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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.

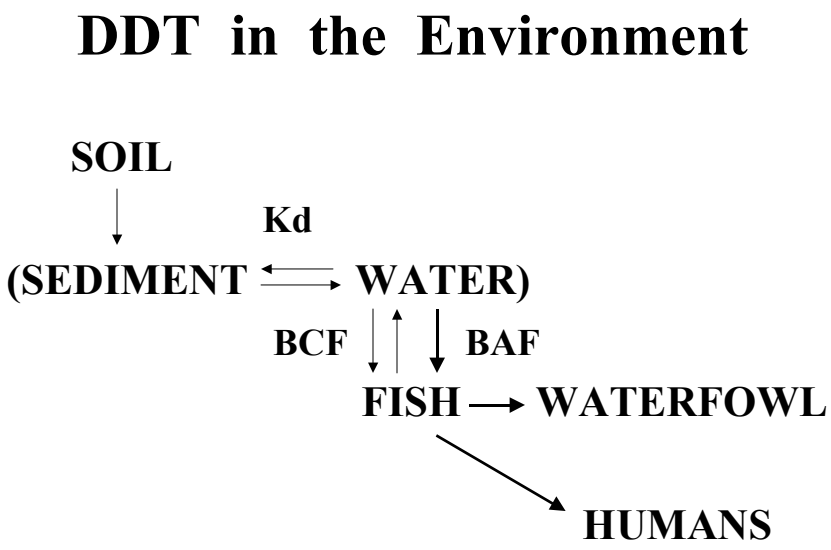

Figure 1. Conceptual model of the fate of DDT in the environment.

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.

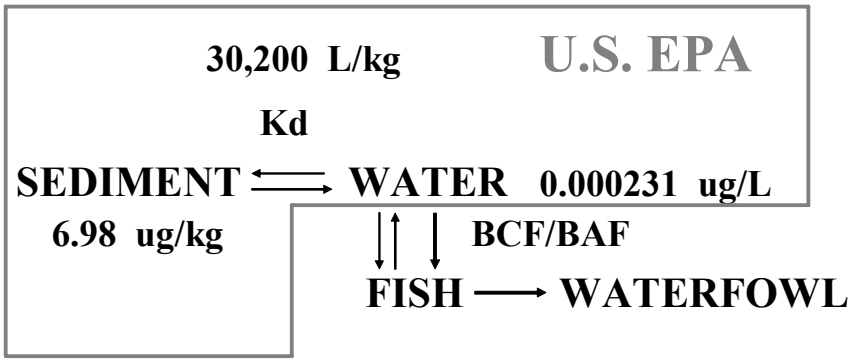

Figure 2. DDT loading concentration determined from a sediment TEL.

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).

