center, Stony Brook, University, Stony Brook, NY, May 2003.

A surface pilus promotes adherence to zooplankton by *Vibrio cholerae*, providing a potential contribution to environmental persistence. Chiavelli, D.A, and R. K. Taylor. Ecological Society of America Annual Meeting, Madison, Wisconsin, U S. A. 2001.

Blurring the line between mutualism and parasitism: costs and benefits of algal epibiosis for *Daphnia*. Chiavelli, D. A. Fifth International Symposium on Cladocera, Plon, Germany. 1999.

Predation rates of zooplankton populations: effects of predator and prey aggregation and predator-predator interactions. Chiavelli, D.A. and C. Folt. Societas Internationalis Limnologiae (SIL) Congress, Dublin, Ireland. 1998.

Interaction between predator and prey aggregation patterns and consumption rates of predator populations. Chiavelli, D.A. and C. Folt. Ecological Society of America Annual Meeting, Albuquerque, New Mexico, U. S. A. 1997.

Aggregated distributions of *Daphnia* influence the colonization dynamics of their epibionts. Chiavelli, D.A. and S.T. Threlkeld. Paper American Society of Limnology and Oceanography Conference, Reno, Nevada, U. S. A. 1995.

Effects of *Daphnia* body size and time elapsed since molting on epibiont burden. Chiavelli, D.A. and S.T. Threlkeld. Third International Symposium on Cladocera, Bergen, Norway. 1993.

***Colacium* epibiosis on zooplankton in Oneida Lake, New York.** Chiavelli, D.A. and E.L. Mills. American Society of Limnology and Oceanography Conference, Halifax, Nova Scotia, Canada. 1991.

PUBLICATIONS

Linking the ecology, epidemiology and pathogenicity of *Vibrio cholerae*: a molecular genetic approach. Chiavelli, D.A., K.L. Cottingham and R.K. Taylor. In Novel Chemical Contaminants and Pathogens in Coastal Waters. N.S. Fisher, Ed., CIESM workshop monograph no. 26, 2004.

Executive Summary. *In* Novel Chemical Contaminants and Pathogens in Coastal Waters. One of 13 authors. N.S. Fisher, Ed. CIESM workshop monograph no. 26, 2004.

Environmental microbe and human pathogen: The ecology and microbiology of *Vibrio cholerae*. Cottingham, K.L., D.A. Chiavelli and R.K. Taylor. *Frontiers in Ecology and the Environment* 1: 80-86. 2003.

The mannose-sensitive hemagglutinin of *Vibrio cholerae* promotes adherence to zooplankton. Chiavelli, D.A., J.W. Marsh and R.K. Taylor. *Applied and Environmental Microbiology,* 67: 3220-3225, 2001.

Host preference, seasonality, and community interactions of zooplankton epibionts. Chiavelli, D.A., E.L. Mills and S.T. Threlkeld. *Limnology and Oceanography,* 38: 574-583, 1993.

The organization of zooplankton epibiont communities. Threlkeld, S.T, D.A. Chiavelli and R.L. Willey. *Trends in Ecology and Evolution,* 8: 317-321, 1993.

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PROFESSIONAL HISTORY

Quantitative Environmental Analysis, LLC, President and Senior Managing Engineer, February 1998 to present
USEPA Science Advisory Board, 2005 to present
HydroQual, Inc., Principal Engineer, 1993 to January 1998
HydroQual, Inc., Consultant, 1980 to 1993
Manhattan College, Professor, 1992 to 1994
Manhattan College, Associate Professor, 1986 to 1992
Manhattan College, Assistant Professor, 1980 to 1986
U.S. Environmental Protection Agency, Environmental Scientist, 1978 to 1980
Manhattan College, Research Engineer, 1975 to 1977

EDUCATION

The University of Texas at Austin, Ph.D., 1980
Manhattan College, M.E., Environmental Engineering, 1975
Manhattan College, B.E., Civil Engineering, 1973

REGISTRATION

Professional Engineer in the States of New York and Texas

EXPERIENCE SUMMARY

Dr. Connolly has worked on more than 35 projects in the areas of contaminant transport and bioaccumulation. These studies have involved field sampling, fine-grained sediment transport analysis, chemical fate modeling and food web bioaccumulation modeling. They have generally been directed to exposure assessment and risk assessment problems related to surface water and groundwater contamination problems for the purposes of evaluation of remedial options or wasteload allocation.

Dr. Connolly also has considerable experience in the areas of ecosystem processes and ecotoxicology. His work in these areas has focused on modeling of population dynamics, the cycling of carbon and nutrients and the relationship between contaminant exposure and toxic effects. The focus of much of this work has been on the development and application of models to evaluate pollutant loadings and the effectiveness of various pollution control strategies.

Dr. Connolly is frequently invited to participate in government and industry sponsored workshops. He is a member of the USEPA Science Advisory Board. He has worked throughout the U.S., in Latin America, and in Europe. He has served as an expert witness for industry and government agencies and has provided testimony before the U.S. Congress and the New York State Assembly.

REPRESENTATIVE PROJECTS

Water Quality/Eutrophication Assessment

Mathematical Modeling of Water Quality in Lake Erie

Client U.S. Environmental Protection Agency, Grosse Ile, Michigan

Project Engineer in charge of data analysis development and calibration of an eutrophication model including multiple algal species and zooplankton, and projections of the effects of reduction in point and non-point nutrient loadings on pollution indicators; lake phytoplankton, nutrient, and dissolved oxygen levels.

Assessment of the Environmental Fate and Impact of ICE-B-GON on Lake Wingra, Wisconsin

Client: Chevron Research Company

Principal investigator for the laboratory determination of the degradation and oxygen utilization kinetics of the de-icing chemical, ICE-B-GON and projection of the effect of the use of this chemical on the dissolved oxygen of receiving waters using Lake Wingra as a case study.

Total Maximum Daily Load (TMDL) Investigations

San Francisco Bay PCBs

Client: General Electric Company

Principal investigator for the review and critique of a draft TMDL document issued by the San Francisco Bay Regional Water Quality Control Board. This study involved the analysis of data and modeling to provide the Board with the information necessary to correct deficiencies in the draft document with regard to natural recovery and the need for, and effectiveness of, available source control options and to develop an effective implementation strategy. It included the development of presentation materials and a face-to-face meeting with the authors of the document.

Coosa River PCBs

Client: General Electric Company

Principal investigator for the review and critique of a draft TMDL document issued by the State of Georgia. This study involved the analysis of data to provide the State with the information necessary to correct deficiencies in the draft document with regard to natural recovery and the need for, and effectiveness of, available source control options and to develop an effective implementation strategy. It included the development of presentation materials and a face-to-face meeting with the State and with EPA Region 4.

Contaminated Sediments Assessment and Management

Peer Review of Contaminated Sediment Remediation Guidance for Hazardous Waste Sites

Client: USEPA

One of three national experts tasked with reviewing the draft guidance document which has been developed to provide technical and policy guidance to project managers and management teams making remedy decisions for contaminated sediment sites.

Investigation of Mercury in Lavaca Bay

Client: Alcoa

Principal investigator for the evaluation of mercury sources and prediction of the impacts of remedial actions and storm events on mercury levels in sediment and biota. The project involves data analysis and the development of linked hydrodynamic, sediment transport, mercury fate and bioaccumulation models. A primary goal is the evaluation of the impact of hurricanes and other rare storms on buried mercury.

Analysis of DDE and PCB Transfer Pathways in the Southern California Bight Ecosystem

Client: National Oceanic and Atmospheric Administration

Principal investigator for the analysis of data and development of food chain models to study the relationship between sediment contamination and levels of DDE and PCBs in fish, mammals, and birds. The purpose of this work was to establish probable sources of contamination in support of a Natural Resource Damages Assessment.

Analysis of the Fate of PCBs in the Hudson River

Client: General Electric Company

Principal investigator for extensive data analysis and modeling studies of the dynamics of PCBs in the Hudson River. This study involved field sampling, data analysis and the development of linked hydrodynamic, physical/chemical, sediment transport and food chain models for the purpose of predicting the effects of alternative remediation plans.

Pathogen Fate and Transport

Modeling Fate and Transport of Pathogenic Organisms in Mamala Bay, Hawaii

Client: Mamala Bay Study Commission

Principal investigator for review of historical data, design of a sampling program and development and calibration of a mathematical model of pathogen fate in Mamala Bay. Goal is to determine pathogen sources and level of control necessary to meet water quality goals.

Evaluation of Cryptosporidium Sources and Fate in Milwaukee, Wisconsin

Client: Sara Lee Corporation

Principal investigator for the evaluation of the likely contribution of various potential sources to the Cryptosporidium responsible for a disease outbreak in the city of Milwaukee.

HONORS

Manhattan College Environmental Engineering Alumni Club Service Award, 1994.
Diplomate Environmental Engineer by Eminence, American Academy of Environmental Engineers, 2002

This panel does not appear to be represented by scientists who were actively investigating the effects of DDT on avian wildlife. The recommendation of 1,000 ppb appears to be based on laboratory studies in less sensitive species. The dose levels in these studies were intentionally high to be sure to cause eggshell thinning and reproductive failure. None of the studies attempted to establish a chronic threshold for these effects. The panel admits that their recommendation, reproduced below, may not protect all species.

Substances Acting After Magnification in Food Chains

Chlorinated Hydrocarbon Pesticides

DDT and Derivatives DDT and its abundant derivatives DDE and TDE have high lipid solubility and low water solubility, and thus tend to concentrate in the lipid, i.e., living fraction of the aquatic environment (Hartung 1967b).[568] DDE is the most stable of the DDT compounds and has been especially implicated in producing thinning of egg shells, increased breakage of eggs, reproductive failure in species occupying the apex of aquatic food chains in areas with long histories of DDT usage.

Reproductive failures and local extirpation associated with egg shell thinning have been reported for several North American bird species. The phenomenon was first described and is most wide-spread for the peregrine falcon (*Falco peregrinus*) (Hickey and Anderson 1968).[574] Since then similar phenomena have been described in Brown Pelicans (*Pelecanus occidentalis*) (Anderson and Hickey 1970)[331] and species of several other families of predatory birds. Further increases of DDE in large receiving basins, such as the Great Lakes, would be expected to increase the extent of reproductive failure among predatory aquatic bird populations. Concentrations as low as 2.8 ppm *p,p'*DDE on a wet-weight basis produced experimental thinning of egg shells in the American Kestrel (*Falco sparverius*) (Wiemeyer and Porter 1970).[599] Heath et al. (1969)[572] induced significant levels of eggshell thinning in mallards after feeding them similarly low levels of DDE. Concentrations of DDT compounds in the water of Lake Michigan have been estimated to be 1 to 3 parts per trillion (Reinert 1970)[569] (Table III-21). Concentrations that would permit the assured survival of sensitive predatory bird species are evidently much lower than that. Because such low concentrations cannot be reliably measured by present technologies and because the concentrating factor for the food chains appears to be variable or is not known, or both, a biological monitoring system should be chosen. If it is desired to protect a number of fish-eating and raptorial birds, it is essential to reduce the levels of DDE contamination, especially in large receiving basins (see Section IV).

The available data indicate that there should not be concentrations greater than 1 mg/kg of total DDT in any aquatic plants or animals in order to protect most species of aquatic wildlife. Present unpublished data indicate effects for even lower levels of DDE to some species of predatory birds (Stickel *unpublished data*).[601]

Present environmental levels vastly exceed the recommended levels in many locations, and continued direct or indirect inputs of DDT would make these recommendations unattainable.

TABLE III-21—Relationship of DDT and Metabolites to Eggshell Thinning

Species	Dosage* wet-weight basis	Pesticide level in eggs	Thinning Percent	Reference
Mallard	1000 mg kg single dose	N.D.†	25	Tucker & Haegele, 1970[570]
Prairie falcon (Falco mexicanus)	N.D.†	8-14 ppm DDE	ca. 6	Enderson & Berger, 1970[569]
		16-20 ppm DDE	ca. 13	
		26-30 ppm DDE	ca. 18	
		36 ppm DDE	ca. 25	
Japanese quail (Coturnix)	100 ppm o,pDDT	23.8 ppm o,pDDT + 53 ppm DDE	4	Gibbs et al., 1969[540]
	100 ppm p,p'DDT	47.0 ppm p,p'DDE	6	
Herring gull (Larus argentatus)	ca. 3.3 ppm total DDT	227 ppm total DDT	N.D.†	Keith, 1966[545]
American kestrel (Falco sparverius)	2.8 ppm p,p'DDE	37.4 ppm DDE	10	Wiemeyer & Porter, 1970[599]
Mallard	**2.8 ppm DDE	N.D.†	11	Heath et al., 1969[572]
	**11.2 ppm DDE	N.D.†	14	

* All tests except the first one are chronic, spanning at least several months.
** Converted from dry-base.
† Not determined.

Recommendation

In order to protect most species of aquatic wildlife, the total DDT concentration on a wet-weight basis should be less than 1 mg/kg in any aquatic plants or animals. (Also see Recommendations for Pesticides, p. 185–186.)

Based on what was known in 1972, the recommendation of 1,000 ppb in fresh water fish to protect wildlife appears to be too high.

MARINE FISH GUIDANCE

The panel for marine fish guidance had one member, Robert Risebrough, who was an active investigator of the effects of DDT on eggshell thinning and reproduction in birds. The chairman, one other member of the panel and 3 advisors to the panel were from Woods Hole Oceanographic Institution, giving the panel a New England orientation. The recommendation follows:

DDT Compounds

DDT compounds have become widespread and locally abundant pollutants in coastal and marine environments of North America. The most abundant of these is DDE [2,2-bis(p-chlorophenyl) dicholoroethylene], a derivative of the insecticidal DDT compound, p,p'-DDT. DDE is more stable than other DDT derivatives, and very little information exists on its degradation in ecosystems. All available data suggest that it is degraded slowly. No degradation pathway has so far been shown to exist in the sea, except deposition in sediments.

Experimental studies have shown that DDE induces shell thinning of eggs of birds of several families, including Mallard Ducks (*Anas platyrhynchos*) (Heath et al. 1969),[48] American Kestrels (*Falco sparverius*) (Wiemeyer and Porter 1970),[77] Japanese Quail (*Coturnix*) (Stickel and Rhodes 1970)[66] and Ring Doves (*Streptopelia risoria*) (Peakall 1970).[57]

Studies of eggshell thinning in wild populations have reported an inverse relationship between shell thickness and concentrations of DDE in the eggs of Herring Gulls (*Larus argentatus*) (Hickey and Anderson 1968).[50] Double-crested Cormorants (*Phalacrocorax auritus*) (Anderson et al. 1969),[31] Great Blue Herons (*Ardea herodias*) (Vermeer and Reynolds 1970),[70] White Pelicans (*Pelecanus erythrorhynchos*) (Anderson et al. 1969),[31] Brown Pelicans (*Pelecanus occidentalis*) (Blus et al. 1972;[36] Risebrough *in press* 1972),[62] and Peregrines (*Falco peregrinus*) (Cade et al. 1970).[27]

Because of its position in the food webs, the Peregrine accumulates higher residues than fish-eating birds in the same ecosystem (Risebrough et al. 1968).[64] It was the first North American species to show shell thinning (Hickey and Anderson 1968).[50] It is therefore considered to be the species most sensitive to environmental residues of DDE.

The most severe cases of shell thinning documented to date have occurred in the marine ecosystem of southern California (Risebrough et al. 1970)[64] where DDT residues in fish have been in the order of 1--10 mg kg of the whole fish (Risebrough *in press* 1972).[62] In Connecticut and Long Island, shell thinning of eggs of the Osprey (*Pandion haliaetus*) is sufficiently severe to adversely affect reproductive success; over North America, shell thinning of Osprey eggs also shows a significant negative relationship with DDE (Spitzer and Risebrough, *unpublished results*).[78] DDT residues in collections of eight species of fish from this area in 1970 ranged from 0.1 to 0.5 mg/kg of the wet weight (Hays and Risebrough 1972).[47] Evidently this level of contamination is higher than one which would permit the successful reproduction of several of the fish-eating and raptorial birds.

Recommendation

It is recommended that DDT concentrations in any sample consisting of a homogenate of 25 or more fish of any species that is consumed by fish-eating birds and mammals, within the same size range as the fish consumed by any bird or mammal, be no greater than 50 µg/kg of the wet weight. DDT residues are defined as the sum of the concentrations of p,p'-DDT, p,p'-DDD, p,p'-DDE and their ortho-para isomers.

At the time of this recommendation, Robert Risebrough had just published an article with Helen Hays on DDT in terns and fish scraps on Great Gull Island 6 miles off the Connecticut coast in Long Island Sound. The 1970 fish data in this study became the basis for the 50 ppb recommendation.

TABLE 2
DDT AND PCB RESIDUES IN FISH BROUGHT BY TERNS TO THE GREAT GULL ISLAND COLONY

Species	N	Mean weight (g)	ppm, fresh weight p,p'-DDE	p,p'-DDD	p,p'-DDT	PCB	ppm, lipid[1] DDT	PCB	DDT/PCB
Alosa aestivalis									
Blueback herring	5	12.4	0.22	0.18	0.011	0.64	6.4	10	0.64
Brevoortia tyrannus									
Atlantic menhaden	7	0.5	0.10	0.037	0.012	0.27	—	—	0.57
Clupea harengus									
Atlantic herring	2	3.3	0.022	0.027	0.00	0.38	—	—	0.13
Etrumeus teres									
Atlantic round herring	10	8.0	0.21	0.11	0.008	1.2	8.3	30	0.28
Anchoa mitchelli									
Bay anchovy	17	2.6	0.15	0.060	0.011	1.1	14	69	0.20
Menidia menidia									
Atlantic silverside	10	6.7	0.28	0.25	0.024	3.2	9.1	52	0.17
Morone americanus									
White perch	2	6.2	0.013	0.007	0.004	0.88	4.8	176	0.027
Scomber scombrus									
Atlantic mackerel	19	4.3	0.034	0.022	0.007	1.2	4.2	79	0.053

[1] Concentration of DDT is the sum of the concentrations of p,p'-DDE, p,p'-DDD and p,p'-DDT.

The DDT measured in the terns and in scraps of fish cast from their nests on Great Gull Island was not reported to have any affect on the terns. However, other reports clearly established the breeding failure of ospreys along the Connecticut coast and on nearby Gardiner Island. The implied assumption in the panel's recommendation is that the ospreys would be eating the same fish with the same level of residues found on Great Gull Island, and therefore that level was clearly toxic. What the summary didn't say was that the ospreys tend to feed along the coast and up the estuaries, resulting in a fish diet quite different from that of the terns. For example, osprey feeding patterns at a location further north are discussed in a report by Greene et al. (1983), part of which is shown below:

RESULTS

Four fish species, i.e. Alewife, Smelt, Pollock and Winter Flounder, comprised more than 94% of the positively identified fish (n = 610) caught by Ospreys. These 4 species varied in spatial and temporal availability to Ospreys over the breeding season. From late April to mid-June, both Alewives and Smelt were highly localized in a few freshwater spawning areas (Fig. 1). During their spawning runs Alewives and Smelt entered the rivers simultaneously in abundant numbers. Schools of fast-swimming juvenile Pollock occurred in Cow Bay and Cole Harbour estuaries from late May until mid-July. By late July, these "harbour Pollock" move into deeper waters (Steele 1963), becoming unavailable to Ospreys. Winter Flounder are cryptic benthic fish occurring on sandy or muddy substrates. The temporal availability of these fish to Ospreys is summarized in Fig. 2.

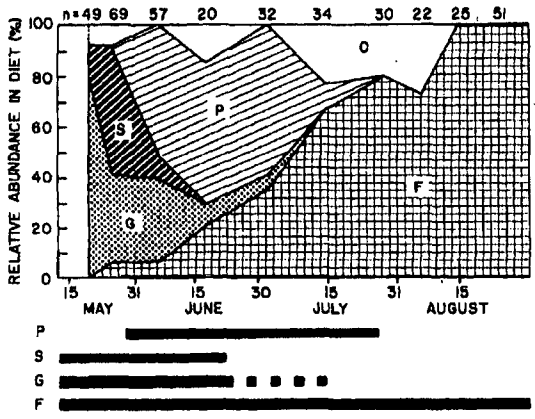

FIGURE 2. Temporal availability of major prey species to Ospreys. G = Alewife, S = Smelt, P = Pollock, WF = Winter Flounder, O = other.

Ospreys used different foraging sites at different times of the breeding season. This shift of foraging sites was accompanied by a marked change in diet. Fig. 2 shows the seasonal change in species caught by Ospreys in Cow Bay. Similar patterns occurred for Ospreys nesting in Cole Harbour (221 observations). In late April and May, Alewives and Smelt were apparently preferred over Winter Flounder; Ospreys travelled up to 10 km inland to hunt for Alewives, and to a lesser extent, Smelt on their spawning grounds. During this period, virtually no fishing was observed along the coast, even though Winter Flounder were present and presumably available. Alewives were taken occasionally and with decreasing frequency as they returned to the ocean from their spawning grounds. During June and July, both Pollock and Winter Flounder were caught, but Ospreys apparently preferred the former. When an Osprey captured a Pollock, indicating that a school was present, other Ospreys fishing over mud flats for Winter Flounder would immediately fly to the other location. Capture of 1 Pollock often stimulated a fishing "frenzy" with as many as 15 Ospreys hovering over the Pollock school. These fishing frenzies usually lasted less than 15 min because Pollock quickly swam away. The hypothesis that Pollock were selected over Winter Flounder was tested using a dichotomized runs test (Sokal and Rohlf 1969). In 7 of 8 feeding periods in which only Pollock and Winter Flounder were caught, Pollock were captured in runs that were significantly non-random ($P < 0.05$). After Pollock moved offshore, Winter Flounder accounted for over 95% of the fish caught.

Other fish eaten by Ospreys but constituting less than 6% of the diet were Cod (*Gadus morhua*), Tomcod (*Microgadus tomcod*), Sculpin (*Myoxocephalus* spp.), and Mummichog (*Fundulus heteroclitus*). Fishermen also reported seeing Ospreys fishing for Mackerel (*Scomber scombrus*), up to 1.5 km offshore during the fall run.

One could conclude from this and other studies that ospreys often catch fish from fresh or brackish water and, therefore, may not have been the best species for assessing the reproductive effect of DDT residues in marine fish.

Fish from the nearby Connecticut River have much higher residues of DDT than the fish cast from tern nests on Great Gull Island, as shown below from Henderson et al. (1971).

TABLE 4.—*Organochlorine insecticide residues in fish—mean values 1968 and 1969 samples*

Station Number and Location		DDT and Metabolites (PPM)[1]		
		Fall 1969	Fall 1968 [2]	Spring 1968 [2]
		ATLANTIC COAST STREAMS		
#1	Stillwater River	.20	.14	.30
#2	Connecticut River	1.55	3.27	.85
#3	Hudson River	2.65	10.10	2.33
#4	Delaware River	10.95	15.66	16.85
#5	Susquehanna River	.68	.98	.92
#6	Potomac River	.60	1.38	.32
#7	Roanoke River	.98	.90	.42
#8	Cape Fear River	1.40	1.23	.49
#9	Cooper River	1.74	2.59	2.91
#10	Savannah River	.63	.59	.40
#11	St. Johns River	.15	.26	.21
#12	St. Lucie Canal	[3] 19.93 [3] (1.01)	3.69	2.52
		GULF COAST STREAMS		

In addition, Ames and Mersereau (1964) reported total DDT levels of 2.5-9.2 ppm in scraps of fish cast from osprey nests on Great Island near the mouth of the Connecticut River. Also, ospreys feeding in the Connecticut River estuary in 1967 were poisoned by dieldrin (Wiemeyer et al., 1975). These facts were known in 1972 and should have been considered by the panel and mentioned in the recommendation. The recommendation of 50 ppb did not take into account all of the available information and may be lower than the guidance that may have come forth, had all of the facts been considered.

The above information sets the stage for considering the adoption of the NAS fresh water and marine fish recommendations for use today, some 34 years later. Much has been learned about DDT and its effects on wildlife since 1972. The feeling among investigators in 1972 was concern, frustration, and even outrage at what was happening to avian species at the top of food chains. Within only a few years, however, recovery was well underway, and by 1980 was nearly complete in many species. The study of the recovery of the sensitive avian species gives us an indication of toxicity thresholds for DDT residues in fish diets. The results of such studies provide a way of observing dose-response over time as residues slowly declined. However, the relationship between fish and egg residues became less certain as levels in the United States declined below probable but unknown levels on wintering grounds in Latin America where DDT use continues today. The focus of this report will be on recovery of the ospreys, since this species is key to the NAS panel's recommendation for marine fish. Subsequent reports will deal with the fresh water fish recommendation and other sensitive avian species.

CHRONOLOGY OF OSPREY STUDIES

Ames and Mersereau (1964) and Ames (1966) reported on the status of the osprey along the Atlantic coast. Most populations were experiencing dramatic declines associated with poor hatching and fledgling rates. Eggs and fish remnants from nests on Great Island at the mouth of

the Connecticut River were assayed for DDT and metabolites in 1962. Eggs contained an average of 8.1 ug/ml (about 9 ppm fresh weight) total DDT and fish remnants cast from the osprey nests contained 2.5-9.2 ppm total DDT. A crude biomagnification factor would be 9/5.7 = 1.6.

In 1963, Ames (1966) again studied osprey eggs from Great Island, but also did a comparison with eggs from Maryland, where Ospreys were experiencing greater reproductive success. A few eggs from other locations along the Atlantic coast were also analyzed for DDT. The results are shown below.

Table 2. *DDT and its metabolites in the eggs of Ospreys from the north-eastern United States*

Locality	Year	No. of eggs	Average volume (ml)	DDE		DDD		DDT		Total residues	
				μg	μg/ml	μg	μg/ml	μg	μg/ml	μg	μg/ml
Maine	1963	3	72	120	1·7	7	0·1	5	0·06	130	1·8
Rhode Island	1963	1	68	500	7·4	100	1·5	ND	ND	600	8·8
Connecticut	1962	6	68	450	6·7	100	1·5	Trace	Trace	550	8·1
Connecticut	1963	15	68	320	4·7	20	0·3	10	0·1	350	5·1
New Jersey	1963	2	Not measured	350	5·1	40	0·6	10	0·1	400	5·9
Maryland	1963	25	70	160	2·3	40	0·6	5	0·07	205	3·0

ND = None detected.

The Connecticut eggs contained an average of 5.1 ug/ml total DDT compared to 3.0 ug/ml in the Maryland eggs. Ames also collected fish from osprey nests in the Maryland and Connecticut studies as shown below in his Table 3.

Table 3. *DDT residues in fish samples from Connecticut and Maryland*

Species	No. of individuals	Total wet weight (g)	DDE		DDD		DDT		Total residues	
			μg	ppm	μg	ppm	μg	ppm	μg	ppm
CONNECTICUT										
Black-backed Flounder	6	376	160	0·4	30	0·1	300	0·8	490	1·3
Windowpane Flounder	2	70	50	0·7	10	0·1	140	2·0	200	2·9
Alewife	4	60	20	0·3	10	0·2	100	1·7	130	2·2
Shad	1	70	80	1·1	40	0·6	100	1·4	220	3·1
Cunner	1	19	Trace		Trace		60	3·1	60	3·1
Eel	1	40	80	2·0	40	1·0	100	2·5	220	5·5
MARYLAND										
Eel	4	572	60	0·1	110	0·2	60	0·1	230	0·3
Yellow Perch	3	256	20	0·1	10	0·04	30	0·1	60	0·2
White Perch	2	93	Trace		Trace		Trace		5	0·05
Striped Killifish	1	22	Trace		Trace		Trace		5	0·1
Menhaden	2	125	Trace		Trace		Trace		5	0·05
Toadfish	1	140	20	0·1	10	0·1	10	0·1	40	0·3

The Connecticut fish residues ranged from 1.3 to 5.5 ppm total DDT, whereas the Maryland fish residues ranged from 0.05 to 0.3 ppm total DDT. The differences in DDT in fish diet, in eggs and reproductive success between the two colonies, is the first report of this kind. The results provide the first indications of the relationship between levels of DDT in the fish diet, in the egg, and hatching success. A crude biomagnification factor for Connecticut osprey in 1963, based on a weighted average fish residue of 2.1 ppm is 5.7/2.1 = 2.7. For the Maryland data, again using a weighted average fish residue, a crude estimate of the bioconcentration factor from fish to egg is 3.3/0.23 = 14. The increase in biomagnification factor with declining fish residues could be the result of slow equilibration between dietary residues and adipose residues in the osprey and/or dietary sources higher in DDT than the fish that were measured. Because of the second possibility, greater weight should be given to fish data based on scraps from osprey nests. Even this data is subject to limitations, however, because what is measured is what the osprey didn't eat and often the remnants are dehydrated, resulting in higher residues than fresh weight. Let us continue on with reports from other investigators documenting the decline in osprey populations.

Peterson and others (1969) reported on declining populations of ospreys in the United States and Europe. The declines were mostly the result of hatching failure and were attributed to pesticides. Henny and Ogden (1970) reported on the breeding success and status of osprey populations in seven states as summarized in their Table 1 below:

Table 1. The estimated present status of osprey populations in portions of seven states. The complete nesting populations of each state were not sampled, thus the total number of active nests presented in this table does not represent the size of the breeding populations and may not represent the status of the complete population in each state.