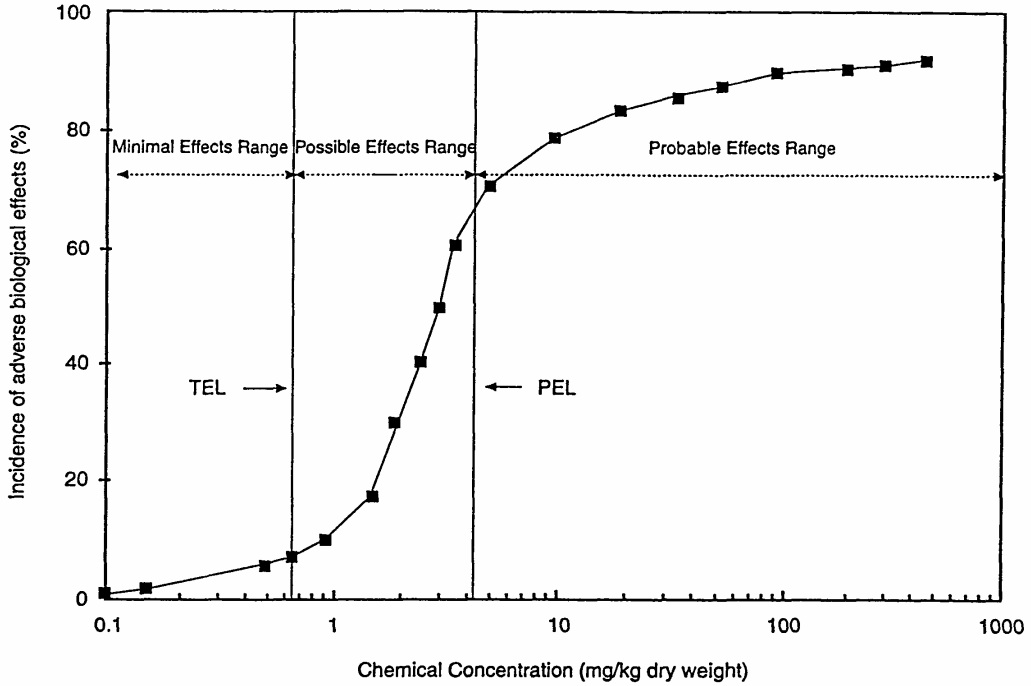

Fig. 1. Conceptual representation of the ranges of contaminant concentrations defined by SQGs and the potential for observing adverse effects within these ranges.

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	Hit	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
3782		*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 (56% 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	0.58+/-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 et 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%	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	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

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

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
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> 46

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.

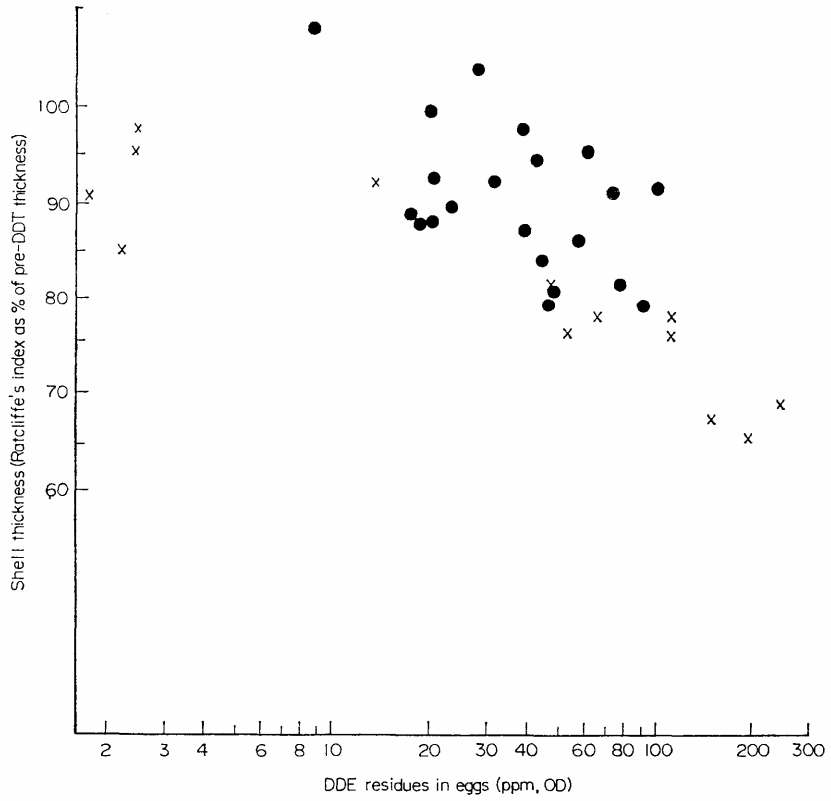

FIG. 3. Relationship between mean clutch shell-thickness and DDE residue of kestrel eggs collected in Ithaca, New York during 1970 (●) and same relationship experimentally induced with dietary DDE (×).

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

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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.

Rose, K., Total Maximum Daily Loads for Organochlorine Compounds in San Diego Creek, Upper and Lower Newport Bay, Orange County, California, draft report, August, 2006.

U. S. Food and Drug Administration, FDA/CFSAN fish and fisheries products hazards & controls guidance, Appendix 5, Table A-5, http://www.cfsan.fda.gov/~comm/haccp4x5.html , 2006.

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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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.**

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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

Quantitative Environmental Analysis, LLC
305 West Grand Ave, Suite 300
Montvale, NJ 07645
(201) 930-9890
(201) 930-9805 *fax*
dchiavelli@qeallc.com

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.