STATE	NO. ACTIVE NESTS (ALL YEARS SUMMED)	YEAR OF STUDY	NO. FLEDGED PER ACTIVE NEST	PERCENT NESTS SUCCESSFUL	ESTIMATED (MINIMAL) ANNUAL RATE DECLINE (PERCENT)	SOURCE OF NESTING STUDY
Florida	83	1968–69	1.22	70	stable	This paper
Minnesota	161	1966–68	1.03	65	2–3	Dunstan 1968
Maryland[b]	136	1964–65	1.03	54	2–3	Reese 1965
Wisconsin	128[a]	1952–59	0.98	53	3–4	Berger and Mueller 1969
Wisconsin	67	1960–65	0.39	30	12–13	Berger and Mueller 1969
Michigan	162	1965–67	0.39	23	12–13	Postupalsky 1969
Maine	8	1964	0.38	25[c]	12–13	Kury 1966
Connecticut	157	1960–63	0.29	23[d]	13–14	Ames and Mersereau 1964
Connecticut	30	1964–65	0.27	27[d]	13–14	Peterson 1969

[a] No data for 1957.
[b] Reese (Personal communication 1968) stated the first year of the study (1963) was preliminary and not as reliable as the following years. It was omitted.
[c] Kury (Personal communication 1969).
[d] Maximum percent of nests successful, assuming one young fledged per successful nest.

Reese (1977) reported on productivity all across the United States for the period 1966-74 as shown in his Table 8 below:

TABLE 8
RECENT NEST SUCCESS IN U.S. OSPREY POPULATIONS[1]

Location	Years	Nests	Nests Successful	Young Produced	Brood Size	Fledglings per nest	Reference
S. Massachusetts	1970–74	73	42	82	1.9	1.12	Fernandez (pers. comm.)
Chesapeake Bay:							
Eastern Bay	1966–74	323	128	229	1.8	0.71	Reese (1975)
This study	1970–74	684	386	741	1.9	1.08	
Choptank River	1968–74	188	106	190	1.8	1.01	Reese (1972, MS)
Smith Island	1968–71	71	55	98	1.8	1.38	Rhodes (1972)
Potomac River	1970–71	237	81	135	1.7	0.57	Wiemeyer (1971, 1977)
Virginia	1970–71	416	203	333	1.6	0.80	Kennedy (1971)
Michigan	1969–74	463	205	405	2.0	0.88	Postupalsky (1977 and pers. comm.)
Wisconsin	1966–69	237	111	193	1.7	0.81	Sindelar (1971)
Minnesota (Chippewa Nat. For.)	1968–72	249	120	216	1.8	0.87	Mathisen (1973)
Wyoming (Yellowstone Nat. Park)	1972–74	107	44	68	1.5	0.64	Swenson (1975)
Montana (Flathead Lake)	1967–70	80	42	77	1.8	0.96	Koplin (pers. comm.)
N. Idaho-E. Washington	1972–73	342	233	481	2.1	1.41	Melquist (1974)
Oregon (Deschutes Nat. For.)	1971	52	31	60	1.9	1.15	Lind (1971)
N. California	1969–71	136	71	139	2.0	1.02	Garber (1972)

[1] Data for all except this study were collected by two or infrequent nest visits and may not allow for mortality between final visit and fledging. Unpublished data are subject to revision.

Studies in other species soon identified eggshell thinning as the primary lesion causing hatching failure. DDE was shown to cause eggshell thinning in numerous declining species, including the osprey. Anderson and Hickey (1972) reported 21 % shell thinning in osprey eggs collected in Connecticut, New Jersey and Maryland in 1957.

Johnson et al. (1975) reported 17 % shell thinning in osprey eggs taken in Idaho in 1972 and 1973. Total DDT in eggs averaged 10.3 ppm. Hatching success was impaired. No fish residue measures were made. The general lack of use of DDT in the nesting grounds led the authors to suggest that exposure to DDT had occurred primarily during migration or at wintering grounds in Central America.

By 1973, fish residues, egg residues, eggshell thinning and hatching success appear to be the critical determinants of the effect of DDE on osprey reproduction. All four parameters are highly correlated in declining species with exposures sufficient to cause eggshell thinning in excess of 10 %. Mechanistic studies suggested that DDE acts directly on the transport, formation and/or deposition of calcium carbonate in the shell gland (e.g., see Risebrough et al., 1969).

Weimeyer et al. (1975) evaluated known factors impacting reproduction in East Coast ospreys. The study period was 1968-69. An egg exchange between nests in Maryland and Connecticut revealed that Connecticut eggs had lower hatching success than Maryland eggs whether they remained in Connecticut or were moved to nests in Maryland. Just the opposite, Maryland eggs had higher hatching success than Connecticut eggs whether they remained in Maryland or were moved to nests in Connecticut. The problem appears to be the egg and not the parents or the setting. This finding is consistent with the direct effect of DDE on the shell gland to produce thinner shelled eggs that are more susceptible to breakage and therefore lower hatching success. DDE levels are higher in fish in some breeding areas than others, explaining the differential productivity along the East Coast.

Fish collected in Connecticut waters contained an average total DDT residue of 2.0 ppm. Fish collected in Maryland averaged 0.2 ppm total DDT. Fish scraps from osprey nests in Connecticut averaged 1.0 ppm, whereas one eel scrap from a nest in Maryland had 0.1 ppm total DDT. Fish scraps were judged to be very slightly dehydrated. Henderson et al. (1971), reported total DDT residues for 1969 in fish of 0.68 ppm for the Susquehanna River and 0.60 for the Potomac River. Both rivers flow into the Chesapeake Bay. Sampling locations on both rivers were in Maryland.

Total DDT in Connecticut osprey eggs collected in 1968-69 was 10.3 ppm. This residue level compares with 10.9 ppm in 1964. Egg residues of total DDT from Maryland averaged 3.1 ppm. Eggshell thinning averaged 15 % in Connecticut eggs and 12 % in Maryland eggs. Only two eggs hatched out of 25 eggs studied in Connecticut. Fifteen eggs hatched out of 38 eggs studied in Maryland. Dieldrin may have contributed to hatching failure in Connecticut. Lethal concentrations of dieldrin were measured in a dead adult osprey found near the Connecticut River in 1967. Crude estimates of biomagnification from fish to egg were 10.9/(2.0 or 1.0) = 5.4 – 10.9 for Connecticut and 3.1/(0.68-0.1) = 4.6 – 31 for Maryland.

In a 1972 study done on an offshore island along the Gulf coast of Florida, Szaro (1978) reported an average of 0.11 ppm total DDT in fish (lipid basis converted to fresh weight assuming 5 % lipid), an average of only 0.43 ppm total DDT in eggs, a 9 % thinning of eggshells and 0.73 young per female. The lower than normal reproductive success was not attributed to the eggshell thinning, which was described as near normal. The fish were scraps taken from the same nests as the eggs. The fish muscle was analyzed. A crude biomagnification factor can be calculated as 0.43/0.11 = 3.8. Whole fish would undoubtedly give a lower biomagnification factor. The population of ospreys in Florida is not migratory, remaining in Florida year-round.

Wiemeyer et al. (1978) reported on studies on osprey reproduction in New Jersey in the years 1970-1974. The egg residue levels and a summary of the population status in comparison with other osprey breeding areas are shown in their Table 2 below:

TABLE 2

Relationships between primary residues in eggs, population trends, and reproductive success of different osprey populations

Population	Year	Average Residues in Eggs (ppm wet weight) p,p'-DDE	Dieldrin	PCB's	Population Trend and Reproductive Success
Potomac River, Maryland[a]	1968-69	2.4	0.25	2.6	Stable population; reproduction slightly depressed.
Lake Coeur d'Alene, Idaho[b]	1972-73	8.5	n.d.	1.2	Stable or increasing population; reproduction normal.
Connecticut[a]	1968-69	8.9	0.61	15.	Declining population; reproduction greatly depressed.
Barnegat Bay Area, New Jersey	1974	16.	0.07	9.0	Declining population; reproduction greatly depressed.
Avalon-Stone Harbor, New Jersey	1970 + 72	14.	0.20	8.8	Declining population; reproduction greatly depressed.

[a] From WIEMEYER et al. (1975).
[b] From JOHNSON et al. (1975); n.d. = not detected.

Fish residue data were not reported. Eggshell thinning is summarized in Table 3 below:

TABLE 3

Changes in shell thickness of New Jersey osprey eggs.

Area	Year	Sample Size[a]	Average Shell Thickness ± 95% CL[b]	% Change from pre-1947
Eastern U. S.[c]	pre-1947	365 (-)	0.505 ± 0.004	--
Barnegat Bay Area	1971	2 (2)	0.485 ± 0.064 (0.48 - 0.49)	-4
Barnegat Bay Area[d]	1974	7 (4)	0.408 ± 0.073 (0.34 - 0.44)	-19
Avalon-Stone Harbor Area[d]	1970 + 72	8 (8)	0.443 ± 0.024 (0.40 - 0.49)	-12

[a] Number of eggs measured; number of clutches represented in parentheses.
[b] Means for current samples are on a clutch basis, while that for pre-1947 is on an egg basis. Complete clutches are usually represented in museum collections (pre-1947), whereas most recent samples are single eggs from clutches. Extremes of clutch means in parentheses. CL = confidence limits.
[c] From ANDERSON and HICKEY (1972).
[d] The eggs represented here are different in part from those that were analyzed for pollutants, as reported in Table 1; see text.

Up until 1974, these breeding populations had high residue levels and poor productivity.

The first report of a significant recovery of ospreys was by Spitzer et al. in 1978, six years after the ban of DDT. Robert Risebrough was an author on this report. Eggs collected from osprey populations in Connecticut and eastern Long Island from 1967 to 1970 had 15-20 % thinning, approximating the critical level associated with hatching failure in other species. DDE levels in osprey eggs from this area declined 5-fold between 1969 and 1976 and 3-fold between 1973 and 1976. "The productivity of these ospreys has since increased from about 0.5 fledged young per pair in 1969 to 1973 to 1.2 fledged young in 1976-1977 (Fig. 1), approaching the range observed in 1938-1942."

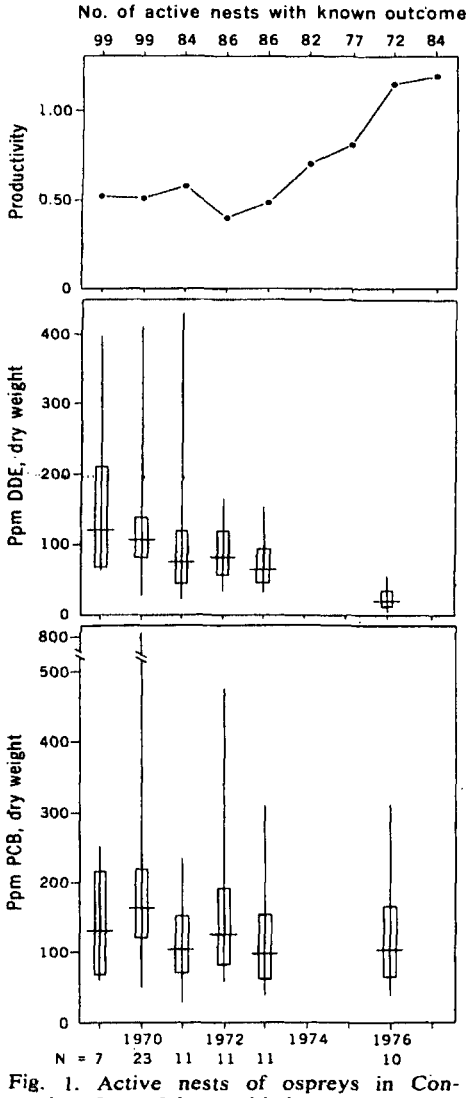

Fig. 1. Active nests of ospreys in Connecticut–Long Island with known outcome, 1969 to 1977; productivity, defined as young fledged per active nest; DDE and PCB residues, parts per million dry weight, with the sample sizes. Horizontal bars are geometric means; rectangles are the 95 percent confidence intervals of the means; vertical lines are the sample ranges.

Productivity improved when DDE residues in eggs fell below 12 ppm (60 ppb dry weight), a finding that is consistent with those of Henny et al. (1977) for other areas. The authors acknowledged that dieldrin probably affected survival and reproduction of ospreys in the Connecticut River estuary. No fish residue data were reported.

MacCarter and MacCarter (1979) reported improving reproduction in osprey at Flathead Lake in Montana even in the face of high egg residues of DDT, as shown in their Table 2 below:

TABLE 2. Residues of DDT and metabolites, dieldrin, and polychlorinated biphenyls (PCB) in addled Osprey eggs from Flathead Lake, Montana.

			Residues (ppm. wet weight)					
			DDT and metabolites					
Year	Nest no.	Egg no.	DDE	DDD	DDT	Total	Dieldrin	Est. PCB
1968[a]	BI-1	1[b]	5.1	1.2		6.3	N/A	N/A
	BI-3	1	7.9	1.3		9.2	N/A	N/A
	DB-1	1	11.4	0.85		12.2	N/A	N/A
	DB-1	2	10.4	4.4		14.8	N/A	N/A
1969	BI-1	1	13.5	2.6		16.4	N/A	N/A
	BI-2	1	6.5	2.0		8.5	N/A	N/A
	BI-2	2	10.1			10.1	N/A	N/A
	DB-1	1	5.2			5.2	N/A	N/A
	DB-1	2	9.5			9.5	N/A	N/A
	BI-5	1	22.6			22.6	N/A	N/A
1970	BI-1	1	16.0		1.5	17.4	N/A	N/A
	BI-2	1	13.5		2.2	15.7	N/A	N/A
	BI-5	1	5.3		0.4	5.7	N/A	N/A
	BI-5	2	3.8			3.8	N/A	N/A
	N-D-1	1	5.9	0.6	1.7	8.2	N/A	N/A
1976[c]	BI-3	1	3.1	0.12		3.22		12.0
	BI-5*	1	37.0	3.3	0.35	40.65		3.3
	CB-8*	1	35.0	5.6		40.60		1.3
1977[c]	BI-3	1	2.9	0.14		3.04		3.3
	BI-5	1	16.0	1.2		17.20	0.05	2.2
	CB-8	1	8.7	1.1		9.8		1.3
	CB-8	2	11.0	1.2	0.20	12.40		0.74

[a]Dry weight converted to wet weight.
[b]Fresh egg collected accidentally.
[c]Eggs analyzed at Patuxent Wildlife Research Center, Laurel, Maryland (S. Wiemeyer et al., unpubl.).
N/A Not Analyzed.
*Cis chlordane detected (0.12) in BI-5 and 0.08 in CB-8.

From 1967 to 1977, the number of breeding adults gradually increased even though productivity was marginal as might be expected with the high levels of DDT residues.

TABLE 3. Nesting productivity of Ospreys at Flathead Lake, Montana.

Year	No. nesting pairs (A)	No. young (B)	No. young fledged (C)	No. nestlings per pair (B/A)	No. fledglings per pair (C/A)
1967	16	18	17	1.12	1.06
1968	20	14	14	0.70	0.70
1969	20	20	15	1.00	0.75
1970	24	33	31	1.38	1.29
1974	28	36	34	1.31	1.21
1975	30	41	38	1.37	1.27
1976	36	43	40	1.19	1.11
1977	38	38	36	1.00	0.95
Total Average	212	243	225	1.15	1.07

Eggshell thinning and fish residues were not reported.

A report by Spitzer et al. in 1983 gave further indication of the recovery of osprey breeding along the northeastern coast as shown in their Figure 2 below.

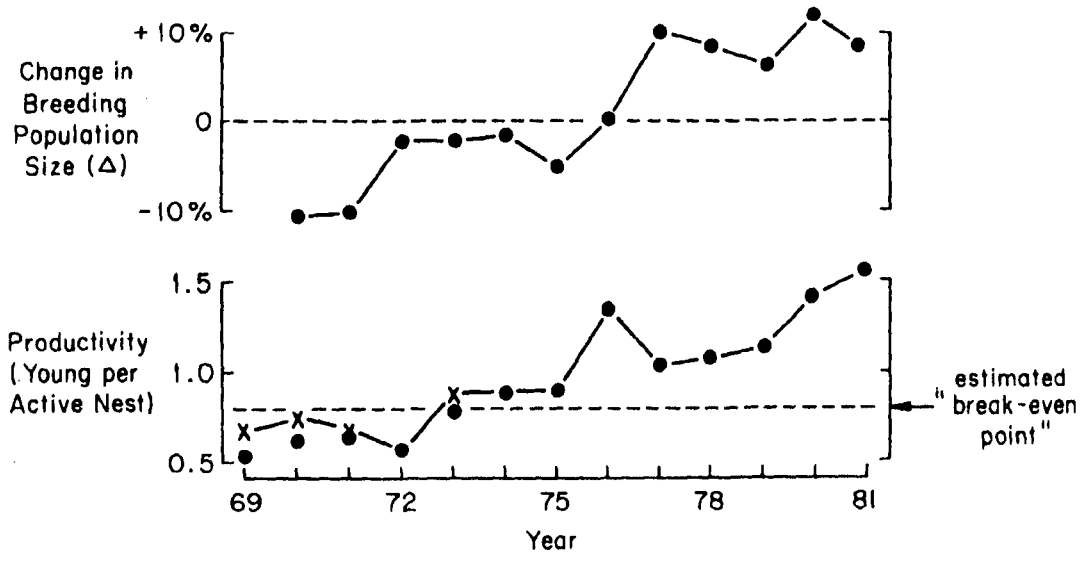

FIGURE 2. A comparison of Osprey reproductive rate and change in population size, N.Y. City to Boston, 1969-1981. Points denoted by "X" on the lower graph are productivity values which include young introduced from Maryland by Spitzer (1978).

The authors noted the lack of measures of DDE in osprey eggs since 1976. Presumably DDE residues were declining as reproduction improved.

They also made note of a brood-size reduction of 50 % or more due to food limitations on Gardiners Island, the island mentioned as impacted by DDT in the NAS recommendation for marine fish. Spitzer and Poole (1980) and Poole (1989) revisted the issue of the struggling population of ospreys on Gardiner Island. The population was decimated by DDT in the 1950s and 1960s. Local citizens took up the cause to save the osprey. They sued Suffolk County to stop spraying DDT for mosquito control and achieved a ban on eastern Long Island. This group later became the Environmental Defense Fund. One of their members, Dennis Puleston, was an author of the 1978 report (Spitzer et al.) on the recovery of osprey populations on eastern Long Island. Recovery of the osprey on Gardiner Island was well underway in the 1970s when reproduction failed again due to a limited food supply. Apparently male osprey had to travel long distances to reliable supplies of fish in the marshes of the south fork of Long Island. According to the authors, when this colony thrived it was dependent on menhaden in nearby Gardiner's Bay. Excessive commercial fishing removed this food source, leading to a marginal food supply.

Reporting on a national survey of osprey breeding in 1983, Henny stated: "Ospreys at locations with poor production have all showed improvement following the DDT ban in 1972."

Wiemeyer et al. (1988) reported DDT effects on osprey eggs and reproduction from several data sets generated in the 1960's and 1970's. Some declines in residue levels and shell thinning were noted. Analysis of the DDE egg residue - shell thinning relationship revealed 10 % thinning at 2.0 ppm, 15 % at 4.2 ppm and 20 % at 8.7 ppm. Reproductive failure was attributed to DDE causing thinning of eggshells. Ospreys were considered to be as sensitive as other sensitive species.

In his book on ospreys, Poole (1989) published a figure relating DDE residues in osprey eggs with eggshell thinning.

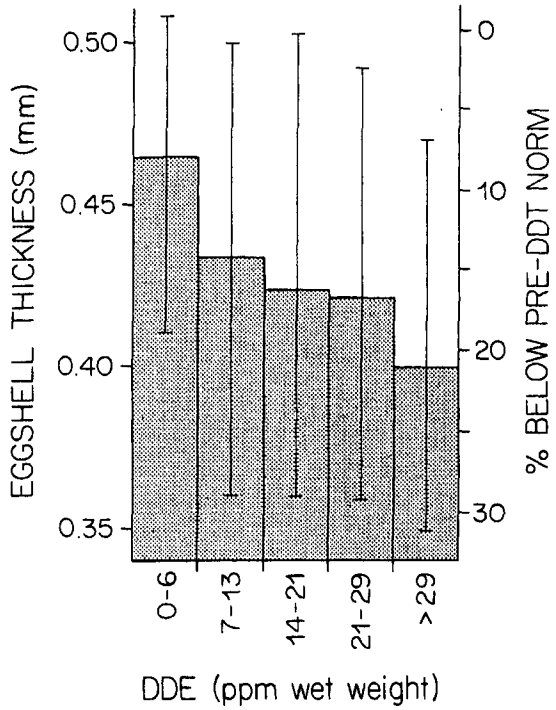

Figure 9.7. Osprey eggshell thickness in relation to DDE residues in the egg. Data shown are mean values (vertical lines show ranges) based on analyses of 112 eggs, most collected in the northeastern United States during the 1960s and 1970s. Data from Spitzer *et al.* (1978) and A. Poole & J. Farrington (unpublished).

Poole's data illustrates the wide variability in eggshell thinning at each residue level, explaining why populations increase even at levels of DDE that result in some shells breaking and failing to produce viable young. Reproductive failure and mortality due to high residues of dieldrin and PCBs, particularly in the 1960s and early 1970s, may account for some of this variablility. Poole also reported on the DDE egg residue – production dose-response as shown in his Figure 9.8.

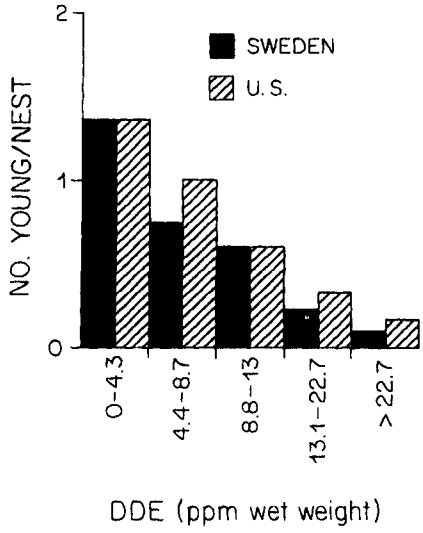

Figure 9.8. Mean brood size at Osprey nests in Sweden (1971–1973) and in New England (USA) (1969–1984), in relation to DDE levels in unhatched eggs from those nests. Swedish data, from Odsjö (1982), show young hatched per nest (N = 108 nests); US data, from Spitzer *et al.* (1978) and Poole & Farrington (unpublished), show young fledged per nest (N = 101 nests).

Poole sets the reproductive effect threshold at 4.3 ppm DDE. This number compares with the 15 % shell thinning suggested by Wiemeyer et al. (1988) at 4.2 ppm DDE.

Schmitt et al. (1990) published the results of a national fresh water fish residue survey for 1984. Total DDT residues in fish from the Connecticut River averaged 0.22 ppm. For all sites sampled nationwide, the trend of the geometric average total DDT residue was 0.39 ppm in 1976-77, 0.36 ppm in 1978-79, 0.32 ppm in 1980-81 and 0.28 ppm in 1984. Schmitt et al. (1981) had earlier published a nationwide level of 1.08 ppm in fish collected between 1970 and 1974. Bilger et al. (1999) discussed EPA analysis of multi-species composite analyses done in 1987. The mean DDE concentration was 0.295 ppm in a nationwide sampling. The USGS multi-species sampling of the lower Susquehanna River basin in 1992 (Bilger et al. [1999]) indicated median residues of 0.250 ppm of total DDT. Variability between sites was very high as shown in results for white suckers collected from the Susquehanna, Hudson and Connecticut River Basins in the authors Figure 2 below.

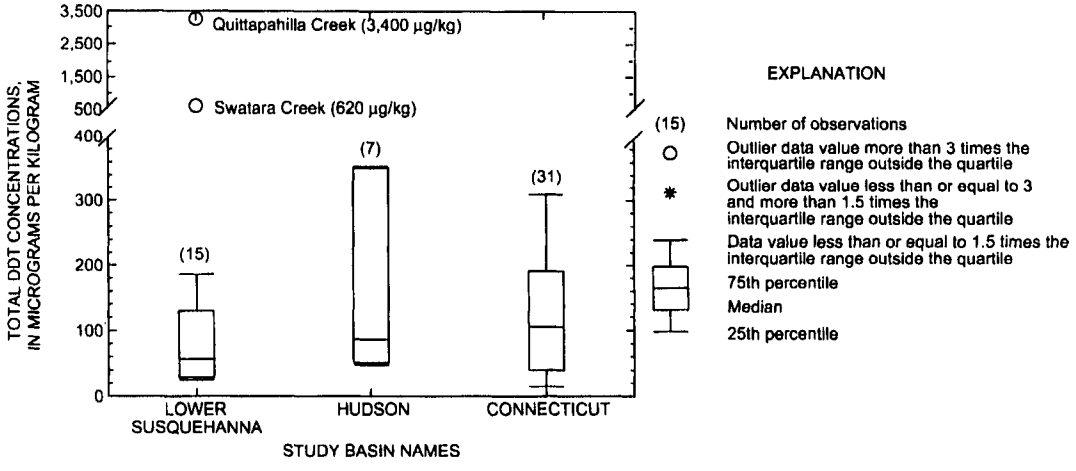

Figure 2. Concentrations of total DDT in white sucker whole fish tissue for the Lower Susquehanna, Hudson, and Connecticut River Basins.

The overall trend for DDT in fish residues in the 1970s and 1980s is a steady decline, although hot spots are clearly evident. If these hotspots are sources of food for ospreys and are missed in fish surveys, then the residue exposures may be greatly underestimated, resulting in an overestimate of biomagnification from fish to osprey egg.

In 1991, Steidl et al. published two papers on osprey reproduction in three regions of southern New Jersey. The three locations were the more polluted Delaware Bay, the less polluted Atlantic Coast and an intermediate location along the Maurice River that flows into the lower Delaware Bay. Eggs were collected in 1985-1989. The authors noted that average fish residue of total DDT in the Delaware River was 0.88 ppm in 1984. Total DDT residues in eggs were low with the highest levels in Delaware Bay as shown in part of the author's Table 1 below.

Table 1. Organochlorine and mercury residues (ppm fresh wet mass)

Region and egg type	n	DDE x̄[a]	DDE Range	DDD x̄	DDD Range
Delaware Bay					
Random[b]	7	3.2	1.7–5.2	0.4	0.3–0.7
Addled[c]	4	2.9	1.6–4.7	0.4	0.3–0.6
All	11	3.1		0.4	
Atlantic Coast					
Random	8	1.2	0.5–2.8	0.2	0.1–0.6
Addled[c]	4	1.6	1.4–1.8	0.2	0.2–0.3
All	12	1.4		0.2	
Maurice River					
Random	2	1.9	1.6–2.3	0.2	0.2–0.2

[a] Geometric mean.
[b] 1 egg contained 0.02 ppm β-BHC.
[c] 3 eggs contained 0.01–0.07 ppm mirex.

Eggshell thickness was negatively correlated to DDE levels in the eggs as can be judged from Table 1 above and the author's Table 3 below.

Table 3. Eggshell thickness and Ratcliffe index of random (1989) and addled (1985–88) osprey eggs, and eggshell fragments (1987–88), from 3 regions of New Jersey.

Region and shell type	n	Eggshell thickness (mm) x̄	Eggshell thickness (mm) SE	% below pre–1947 thickness[a] x̄	% below pre–1947 thickness[a] SE	Ratcliffe[b] index x̄	Ratcliffe[b] index SE
Delaware Bay							
Random	7	0.444	0.020	12.0	3.9	2.10	0.11
Addled	8	0.466	0.014	7.8	2.7	2.19	0.07
Fragment	2	0.430	0.005	14.9	1.0		
All types	17	0.453	0.011	10.4	2.1	2.15	0.06
Atlantic Coast							
Random	8	0.485	0.020	4.0	3.9	2.34	0.09
Addled	22	0.488	0.011	3.3	2.2	2.36	0.05
Fragment	19	0.472	0.011	6.5	2.1		
All types	49	0.482	0.007	4.7	1.4	2.36	0.05
Maurice River							
Random	2	0.490	0.045	3.0	8.9	2.43	0.16
Fragment	2	0.465	0.005	7.9	1.0		
All types	4	0.478	0.020	5.5	3.9	2.43	0.16

[a] Compared to data from Anderson and Hickey (1972).
[b] Our Ratcliffe Index values might not be comparable to pre-DDT values because methods of removing egg contents differed.

Apparently, eggs with shells thinned near to or at 15 % had a greater probability of breaking, contributing to the lower productivity observed in Delaware Bay compared to the other two locations as shown in the author's Table 2 below.

Table 2. Reproductive parameters of ospreys nesting in 3 regions of New Jersey, 1987–88.

Region	*n*	% eggs hatched	$\bar{x}$ young fledged/ pair	% nest success[a]
Delaware Bay[b]	24	50.0[c]	1.08	50.0
Atlantic Coast[b]	38	68.5	1.61	78.9
Maurice River	6	62.5	1.33	66.7

[a] Nests fledging ≥1 young.
[b] Data from Steidl et al. (1991).
[c] n = 12 nests.

Analysis of DDT residues in known prey fish revealed the following results.

Table 5. Organochlorine, lead, and mercury residues (ppm fresh wet mass) in fish collected from 3 regions of New Jersey, 1989.

Region and species	*n*[a]	DDE	DDD	Dieldrin	α-Chlordane	*trans*-Nonachlor	PCB's	Mercury	Lead	% moisture
Atlantic Coast										
Menhaden	5	0.05	0.04	nd[b]	0.02	0.01	0.28	0.03	0.29	62.8
Delaware Bay										
Menhaden[c]	5	0.17	0.12	0.04	0.08	0.03	0.46	0.04	0.30	66.2
White perch[d]	5	0.68	0.27	0.04	0.12	0.07	1.20	0.08	0.55	71.8
Channel catfish[e]	2	0.25	0.14	0.05	0.06	0.03	0.67	0.06	0.33	71.4
Maurice River										
White perch	6	0.05	0.03	0.01	0.01	0.01	0.18	0.20	0.24	72.2
Channel catfish	2	0.08	0.03	nd	0.02	0.01	0.34	0.24	0.10	76.0

[a] Number of fish in composite sample.
[b] None detected.
[c] Composite contained (ppm) 0.02 *p,p'*-DDT, 0.03 *o,p'*-DDE, 0.08 *o,p'*-DDD.
[d] Composite contained (ppm) 0.11 *o,p'*-DDE, 0.27 *o,p'*-DDD.
[e] Composite contained (ppm) 0.03 *o,p'*-DDE, 0.05 *o,p'*-DDD.