JOHN P. CONNOLLY, Ph.D., P.E., DEE

CONTACT INFORMATION

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305 West Grand Ave, Suite 300
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(201) 930-9890
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jconnolly@qeallc.com

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

APPENDIX 2

FLOW SCIENCE AND DR. JAMES L. BYARD, D.A.B.T. (2006)

SUPPLEMENTAL REPORT ON ORGANOCHLORINE COMPOUNDS: TOXAPHENE IN THE NEWPORT BAY WATERSHED

Flow Science Incorporated
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Supplemental Report on Organochlorine Compounds: Toxaphene in the Newport Bay Watershed

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SUMMARY

In 2002 the EPA established a Toxic Pollutants Total Maximum Daily Load (TMDL) that developed target loads for organochlorines (including DDT, chlordane, toxaphene, PCBs, and dieldrin) for portions of 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. The purpose of this report is to present data relevant to the proposed toxaphene TMDL and to suggest changes to that TMDL document based on the data presented.

Of the several toxaphene concentration targets proposed by the Regional Board for application in the Newport Bay watershed draft organochlorine TMDL document (see Table 1), two have been evaluated in this report—a sediment screening value of 0.1 ppb, and the OEHHA fish tissue guidance value of 30 ppb. In his review of available data, Dr. James Byard found two problems with applying the sediment screening value in the watershed. First, the value was calculated on the basis of a methodology (equilibrium partitioning) that the Regional Board has explicitly rejected in the past. Second, the value was calculated using outdated information. If more current information is used—information published by the Regional Board and the U.S. EPA—a screening value of 15.8 ppb results. If this were the concentration target that governed sediment concentrations in the watershed, there would be no need for a toxaphene TMDL.

The OEHHA screening value for toxaphene of 30 ppb as a fish tissue criterion is also evaluated in this report. Use of the OEHHA screening value as a target for the TMDL is inappropriate for several reasons. First, OEHHA values were intended to identify locations for further study, and were never intended to be regulatory endpoints. Additionally, OEHHA is in the process of revising their guidance value for toxaphene from 30 ppb to 220 ppb. No fish tissue sample from species commonly consumed by humans over the period of record in the Newport Bay watershed (i.e., since 1985) has ever exceeded 220 ppb.

Moreover, Newport Bay watershed toxaphene concentrations in red shiner fish tissue exhibit a strong decline over time, which is expected to continue into the future. While mussel tissue concentrations from the watershed do not exhibit strong trends over time, data collected since 1980 (when sampling began) indicate that 76% of mussel samples from the watershed exhibited toxaphene concentrations below the analytical detection limit. Moreover, the rate of non-detection has remained consistent over time. Also, 100% of available water samples and all of the most recent agricultural soil data (from 2004) exhibit toxaphene concentrations below analytical detection limits. Together, these data further suggest that a toxaphene TMDL is unnecessary.

Finally, a review of relevant literature indicates that the half-life of toxaphene in soil ranges from 1 to 14 years, where anaerobic conditions lead to shorter half-lives and aerobic conditions lead to longer half-lives. For sediments under aerobic conditions in the watershed (i.e., agricultural soils), we estimate that toxaphene mass has declined by at least 63% since the ban of the pesticide in 1990. For sediments under anaerobic conditions in the watershed (i.e., submerged bay and creek sediments), we estimate that toxaphene mass has declined by virtually 100% since 1990. These substantial natural rates of toxaphene removal and degradation likely account in some measure for the decline observed in red shiner tissue concentrations over time, and for the low concentrations observed in other media. In fact, the red shiner data can be used to estimate the half-life of toxaphene in the watershed, as toxaphene concentrations in the tissues of biota are a direct measure of biota exposure. These data indicate a toxaphene half-life in the watershed of about 3.4 years (obtained from red shiner data), well within the range of published data on the half-life of chlordane in the environment.

INTRODUCTION

In 2002 the EPA established a Toxic Pollutants Total Maximum Daily Load (TMDL) that developed target loads for organochlorines (including DDT, chlordane, toxaphene, PCBs, and dieldrin) for portions of 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. Toxaphene was banned in 1990. 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 – rather, these studies have found that other compounds are more 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).

This report supplements the report "DDT Analysis for the Newport Bay Watershed" (Flow Science et al., 2006). The primary purposes of this report are as follows:

1. To present analysis to date of the toxaphene standards proposed for use by the SA RWQCB in their draft organochlorine TMDL document. Specifically, analysis covers the proposed sediment screening value of 0.1 ppb and the proposed OEHHA fish tissue value of 30 ppb.