One should keep in mind that these fish samples were not scraps from the osprey nests but fish caught locally in the breeding grounds. Since ospreys often feed up the rivers from their breeding grounds, more contaminated fish may well have been consumed. Also, viscera were removed from whole fish. Viscera would contain liver, some adipose tissue and other organs that would be expected to have relatively high concentrations of DDT. Finally, these fish were caught in 1989 and the eggs were collected from 1985 to 1989. Some decline in fish residues from 1985 to 1989 would be expected, based on data from other locations. Even given all of the above, crude bioconcentration factors can be calculated as 5.7/0.54 = 5.7 for the Delaware Bay, 1.4/0.09 = 15.6 for the Atlantic coast and 1.9/0.095 = 20 for the Maurice River.

As the fish DDT levels decrease, the bioconcentration factor increases. This pattern will be even more evident as fish residues continue to decrease. One must keep in mind that as DDT residues continue to decrease in the United States, following the ban in 1972, exposure to DDT in wintering grounds in Latin America will account for an increasing proportion of egg residues. DDT use continued in Latin America after 1972 and is still in use in some locations today. These wintering ground exposures become more important as residues in fish in the U. S. continue to decrease. The multi-year half-life of DDT ensures that the highest exposures will be reflected in adipose concentrations that are passed directly into the yolk of the egg.

Other contributing factors to reproductive effects in ospreys in southern New Jersey include the presence of 4.1 to 26 ppm PCBs in the osprey eggs from Delaware Bay. The authors noted that the Delaware Bay is routinely dredged to maintain a shipping channel to ports on the Delaware River. They suggested that dredging exposed biota to old sediments containing higher residues of DDT and PCBs, resulting in a slower decline of residues and the persistence of effects no longer seen at other locations. Another factor is the travel time required to catch fish due to the lack of clarity of the water in the nesting areas that are in the more polluted parts of the Bay. Long travel times did not limit the food supply but did increase the time the nests were unattended, leading to potentially greater predation by great horned owls.

Considering the importance of the unknown exposure of ospreys to DDT in wintering grounds, digression to a 1982 article by Henny et al. is enlightening. This article reports the measurement of DDT in the blood of peregrine falcons captured during migration north in the spring and south in the fall. The peregrine falcon migration is similar to that of the osprey. Table 1 is most informative of the importance of the winter ground exposures in the late 1970s.

TABLE 1. DDE (geometric means, ppm wet weight) in blood plasma of Peregrine Falcons captured during migration at Assateague Island, Maryland/Virginia and Padre Island, Texas.

	Maryland/Virginia[a]			Texas			Maryland/Virginia			Texas		
Year	Mean	(95% C.I.)	n	Mean	(95% C.I.)	n	Mean	(95% C.I.)	n	Mean	(95% C.I.)	n
	HY♀♀						HY♂♂					
1976-77	0.11	(0.07–0.19)	15	0.05	(0.03–0.08)	15	0.08	(0.05–0.14)	9	0.16	—	2
1978	0.04	(0.02–0.08)	25	0.03	(0.02–0.07)	20	0.03	(0.01–0.10)	8	0.06	(0.03–0.09)	16
1979	0.07	(0.05–0.10)	36	0.05	(0.04–0.07)	74	0.08	(0.06–0.11)	26	0.05	(0.04–0.08)	22
Totals	0.06	(0.05–0.09)	76	0.05	(0.04–0.06)	109	0.07	(0.05–0.09)	43	0.06	(0.04–0.08)	40
	SY♀♀						ASY♀♀					
Fall												
1976-78	0.82	(0.44–1.53)	11	0.28	(0.01–6.75)	4		—		0.60	(0.27–1.33)	6
1979	0.64	(0.38–1.07)	6	0.27	(0.02–3.91)	3	0.71	(0.14–3.67)	5[b]	0.33	(0.14–0.77)	12
Totals	0.75	(0.50–1.13)	17	0.28	(0.07–1.16)	7	0.71	(0.14–3.67)	5	0.40	(0.22–0.72)	18
Spring												
1978-79		—		1.43	(0.52–3.87)	8		—		0.88	(0.60–1.29)	21
1980		—		0.42	(0.24–0.73)	19		—		0.62	(0.48–0.79)	63
Totals		—		0.60	(0.36–1.00)	27		—		0.67	(0.55–0.83)	84

[a]Excludes 3 HY♀♀ that were released along East Coast by Cornell University biologists.
[b]Includes one sample from 1978.

The table requires explanation. HY falcons are those migrating in the year they hatched. SY falcons are second year falcons and ASY means falcons migrating after their second year. Focusing on the Texas data for female falcons, one can see that just fledged falcons on their way south have quite low levels of DDE. SY falcons returning north in the spring of the next year have more than 10-times as much DDE in their plasma. Plasma levels are lower in SY falcons

migrating south from northern breeding areas. Apparently body burdens gained in the south during the winter are decreasing in the north during summer due to both egg laying and ever decreasing exposures in the northern breeding areas. The same pattern should apply to the osprey.

This exposure paradigm is even more important for the osprey since fledglings do not return to northern breeding grounds until their third year. Southern exposures to DDT would explain the ever increasing bioconcentration factors calculated from measures of just northern exposures. As DDT levels decreased in the United States to levels below those in Latin America, the importance of the unknown southern exposure eventually becomes essential to understanding the relationship between DDE residues in the fish diet and levels in eggs associated with thinning and hatching failure. With the understanding gained from this digression, let us resume reviewing the chronology of studies of the effects of DDE on osprey reproduction.

Audet et al. (1992) measured DDT residues in osprey eggs from three locations on the East Coast and compared them with residue levels in the early 1970s. The study was prompted by the finding of an isolated area in Chesapeake Bay with declining nestling survival. Median DDE levels in 1986 were 2.3 ppm in an area of declining fledgling survival (Martin Refuge), 0.65 ppm in coastal Virginia and 0.56 ppm in southern coastal Massachusetts. Relatively high ratios of DDT to DDE in the eggs from Massachusetts prompted the authors to suggest recent exposure to DDT from an unknown source (winter breeding grounds?). Eggs taken in 1972-73 from the same area of Massachusetts had DDE residues of 4.2 ppm. The authors concluded that the 0.65 and 0.56 ppm levels of DDE: "were well below reported values associated with biologically significant effects on eggshell thickness and reproductive success." In 1973, the Martin refuge had a median DDE level in eggs of 3.4 ppm with 17 % eggshell thinning, but nonetheless, 1.5 young per active nest. Productivity of 1.5 young per active nest was considered by these authors to be excellent. No reason was given or suggested for the declining fledgling survival at the Martin Refuge in 1986. Fledgling survival data was not reported.

Falkenberg et al. (1994) provided data on a nonmigratory population of osprey and their prey from the south coast of Australia. Six eggs collected in 1987 had an average total DDT residue of 0.22 ppm. Total DDT residues in 3 species of prey fish averaged 0.3 ppm giving a very low biomagnification factor of 0.73. Shells of osprey eggs collected in 1987-88 were no thinner than shells of eggs collected prior to the DDT era. The biomagnification of DDT into osprey eggs is so low in this study as to put into question the representativeness of the fish samples as a significant part of the diet eaten by osprey that produced the eggs collected in the study. The determination of a biomagnification factor is theoretically more certain in a nonmigrating population. Most likely, the biomagnification factor is small, based on studies in the 1960s and early 1970s in the U. S., probably less than ten.

In 1997, Ewins published an article about the behavior and history of osprey in North America. Figure 1 from Ewins illustrates the recovery of ospreys in Wisconsin and the Georgian Bay area of the Great Lakes Region.

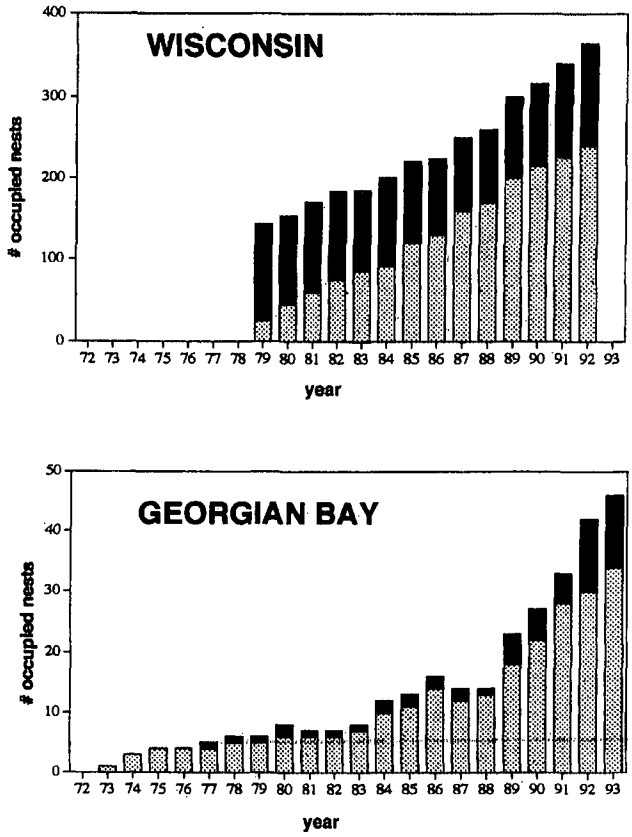

Figure 1. Changes in breeding populations of Ospreys since early 1970s in Wisconsin and Georgian Bay (Lakes Huron and Ontario), at artificial nest-platforms (stippled) and other (solid shading) sites. Most "other" sites were in trees. Wisconsin data are from Gieck et al. (1992).

Woodford et al. (1998) reported geometric mean DDE residues of 0.20 to 0.52 ppm in osprey eggs collected in 1992-93 from two breeding areas in central and northern Wisconsin.

Ewins et al. (1999) reported on eggs collected between 1980 and 1989 from two osprey breeding areas in central Michigan. The known age of each female osprey producing the eggs permitted a study of DDT residues in eggs produced by females from 3 to 15 years of age. No age related changes were found. The egg residues were independent of the age of the female. DDE averaged 1.2 ppm. Eggshell thickness increased from 1980 to 1989. Eggs collected from 1980 to 1984 were 5 % thinner and eggs collected from 1985 to 1989 were 3 % thinner than eggs collected prior to the DDT era. Eggs collected from the same areas in 1972-73 had geometric mean concentrations of 5.1 ppm DDE and 10 % average shell thinning. The decrease in DDE residues was associated with improved reproduction and population increases. Apparently female osprey in Michigan reached a steady-state DDE residue level in their tissues in the first 2-3 years of life (most of that time is spent on the wintering grounds in Latin America). Part of this ongoing steady-state is the elimination of accumulating adipose residues by laying eggs. The DDE residues in eggs from midwestern breeding grounds and some east coast locations

appear by the mid 1990s to be below levels associated with any significant effects on shell thickness or hatching success.

Elliott et al. (2000) reported on DDT residues in osprey eggs collected from the Columbia and Fraser River areas in the northwest. DDE residues were high and variable. Geometric means ranged from 1.0 to 13.8 ppm by area and year from 1991 to 1997. No trends by area or year were evident. Individual eggs ranged from 0.1 to 23.7 ppm DDE. DDE/DDT ratios were also highly variable. Some of the locations were in forested wilderness areas where little DDT had been used. Fish sampled in 1994 from these remote areas contained less than 0.005 ppm total DDT. The authors suggested that DDT was coming from an outside source, possibly from wintering grounds in southern Mexico. Another factor is the very high rate of DDT applications to apple orchards during the DDT use era (Blus et al., 1987). Twenty three per cent of the osprey eggs had DDE residues greater than 4.2 ppm, the level associated with eggshell thinning significant to hatching success.

Clark et al. (2001) published a followup study of the Steidl et al. (1991) Delaware Bay study summarized above. Comparisons between 1989 and 1998 at three locations in southern New Jersey were made in residue levels in eggs and fish, eggshell thinning, and productivity. DDE residues in osprey eggs had declined to 1.4 ppm with an associated eggshell thinning of 7 % in the more contaminated Delaware Bay area. "PCBs and DDE in osprey eggs were below levels considered to be toxic to egg development." Fish were collected in the same manner as in 1989. Total DDT residues in fish for the Delaware Bay averaged 0.23 ppm. Biomagnification factors from fish to eggs ranged from 9 to 11. Osprey productivity increased to 1.1 young per nest in the period from 1994 to 1998. Availability of nest structures and owl predation were thought to be limiting the population of ospreys in the Delaware Bay area.

In 2003, Martin et al., reported on ospreys in Great Lakes Canada. The study was conducted in 1991-95. DDE levels averaged 1.3-2.9 ppm in five study areas. A few eggs exceeded the 4.2 ppm (15 % eggshell thinning) threshold, suggesting that reproduction in a few individual ospreys was affected. The authors concluded, however, ."...ospreys now appear to be relatively unaffected by current low levels of chlorinated hydrocarbon contaminants."

Henny et al. (2003) reported on a detailed 1993 study of bioaccumulation of DDE from fish to osprey eggs in Oregon. The number of breeding pairs along the Willamette River increased from 13 in 1976 to 78 in 1993 and 234 in 2001. Overall productivity was 1.67 young per active nest. The geometric mean DDE residues in eggs was 2.3 ppm. Two of the ten eggs analyzed had levels of DDE that would be expected, based on other studies, to have reduced hatching success as a result of cracked shells.

The median level of DDE in the major food fish for ospreys, the largescale sucker, was found to be only 0.022 ppm. This very low fish residue resulted in a bioaccumulation factor for fish to osprey eggs of 87, prompting the authors to suggest that ospreys received significant exposures during winter migration to southern Mexico and Central America. This idea was reinforced by lower than expected bioaccumulation of PCBs and unexpectedly high levels of DDT in some eggs. However, others have reported much higher levels of DDE in largescale

suckers from the Willamette River. A single composite collected in 2000, contained 0.835 ppm (EPA, 2006). A bioaccumulation factor from this fish residue value would be 2.3/0.835 = 2.8.

In a chapter in Raptors Worldwide (2004), Henny et al., described a study of the effects of DDE residues on osprey eggshells and reproduction at nest sites along the Columbia River in northwestern United States. The number of ospreys had been increasing with each survey through 1998. Mean productivity was 1.64 young per active nest. Eggs were collected in 1997 and 1998. Table 5 from Henney et al. (2004) summarizes the findings on reproduction, eggshell thinning and DDE residues

Table 5. Number of young Ospreys produced per nest (with one egg collected) in relation to DDE concentrations in the sample egg collected, and eggshell thickness.

	Number of Nests with DDE ($\mu g\ kg^{-1}$)		
Number of Young	< 4200	4200-8000	> 8000
0	1	3	3
1	6	3	2
2	10	6	3
3	1	0	0
Active Nests	18	12	8
Successful Nests	17	9	5
Adv. Young	29	15	8
Young/Successful Nest	1.71	1.67	1.60
Young/Active Nest	1.61	1.25	1.00
Geo. Mean DDE ($\mu g\ kg^{-1}$)	2131	5473	10510
Mean Shell Thickness (mm)	0.488	0.441	0.419
Shell Thinning[a]	-3.4%	-12.7%	-17.0%

Note: One nest sampled did not have complete information for productivity (it was excluded), and 10 nests were included from the Willamette River in 1993 (Henny and Kaiser 1996).

[a] Compared to 0.505 mm for pre-DDT era eggshells from eastern U.S.A. (Anderson and Hickey 1972).

Dividing the nests into three classes by DDE egg residue level indicates a dose-response for thinning of eggshells and impairment of reproduction. Even at these high levels, with measurable impacts, the osprey population continues to grow. The geometric mean residue of DDE in eggs from nests along the Columbia River was 4.9 ppm, a value quite a bit higher than residues reported by the same authors for eggs collected in 1993 along the adjoining Willamette River. These residues are the highest reported nationwide for osprey eggs during the late 1980s and 1990s. Henney et al. suggest the possibility of exposure to DDT on the wintering grounds in southern Mexico and Central America. Another explanation could be the high application rates of DDT to apple orchards, creating pockets of high residues in soil and biota, including fish (Blus et al., 1987).

Fish residues were stated to be elevated but levels were not reported. Previous investigations from 1991-1993 were cited by the authors to have found an average of 0.089 ppm DDE in largescale suckers, an important food fish for the ospreys. Schmitt et al. (1990), had reported 1.0 ppm total DDT in largescale suckers from the Columbia River in 1984. A recent study (EPA, 2006) reported average total DDT residues of 0.450 ppm in largescale suckers

collected in 1996-1998 from the Columbia River Basin. Figure 2-4b from the report illustrates the high variability in the fish residues at different locations, explaining to some degree the high variability in DDE levels in osprey eggs.

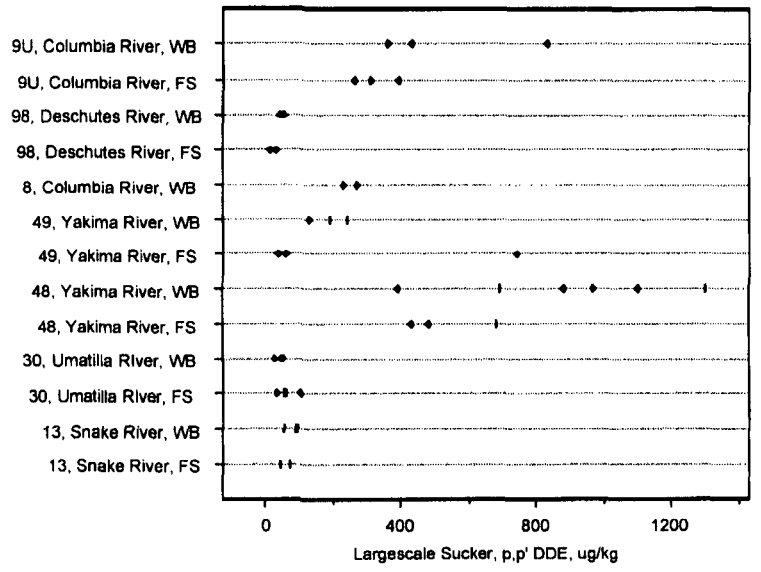

Figure 2-4b. Study site specific concentrations of p,p DDE in largescale sucker composite fish tissue samples from the Columbia River Basin.

A crude estimate of the biomagnification of DDE from fish to egg would be 4.9/(0.450-0.089) = 11-55.

Martell et al. (2001), used satellite telemetry to track the migration of osprey from northern breeding areas to southern wintering areas. Figure 2 from the publication shows that east coast ospreys winter primarily in Brazil, west coast ospreys winter primarily in southern Mexico and midwestern ospreys winter in both locations or in between.

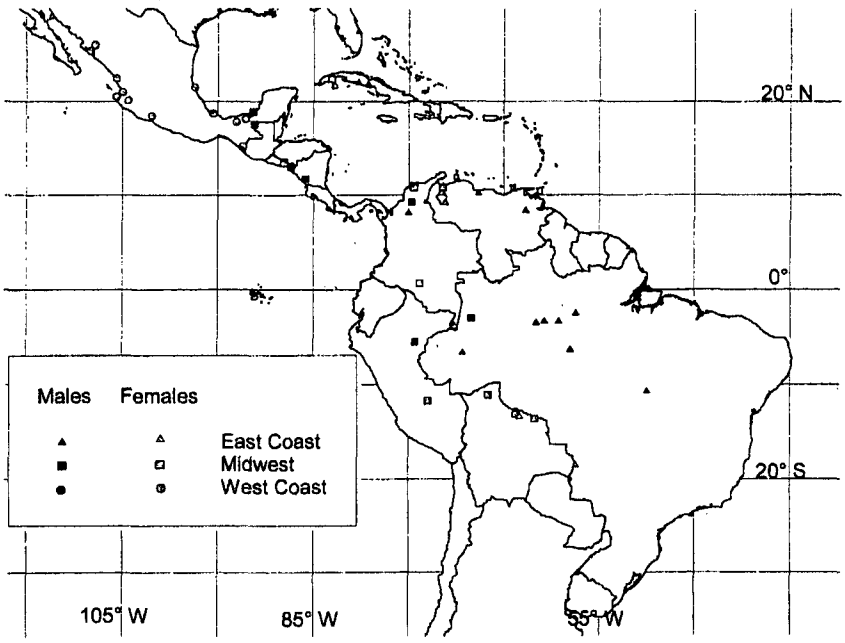

FIGURE 2. Wintering locations of North American Ospreys as determined by satellite telemetry.

Mora (1997) reviewed available information on reports of DDT contamination of migratory birds in Mexico. Contamination generally was found to be similar to that in southwestern United States through the 1980s. Mora summarized the use of DDT in Mexico through 1996.

Rattner et al. (2004), reported on contaminant exposure and reproductive success of ospreys in the Chesapeake Bay area. From a population estimated at 1,450 nesting pairs in 1973, the Chesapeake Bay osprey population more than doubled to an estimated 3,473 pairs by 1995-96. However, reproduction rates have not fully recovered in the more polluted waters of the Bay. Geometric means of DDE levels in eggs collected in 2000 from different parts of the Bay ranged from 0.4 to 1.2 ppm. Eggshell thinning ranged from 0 to 9 %. PCBs were as high as 19 ppm in eggs from nests in the more polluted areas. A limited sample of fish scraps from nests in some of the less polluted areas contained less than 0.050 ppm DDT homologues corresponding to 0.4 to 0.8 ppm total DDT in osprey eggs from those areas. A crude estimate of the bioaccumulation of total DDT from fish to eggs would be 0.4 to 0.8/<0.05 = >8 to >16. Marginal productivity in the more polluted areas was not linked to egg concentrations of DDE. DDE levels in osprey eggs from the Chesapeake Bay have decreased 10-fold from the DDT use era. "...concentrations of p,p′-DDE...in sample eggs did not cause direct and biologically significant toxic effects on osprey reproduction in Chesapeake Bay regions of concern."

A third study of the Delaware Bay was conducted in 2002 by Toschik et al. (2005). Geometric mean DDE levels in eggs from four parts of the Bay were 0.4-1.8 ppm. Eggshells from the northern part of the Bay were 10 % thinner. A few eggs from failed nests contained more than 4 ppm DDE. "All nestlings appeared in good health; no external lesions or other abnormalities were found." "Additionally, no evidence of chromosomal damage in nestlings was found." Based on only a few eggs, DDE in eggs from the Prime Hook National Refuge were 0.6 ppm in 2002 compared to 5 ppm in 1974. Marginal reproduction rates in the more polluted areas were the result of lost eggs. Lost eggs can be the result of damaged or cracked eggs tossed out by the parents, eggs lost from precarious nests (e.g, on floating buoys), human interference, or predation. Some of these factors are more prevalent in the more polluted areas because they are also the more urbanized and industrialized areas. The authors concluded that "...the latitudinal trends seen in egg contaminant exposure are unlikely to result from contaminant exposure on the wintering grounds." This idea is somewhat contradicted by the wide range of DDE levels in eggs from each area (overall range of 0.17 to 4.61 ppm). No fish residues were reported.

ANALYSIS

Considering the information that has been reviewed and summarized, can we determine a threshold for the action of DDE on reproduction in osprey? A lot is known. However, one is also aware of unknown exposures and high variability of residues and response. A wide range of endpoints and approaches can be taken.

For a given breeding area, a field study where no significant eggshell thinning was found, could be considered a threshold for the DDE residues in those eggs. The threshold for that finding is probably several hundred ppb DDE. A threshold for increased shell breakage and reduced hatchability is approximately 3-4 ppm DDE. Recovery and stabilization of DDE poisoned populations of ospreys has been associated with DDE egg residue levels as high as 5-8 ppm.

Although postulated, toxicity has not been shown for DDE residue levels in eggs that cause shell thinning up to 10 %. Thinking in evolutionary terms, normal eggshell thickness must have evolved to prevent breakage during incubation as well as provide gaseous exchange and an appropriate degree of hydration. There is a considerable range in normal eggshell thickness. Hatching success, as well as the health of the fledgling, does not appear to be compromised by minimal shell thinning. There is some uncertainty here, but the recovery, stability and health of populations still experiencing marginal shell thinning, suggests no detrimental effect. In addition to choosing a threshold for toxicity, one must also determine an appropriate biomagnification factor from fish to egg.

Osprey are opportunistic feeders, catching the most nutritous and easiest to catch species at any given location and time. Typical prey species vary with season, latitude and whether the location is coastal or inland. We should expect, therefore, some variation in the biomagnification from fish to eggs. The variation in literature values, however, appears more

related to a lack of representative sampling of fish from breeding grounds and a lack of data on residues in fish from wintering grounds.

The flounder, menhaden and largescale sucker appear to be the most important food species for osprey studied in North America. The largescale sucker is a fresh water species. Only the menhaden is among the species relied upon by the NAS panel in setting the marine fish recommendation to protect wildlife. For the 22 determinations of biomagnification from fish to egg determined from data in the reports above, there is considerable uncertainty. Therefore the best estimate from this data might be the median value of 10 (0.73-87, n=22). Values based on fish scraps cast from the nest range from 1.6 to 31 (n = 5) with a median of 10.9. For reasons explained previously, a value of 10 is most likely to be high. For example the two values from nonmigratory populations were 0.73 and 3.8.

A recommendation for DDT residues in marine fish should not consider DDD, because DDD has not been shown to cause shell thinning and is not converted to DDE. DDE causes eggshell thinning and DDT can be converted to DDE. DDT and DDE are the important residues.

If the recommendation is to protect the osprey as a sensitive representative for other fish-eating species, as implied in the NAS recommendation, then one needs to select a threshold level in eggs and divide by an appropriate biomagnification factor. If one were to use a threshold that is half of the approximate lower end of the hatchability effect threshold and divide by a biomagnification factor of 10, the recommendation would be 150 ppb in fish. This level is 3 times what the NAS panel recommended, but is based on additional information that they overlooked or wasn't known until after 1972. The value of 150 ppb is the same as reported in the diet of a recovering population of brown pelicans by Anderson et al. (Risebrough was a coauthor of this report) in 1975. The 150 ppb residue in fish in the 1975 report became, in 1980, the basis for the current National guidance and later the State standard (CTR standard) for DDT in the water column. Both recommendations would then be the same, as they should be, since both are based on protection of eggshell thinning in similarly sensitive species.

The SARWQCB insistence upon using the NAS 50 ppb guidance in fish ignores the oversight of existing information in 1972 and the subsequent 34 years of research on the recovery of sensitive avian species from the reproductive effects of DDE. The SARWQCB position is even less tenable in that they chose to also use the 1,000 ppb guidance for fresh water fish, when the two guidance numbers are based on essentially the same data. The SARWQCB appears to be stuck on published numbers rather than trying to understand and apply the science behind the numbers. The guidance specifically states that local conditions are to be considered. The very recent successful fledging of three chicks by a nesting pair of ospreys in the Watershed is significant.

CONCLUSIONS

The following points can be made concerning the NAS recommendation for DDT residues in marine fish to protect wildlife and what is currently known about the effects of DDT on reproduction in ospreys, the species that is the basis for the NAS recommendation.

- The NAS panel did not consider important available information concerning the effects of DDT on reproduction in ospreys that may well have caused them to recommend a residue higher than 50 ppb.

- A review of the effects of DDT on reproduction in ospreys documents the nationwide recovery of breeding populations as residues have declined in fish and eggs.

- As residues and effects declined in northern breeding grounds, continued use of DDT in wintering grounds in Latin America became more important in limiting egg residue decline

- High uncertainty in estimating biomagnification of DDT from fish to eggs has resulted from hot spots of contamination in northern breeding grounds and unknown contributions from wintering grounds in Latin America.

- Effect thresholds for DDE residues in eggs appear to be several hundred ppb for eggshell thinning, 3-4 ppm for hatching success, and 5-8 ppm for population stability.

- A recommendation for marine fish of 150 ppb DDT is made from half the low end of the threshold for hatching success divided by a biomagnification factor of 10. The 150 ppb recommendation is the same as that for the brown pelican study used as the basis for the National criterion and State CTR standard for DDT in the water column.

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Appendix D: Dr. James L. Byard, "Scientific Commentary on the Effects of DDT on Reproduction in Cormorants and Terns"

SCIENTIFIC COMMENTARY ON THE EFFECTS OF DDT ON REPRODUCTION IN CORMORANTS AND TERNS

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SUMMARY

Cormorants and terns are less sensitive to the reproductive effects of DDTs than ospreys and brown pelicans. Residues of DDE in excess of 10 ppm, resulting in eggshell thinning of 15 % or greater, are necessary to produce significant hatching failure. A Department of the Interior publication on toxicity thresholds for DDTs in avian species was found to contain errors and serious misrepresentations of published scientific studies.

INTRODUCTION

DDT was introduced in 1947 and cancelled in 1972. During and for some time after this period, reproduction was inhibited in many avian species. DDE, a stable metabolite of DDT, bioaccumulated up food chains to reach toxic levels in the shell gland. Calcium deposition was inhibited, resulting in thinner shells. At a critical thinning of around 15 % or higher, egg shells cracked more easily during incubation, resulting in hatching failure. No other low-level chronic effects have been widely acknowledged by researchers studing the effects of DDT on wildlife.

More than 500 research articles have been published on the reproductive effects of DDT in avian species. The wide variation in species sensitivity has been well delineated. The most sensitive species appear to be those who eat fish or other birds. Among these species, the most sensitive are those in which DDE is most potent in blocking calcification in the shell gland. These include the brown pelican, osprey, white-faced ibis and peregrine falcon as the most sensitive species. Many other species are nearly as sensitive. Raptors feeding on rodents and insects appear to be less sensitive.

A detailed report of the effects of DDT on shell thinning and hatching success in the brown pelican, osprey, petrels, and sparrow hawk can be found in other chapters where DDT effects in these species played a central role in establishing guidance levels in fish and water. This report will review the effects of DDT on reproduction in cormorants and terns. These species are not the most sensitive. However, they do reside in Newport Bay and Watershed and they are to be used as receptors for a sediment to wildlife modeling study about to be reported by Ben Greenfield at the San Francisco Estuary Institute (SFEI). The review will be in chronological order and will begin with cormorants.

CORMORANTS

Eleven cormorant and five white pelican colonies were studied by Anderson et al. (1969) in the upper midwest and central Canadian provinces in 1965. DDE residues were as high as 45 ppm in cormorant eggs and 4.8 ppm in white pelican eggs with averages of 10.4 and 1.7 ppm, respectively. Egg size, weight and thickness varied between the locations. Egg laying is a mechanism for excretion of DDT. Egg residues are more closely related to residues stored in lipid than recent dietary intake. Eggshell thickness was decreased 4.5 % in white pelicans and 8.3 % in cormorants. Increases in shell thickness during rebreeding suggests that low levels of DDT in local diets was more important than reductions in DDT by utilization of lipid stores during breeding. One population of cormorants, with a 25 % decline in eggshell thickness, had recently decreased to nearly zero. At the same location, a reasonably stationary population of great blue herons persists. The authors claim that the eggshell thinning-DDE regression is linear to zero concentration of DDE. A minimal effect level could not be established. Figure 3, reproduced below from Anderson et al. (1969), illustrates the eggshell thinning dose-response in cormorants.

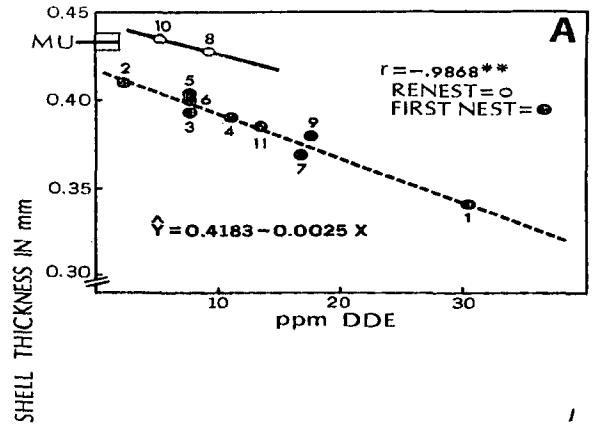

FIGURE 3. Relationships between DDE residues (A), estimated PCB residues (B), and shell thickness in Double-crested Cormorants, plotted on a colony-basis. Individual points are numbered in accordance with Figure 1. Open circles represent renest colonies (the original colony of the season was destroyed or disturbed away from the first-nest site, therefore, phenologically behind other colonies from the same general latitude and longitude) and closed circles represent first-nest colonies. "MU" in the upper figure represents the museum mean thickness (Table 5), bounded by 95% Confidence Limits. Figure 3A, $P<0.001$; Figure 3B, $P<0.01$. The line-of-fit for renests in A, was fitted by eye but clearly resembled the calculated regression based on individual pools. A line-of-fit for renests in B., though significant on an individual-pool basis, was not obvious on a colony basis.

Faber and Hickey (1973) reported on a 1969-1970 survey of egg residues and eggshell thinning in fish-eating birds from the upper Great Lakes states and Louisiana. The results are summarized in Figure 1 below. The authors suggest that significant decreases in shell thickness will be found in virtually all fish-eating birds in these parts of America. "We are uncertain about the biological significance of decreases in shell thickness below 10 %. Certainly, widespread eggshell breakage does not occur with changes below this magnitude." The level of DDE residue necessary to cause eggshell thinning varies greatly among species. This point is illustrated in their Figure 1, reproduced below.

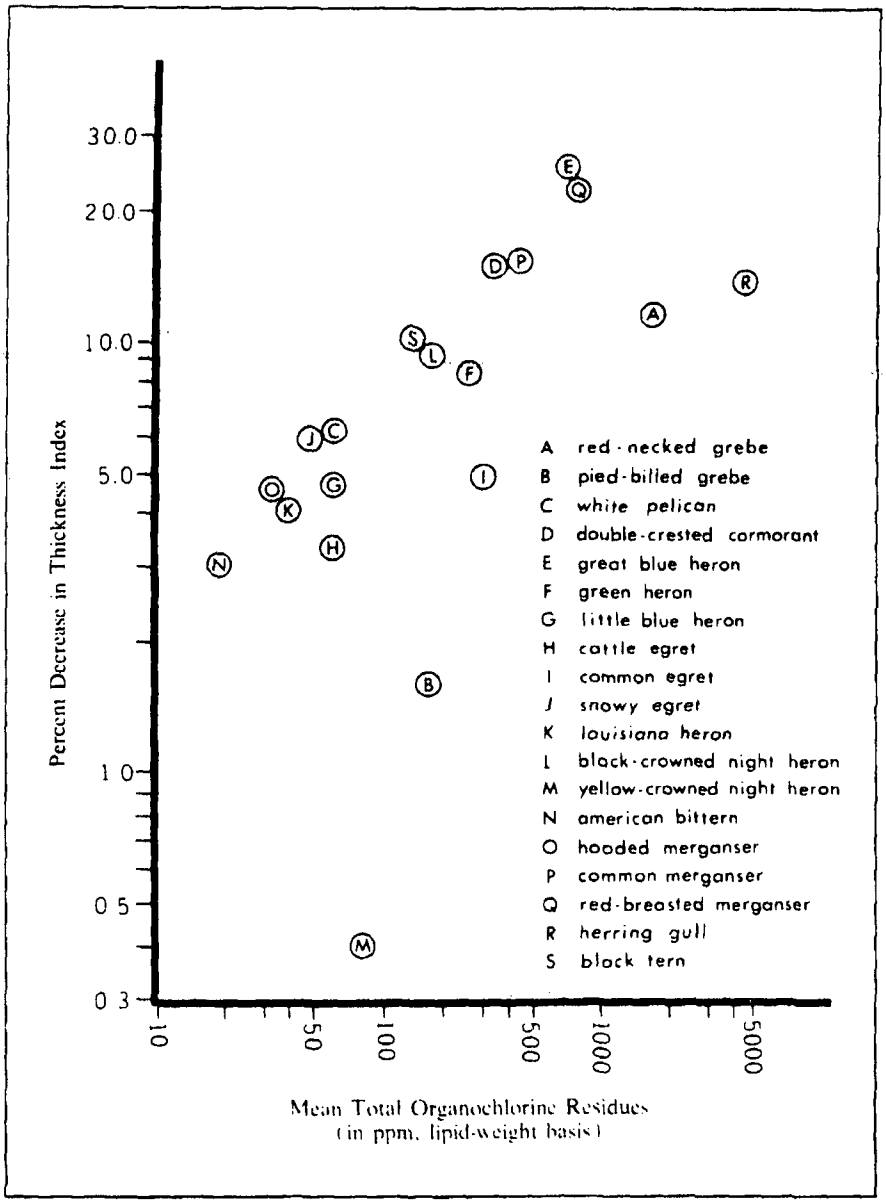

FIGURE 1.—*Mean shell-thickness index changes and mean residue levels for each species (log-log basis)*

Gress et al. (1973) reported on a survey of double-crested cormorant breeding colonies in the Channel Islands and the islands off of the west coast of Baja, California in 1969-1972. Breeding was almost nonexistent in colonies on the Channel Islands and South Los Coronados Island. Breeding appeared unaffected on San Martin Island further south. No crushed eggs were found on San Martin. Eggshell thinning was 29 and 38 % on Anacapa and Los Coronados, respectively. "The San Martin eggshells show no significant differences of any of the

parameters from the museum specimens." DDE residues in eggs were 32, 24 and 1.7 ppm on Anacapa, Los Coronados and San Martin, respectively. Other studies on double-crested cormorants did not find reproductive impairment with DDE residues as high as 10.4 ppm DDE associated with 8.3 % eggshell thinning. "The comparatively low levels of DDE reported suggest that the degree of thinning, if present, would not be sufficiently great to affect reproductive success." Comparisons with studies of interior populations indicated that the relationship between DDE residues and eggshell thinning were the same. In addition, 80 % of the variation in eggshell thickness could be explained by the regression on the natural log of DDE. The 1972 survey suggested that both the brown pelican and double-crested cormorant were beginning to recover. The recovery was attributed to the fact that the DDT manufacturing plant in Los Angeles stopped discharging wastes to the Los Angeles outfall in April, 1970.

Morrison et al. (1978) reported on DDE residues and shell thickness in cormorant eggs collected in Texas in 1976-1977. The results were compared with an earlier study by King (1977) in which cormorant eggs were collected in 1970. The results of the King study were provided by personal communication to the authors from K. A. King. DDE residues had declined dramatically from the 1970 to 1976-1977 eggs as shown in Table 1 below reproduced from Morrison et al. (1978).

TABLE 1

RESIDUES IN OLIVACEOUS CORMORANT EGGS IN TEXAS[1]

	1970 (n = 5)			1976–77 (n = 7)			
Residue	$\bar{x}$	S.E.	(%)	$\bar{x}$	S.E.	(%)	% Change
p, p′-DDE	6.22	2.08	100	0.400	0.036	100*	-93.6
Dieldrin	0.30	—	20[4]	0.018	0.003	100	-94.0
PCB[2]	32.00	5.83	100	1.890	0.275	100**	-94.1
Heptachlor Epoxide[3]				0.032	0.016	100	—

[1] Values represent residues on a wet-weight basis.
[2] Arochlor 1254 and 1260.
[3] This residue was separated from PCBs in 1977 eggs only.
[4] Dieldrin found in detectable levels in only 1 egg in 1970 (1970 $\bar{x}$ for dieldrin, all eggs = 0.06 ± 0.134; −70%; $p > 0.05$).
* $p < 0.05$, ** $p < 0.01$, t-test.

Eggshell thickness was not significantly affected in either the 1970 or 1976-1977 studies, although the latter shells were thicker as shown below in Table 2 below from the same publication.

TABLE 2

SHELL THICKNESS OF OLIVACEOUS CORMORANT EGGS IN TEXAS (MM)

Date	n (eggs)	$\bar{x}$	S.E.	% Change from Pre-1940	% Change from 1970
Pre-1940	75	0.328	0.004	–	–
1970	24	0.323	0.006	−1.5	–
1976–77	21	0.341	0.004	+4.0*	+5.5*

* $p < 0.05$, t-test.

The authors concluded that there was little difference in thickness between the pre-DDT era shells, the 1970 shells and the 1976-1977 shells. "Most authors agree that a 10-20 % change in shell thickness is needed before reproductive failures are indicated." and "Cormorant eggshell thickness was apparently not affected by residues in the 1970's in Texas."

Pearce et al. (1979) reported DDE residues in cormorant eggs collected along eastern Canadian coastal waters from 1970 to 1976. Average residues by site ranged from 1.49 to 8.57 ppm. Individual eggs ranged from 0.16 to 20 ppm DDE. The authors report measuring shell thickness, but no data were reported. The authors claim that 10 ppm DDE in eggs produces 20 % shell thinning. This conclusion was based on an extrapolation of the residue - shell thinning data. Again, no data or regression plots were reported in the article.

Weseloh et al. (1983) reported on the status of double-crested cormorant colonies in Lake Huron. Six colonies were studied in 1972 and 1973. DDE residues in eggs averaged 14.5 ppm. Eggshell thickness was reduced an average of 23.9 %. Egg breakage, hatching failure, and population declines were evident.

Fossi et al. (1984) reported high levels of DDE in cormorant eggs collected from the Danube Delta. DDE levels in 13 eggs averaged 9 ppm. Eggshell thickness was not measured. The authors noted that: "Despite the heavy contamination of the eggs, however, the population of the colonies of Common Cormorant seem to have stabilized..."

King and Krynitsky (1986) studied cormorants nesting in Galveston Bay from 1980 to 1982. DDE levels in eggs averaged 1.73 ppm in 1980 and 0.67 ppm in 1981. Mean shell thickness for the period 1980 to 1982 was similar to eggs collected prior to the DDT era. Eggs collected from Galveston Bay in 1970 (King et al., 1978) were 7 % thinner; eggs collected in 1980 were 5 % thinner; eggs collected in 1981 were 3 % thinner; eggs collected in 1982 were 1 % thicker. The 3 % and 1 % effects were not statistically significant. One egg collected in 1980 was 22 % thinner than pre DDT era eggs. Although not indicated by the authors, this egg may have contained the highest residue measured in the 1980 eggs (N = 13). That level was 31 ppm DDE. The authors noted that cormorant populations had remained stable in recent years.

Dirksen et al. (1995) reported a detailed study of organochlorines in cormorants in the Netherlands. Reproductive effects of DDE were confounded by high levels of PCBs in adult tissue and eggs. However, the authors concluded that 4 ppm DDE in cormorant eggs produced 5 % shell thinning. They also noted that the threshold for population reproductive failure and population instability was associated with shell thinning of 20 %. This level of thinning was associated with egg residues of 10 ppm.

In 1998, the Department of the Interior published a National Irrigation Water Quality Program Information Report No. 3 titled: Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment. DDT. The U. S. Fish and Wildlife Service participated and presumbably wrote the section on toxicity to avian species. According to the report: "Toxic effect levels for various types of birds are presented in table 16." Beginning on page 70, Table 16 lists various avian species, the DDTs studied, the concentration in eggs, the effects observed, and the reference. For the double-crested cormorant, a concentration of 10 ppm of DDE in eggs is stated to cause 20 % shell thinning. The reference for this data point is the Pearce, et al (1979) article discussed above. This study claims to have measured shell thinning and to have correlated the shell thinning with DDE residues. However, no shell thinning data and regression plots are to be found in the publication. Hence, this data point in the Department of Interior study is based only on a statement without data or analysis. Comparison of other data points in Table 16 with the referenced article revealed even more troubling findings.

For example, Table 16 lists 1 ppm DDE in Western grebe eggs as causing 1 % shell thinning. The DDE concentration reported in the cited study was 1.4 ppm not 1 ppm (Boellstorff et al., 1985). The 1 % was reported by Boellstorff et al. (1985) to not be statistically significant. The authors concluded: "Thickness of grebe eggshells collected at Tule Lake NWR in 1972 and 1981 and in northern California from 1952-1960 were not significantly different from each other and were not thinner than eggs collected before 1947 (Table 4)."

The very next line in Table 16 states that 5.4 ppm DDE caused 2.3 % eggshell thinning and reduced productivity. The research article cited for this data point (Lindvall and Low, 1980) reports a DDE residue of 6.6 ppm and a thinning of 3.1 %. The authors did not conclude that productivity was reduced. To the contrary, the authors concluded: "The small amount of eggshell thinning seen in western grebe eggshells at Bear River MBR appeared to have little or no effect on reproduction, because no crushed, cracked, or broken eggs were seen during this study. Average brood sizes of 1.6 in 1973 and 1.8 in 1974 from Bear River compare well with the Rudd and Herman determination of a normally reproducing population (18)."

The Department of the Interior report also states in Table 16 that less than 1 ppm DDE produced 6.5 % shell thinning in black-crowned night-herons. The reference for this data point (Findholt and Trost, 1985) reported a linear regression of shell thickness and log DDE egg residue that had a zero residue intercept of 0.26 mm. Since pre DDT era shells in this study were 0.275 mm, the linear regression is likely to be inaccurate, particularly at low residue levels. A similar phenomenon has been reported in brown pelican studies. The obvious fallacy in the

Table 16 listing is made clear by the fact that eggs containing 1.01 to 4.0 ppm DDE had thicker shells than eggs with less than 1 ppm DDE.

Table 16 states that 0.52 ppm DDE in common goldeneye eggs causes 15.4 % shell thinning and egg breakage. The 15.4 % shell thinning is a comparison of 1981 Minnesota colonies with North Dakota and Manitoba eggs collected in 1896 and 1903. The authors (Zicus et al., 1988) conclusion on egg breakage is as follows: "The high rate of egg breakage observed for Common Goldeneyes may be related to eggshell thinning or may be characteristic of the species and perhaps a result of frequent nest parasitism."

Finally, Table 16 states that 12 ppm DDE in Leach's storm petrel eggs results in 12 % eggshell thinning. The cited reference (Noble and Elliot, 1990) reports only on raptors and makes no mention of Leach's storm petrel. The Department of Interior report repeatedly makes errors and misrepresentations of the literature findings on the effects of DDT on avian reproduction. At best the report is done incompetently and at worst is an intentional misrepresentation to achieve a higher potency for DDT in avian species than is supported by scientific study.

Custer et al. (1999) reported on cormorant colonies on Cat Island in Green Bay, Wisconsin. Eggs contained 3.9 ppm DDE and 13.6 ppm PCBs. DDE concentration correlated with decreased shell thickness and hatching failure (thinning data were not reported). However, the authors concluded that reproductive performance was generally good to excellent compared to other locations, including those considered to have low levels of persistent organochlorine contamination. "Number of young produced (2.0-2.3 to 12 d of age) was also similar or greater than the 0.7 to 2.5 young per nest reported in relatively uncontaminated colonies."..."DDE-contamination does not seem to be a significant risk factor to double-crested cormorant populations in this region." A low level of chick deformities was not attributed to DDE.

TERNS

Vermeer and Reynolds (1970 reported DDE levels in eggs of common terns collected in 1968 and 1969 in central Canada. DDE residue levels ranged from 2.04 to 25.2 ppm. The authors noted the importance of wintering ground exposures.

Switzer et al. (1971) studied common terns in Alberta, Canada in 1969. Reproduction was poor. DDE levels in eggs were so variable that a correlation with shell thinning could not be established. DDE residues in eggs averaged 7.57 ppm. DDE residues in resident fish were below 0.03 ppm. The authors concluded that exposure to DDE occurred primarily at wintering grounds. The authors (Switzer et al., 1973) continued to study the breeding colony of common terns in 1970. In 1970, reproduction had improved. DDE residues had fallen to 4.52 ppm. DDE residues were concluded to be due to exposures at wintering grounds located from Southern California to Peru. This second, more careful study resulted in a correlation between DDE residue and shell thinning, although the authors did not attribute reproductive failure to DDE.

Fox (1976) reported on reproductive studies in common terns in Alberta in 1972. Reproduction was not sufficient to maintain the population. Eggshell thickness decreased 3.8 % in all eggs and 13.5 % in dented eggs collected from the colony. Detailed chemical and morphological studies of the eggs were described. Average DDE residues were 3.98 ppm. The fish diet contained only 0.02 ppm DDE. Fox concluded that most of DDE exposure occurred at wintering grounds. He concluded that DDE and other organochlorines were causing a variety of effects on eggs and the embryo at exposures below those known to cause shell thinning and reduced hatching success.

Pearce et al. (1979) concluded that DDE egg residues of 0.49 to 1.11 ppm were not affecting reproduction in common terns in five colonies in eastern Canada.

Nisbet (1982) measured DDE residues of 0.59 to 0.66 ppm in common tern eggs from Massachusetts in 1973. The focus of the study was on differences in residue level and shell thickness in the order eggs were laid. No conclusions were reached as to the significance of DDE residues to reproductive success.

Ohlendorf et al. (1985) reported on a 1981 study of Caspian and elegant turns breeding colonies in the south end of San Diego Bay. The Caspian terns were experiencing eggshell thinning, reduced hatching success and residues of DDE averaging 9.30 ppm in eggs. Elegant terns had DDE residues averaging 3.79 ppm in eggs and were experiencing comparatively successful breeding. Three Forster's tern eggs, in which chicks died during hatching, had residues averaging 3.72 ppm DDE. The difference in DDE exposure was attributed to a difference in foraging areas for the two species of terns. Caspian terns tended to forage in the salt marshes of the lower Tijuana River. Fish brought back to nests by caspian terns contained DDE residues as high as 3.0 ppm. Topsmelt was observed to be the major food prey species for Caspian terns and least terns. Other prey species containing relatively high levels of DDE included California halibut and black surfperch. Elegant terns foraged offshore in La Jolla Cove or near Isla Los Coronados. A major food prey species for elegant terns was the northern anchovy.

The range of DDE residue levels in Caspian tern eggs was 2.1 to 56 ppm (Ohlendorf et al., 1985). Eggs that appeared normal were no thinner than pre-DDT era eggshells. Broken eggs or eggs containing chicks that died during hatching averaged 14.4 % thinner shells than pre-DDT era eggshells. The high variability in residue level and reproductive effect was apparently related to the level of contamination in foraging areas and choice of prey species. Studies in common terns are cited that suggest that eggshell thinning and reproductive effects are seen when egg DDE residues exceed 4 ppm.

Ohlendorf et al. (1985) noted that the elegant tern colony had high hatching success in 1980 and 1981 with low incidence of embryo mortality or chicks dying in hatching. "Of the chicks that hatched, more than 97 % survived to fledging."

In 1982, Ohlendorf et al. (1988) studied tern populations in San Francisco Bay and Elkhorn Slough along the California Pacific coast. Geometric mean concentrations of DDE in were 6.93 ppm in Caspian tern eggs and 1.92 ppm in Forster's tern eggs collected in San Francisco Bay. DDE averaged 7.64 ppm in Caspian tern eggs from Elkhorn Slough. Differences in egg residues were attributed to differences in foraging areas and wintering grounds. The authors cite a midwestern study that found good reproduction success in Caspian terns with DDE residues similar to those reported for the two California populations.

King et al. (1991) reported a 1984 study in Forster's and Caspian tern populations on the Gulf coast of Texas. DDE residues in Forster's tern eggs averaged 0.8 and 1.6 ppm in two different populations. Eggshell thinning was 7 %, thinning below that associated with lowered reproduction. The authors stated that: "While 5 to 7 % shell thinning is statistically significant, it is probably not biologically significant. Numerous field studies have shown that average eggshell thinning of less than 10% is seldom associated with egg breakage and population decline (Anderson *et al.* 1969; Blus 1970, 1982; King *et al.* 1980)." Caspian tern eggs had average DDE residue levels of 2.2 ppm. Caspian and least tern eggshells were no different in thickness than pre-DDT era eggshells.

Hoffman et al. (1993) reported DDE residues of 1.7 to 2.9 in eggs collected in 1985 from several populations of common terns in the Great Lakes area. Embryotoxicity observed in the study was attributed to PCBs and dioxins and not to DDE. "Other examined contaminants, including DDE, other organochlorine pesticides, and mercury, were not directly related to these effects."

DDE residue levels were reported in eggs of California least terns collected from 1981 to 1987 from colonies in San Francisco and San Diego Bays (Hothem and Zador, 1995). The authors noted that: "California least terns are primarily piscivorous during the nesting period (Massey 1974), feeding predominantly on jack-smelt (Atherinops californiensis), topsmelt (A. affinis), and northern anchovy (Engraulis mordax) (Atwood and Minsky 1983)." and "...California least terns forage mostly within 3.2 km of their nest sites during the incubation and chick-feeding stages (Atwood and Minsky 1983; Massey et al. 1992)." Table 1 below, reproduced from Hothem and Zador (1995), summarizes DDE residues in eggs collected from the two bays.

Table 1. Geometric mean concentrations of mercury (Hg) and selenium (Se) (μg/g dry wt) and organochlorines (μg/g fresh wet wt) in eggs of least terns from San Francisco Bay and San Diego Bay, California, 1981-1987.

	San Francisco Bay			San Diego Bay		
Contaminant	N[1]	Mean	Min/Max	N	Mean	Min/Max
Hg	11	1.88	1.3-3.2	15	1.07	0.56-2.8
Se	12	2.67	2.5-3.1	17	2.41	1.6-2.9
Oxychlordane	13	0.013	ND[2]-0.039	18	0.014	ND-0.092
trans-Nonachlor	13	0.148	0.094-0.32	18	0.097	0.031-0.21
Total PCBs	13	3.66	2.1-5.2	18	1.22	0.71-3.1
p,p'-DDE	13	1.02	0.55-1.9	18	0.936	0.031-1.7
Dieldrin	13	0.095	0.053-0.19	18	0.011	ND-0.038
cis-Nonachlor	13	0.022	ND-0.17	18	NC[3]	ND-0.10

[1]N = sample size; [2]ND = not detected, below the LOD; [3]NC = not calculated, <50% of samples with detected analyte.

The authors noted: "Blus and Prouty (1979) found concentrations in least terns (0.19-1.22 ug/g) from South Carolina that were not thought to pose any threat to reproduction. Similar values have also been reported not to adversely affect reproductive success in common and Forster's terns (Custer et al. 1983; King et al. 1991)."

Hothem and Powell (2000) reported DDE residues in 72 California least tern eggs collected in 1994 along the southern California coast. DDE concentrations ranged from 0.230 to 0.562 ppm from three sites in and around San Diego Bay. The authors concluded that: "Likewise, DDE should not pose a threat to either species in our study."

CONCLUSIONS

- Terns and cormorants are less sensitive to the reproductive effects of DDTs than ospreys and brown pelicans
- Residues of DDE in eggs in excess of 10 ppm, resulting in eggshell thinning of 15 % or greater, are necessary to produce significant hatching failure.
- A Department of the Interior publication on toxicity thresholds for DDTs in avian species was found to contain errors and serious misrepresentations of published scientific studies.

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Appendix E: Dr. Ronald S. Tjeerdema, "Review of the History of DDT in Marine Mammals of Potential Importance to Newport Bay, CA"

REVIEW OF THE HISTORY OF DDT IN MARINE MAMMALS OF POTENTIAL IMPORTANCE TO NEWPORT BAY, CA

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SUMMARY

Concentrations of DDT and/or its degradation products have been reported in various marine mammals since the mid-1960s, indicating their ability to accumulate the highly fat-soluble compounds. Via biomagnification, blubber concentrations in fish-eating harbor seals, California sea lions, and Pacific bottlenose and common dolphins have typically been in the parts-per-million range. Since they filter feed at lower levels of the marine food web, blubber levels in baleen whales such as gray and minke whales have tended to be in the parts-per-billion range. In general, ΣDDT concentrations in all species of importance to Newport Bay, California, are declining, which reflects currently decreasing environmental concentrations worldwide as well as in the region. Due to strictly limited access to relevant marine mammals, there were virtually no published reports encountered via electronic search describing the toxic actions of DDT and/or its degradation products deduced from controlled potency or mechanistic studies utilizing statistically-relevant population sizes. With continued access limitations and housing and handling difficulties, the potential toxic effects of DDT and/or its residues are not likely to be delineated in the near future. Since shellfish tissue concentrations are on the decline in Newport Bay, and since marine mammal visitation to the region is limited and transitory, it is unlikely that sufficient concentrations will be accumulated by marine mammals in the region to cause toxic consequences.

INTRODUCTION

In April, 2006, a comprehensive review of relevant scientific literature was undertaken to assess what is currently known regarding the effects of DDT in marine mammals either resident to, or capable of visiting, Newport Bay, California. The first step was to determine the species that should be included. As documented in Appendix I, while there are numerous marine mammal species found in the northwestern Pacific Ocean, relatively few species reside in, or visit, Newport Bay. Those that may potentially reside in the area for significant periods include the California sea lion (*Zalophus californianus*) and harbor seal (*Phoca vitulina*). Those species that may enter Newport Bay for at least short periods – an unlikely but conservative approach – include the Pacific bottlenose dolphin (*Tursiops gilli*), rough-toothed dolphin (*Steno bredanensis*) and common dolphin

(*Delphinus delphis*), and two filter-feeding baleen whale species – the minke whale (*Balaenoptera acutorostrata*) and the migratory gray whale (*Eschrichtius gibbosus*; Ingles, 1965; Burt, 1975).

Therefore, electronic database searches were conducted via both the ISI Web of Science and BIOSIS Previews using the following topical keywords:

Seals and DDT
Sea Lions and DDT
Dolphins and DDT
Whales and DDT

Several hundred documents dating from the mid-1960s through 2006 were identified, but most involved species not relevant to the Newport Bay region (i.e. not listed above). However, a significant number of reports were identified and are summarized below. While no search can necessarily identify and locate all publications on a topic, those summarized below provide a reasonable summary of what is currently known regarding DDT in marine mammals that may either reside in or visit Newport Bay.

One important factor to consider in this review is the virtual absence of publications that describe the toxic effects or endpoints of DDT in the subject marine mammals. There are two key reasons for this. First, logistically specimens of these sorts of marine mammals are very difficult to directly utilize in the statistically-significant numbers needed for valid potency or other mechanistic investigations. While sea otters may only weigh a few pounds, whales are excessively large and not practical to handle or house. Second, marine mammals have been protected by the United States Government for many years, which has significantly reduced access for any purpose, including research. Therefore, nearly all the papers published to date involve the measurement of DDT residues in tissues obtained from either live or dead (stranded and often decaying) animals. Such information can at least give an approximate estimate of the residues encountered by the subject marine mammals – and their ability to accumulate them. Therefore, below is a brief summary of the published reports involving DDT in marine mammals of importance to Newport Bay.

CHRONOLOGY OF MARINE MAMMAL STUDIES

The DDT concentrations below are reported as ΣDDT (sum DDT), which typically represents the sum of either three (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE) or six (*p,p'*-DDT + *p,p'*-DDD + *p,p'*-DDE + *o,p'*-DDT + *o,p'*-DDD + *o,p'*-DDE) congeners. When the sum is reported, it will be defined to avoid confusion. Also, unless otherwise indicated, all residue values reported below are based on wet sample weight – concentrations reported on a lipid weight basis can average four or more times higher than those reported on a wet weight basis. Note that while many reported values are geometric means (delineated below), some are arithmetic means.