2. To summarize data from the Newport Bay watershed on toxaphene concentrations in various media, including fish and mussel tissue, creek and bay sediment, water, and agricultural soils. Comparable data for DDT were previously presented in Flow Science et al. (2006).

3. To summarize data on natural removal rates for toxaphene from watershed soils and sediments.

4. To discuss the relevance of these data to the proposed TMDL for organochlorines in portions of the Newport Bay watershed. Specifically, this report aims to recommend changes to the recently issued TMDL staff report and implementation plan.

PROPOSED TOXAPHENE TARGETS

In their forthcoming TMDL, the Santa Ana Regional Board proposes to apply several standards for toxaphene to levels of the pesticide in different media. 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.

NEW YORK STATE SEDIMENT SCREENING VALUE

As noted in Table 1, the Regional Board proposes to use a sediment screening concentration for toxaphene of 0.1 ug/kg (0.1 ppb). This screening value was published by the New York State Department of Environmental Conservation in 1998. Application

of the sediment screening value to the San Diego Creek watershed as proposed would require that toxaphene and sediment loads be reduced by 99% from current conditions. However, a recent detailed review of the screening value by Dr. James Byard indicates several problems with the SA RWQCB's use of the value (see report appendix for detailed results of Dr. Byard's review).

First, the screening value was derived using equilibrium partitioning methodology. According to this methodology, the concentration of biologically available contaminant is calculated based on a water concentration and the degree to which the contaminant tends to sorb to organic matter in sediment. This latter characteristic is accounted for by using an equilibrium partition coefficient, K_{ow}. However, although this methodology has been endorsed by the U.S. EPA Science Advisory Board and has been proposed by stakeholders in the Newport Bay watershed, the Regional Board has explicitly rejected its use in developing TMDLs. Given this prior rejection of the methodology on which the screening value is based, it is unclear why the Regional Board is willing to adopt the screening value.

Second, even if the Regional Board is willing to use the screening value, Dr. Byard found that the K_{ow} value used by the New York State Department of Environmental Conservation in deriving the screening value was outdated and 158 times lower than the K_{ow} published by both the U.S. EPA in their 2002 organochlorine TMDL for the Newport Bay watershed, and the Regional Board in their recently published draft organochlorine TMDL report. Dr. Byard found that using the updated K_{ow} in the New York State methodology yielded a sediment screening value of 15.8 ppb. This updated screening value corresponds to a toxaphene loading capacity in the San Diego Creek watershed that exceeds current loads. Thus, if the correct methodology is applied in determining the sediment screening value, there is no need for a toxaphene TMDL in the watershed.

OEHHA SPORT FISH GUIDANCE LEVEL

In the draft organochlorine TMDL report, the Regional Board presents the guidance tissue level for toxaphene in California sport fish published by the Office of Environmental Health Hazard Assessment (OEHHA) as the governing standard for fish tissue concentrations in the watershed. The OEHHA value for toxaphene is currently 30 ppb. However, OEHHA is in the process of revising this value. The OEHHA draft report entitled "Development of Guidance Tissue Levels and Screening Values for Common Contaminants in California Sport Fish: Chlordane, DDTs, Dieldrin, Methylmercury, PCBs, Selenium, and Toxaphene" (February 2006) lists a revised guidance level of 220 ppb for toxaphene. This revised value of 220 ppb represents OEHHA's most up-to-date assessment of human health hazards related to fish tissue consumption. If an OEHHA value is to be used to regulate fish tissue concentrations in

the watershed—a regulatory role which OEHHA values were never intended to play—220 ppb should be used, not 30 ppb as the draft organochlorine TMDL proposes.

Moreover, since June 1998 all red shiner concentrations sampled in the watershed have been below 220 ppb (see next section). In the record of data for species other than red shiner (dating to 1985), no tissue sample has ever exceeded 220 ppb. If it is further considered that red shiner are not generally consumed by humans—they are generally a bait fish—this means that data for typically-consumed fish species have never exceeded the revised OEHHA value during the period of record in the Newport Bay watershed. This suggests that a TMDL is not necessary for the purposes of reducing toxaphene concentrations in fish species inhabiting the Newport Bay watershed.

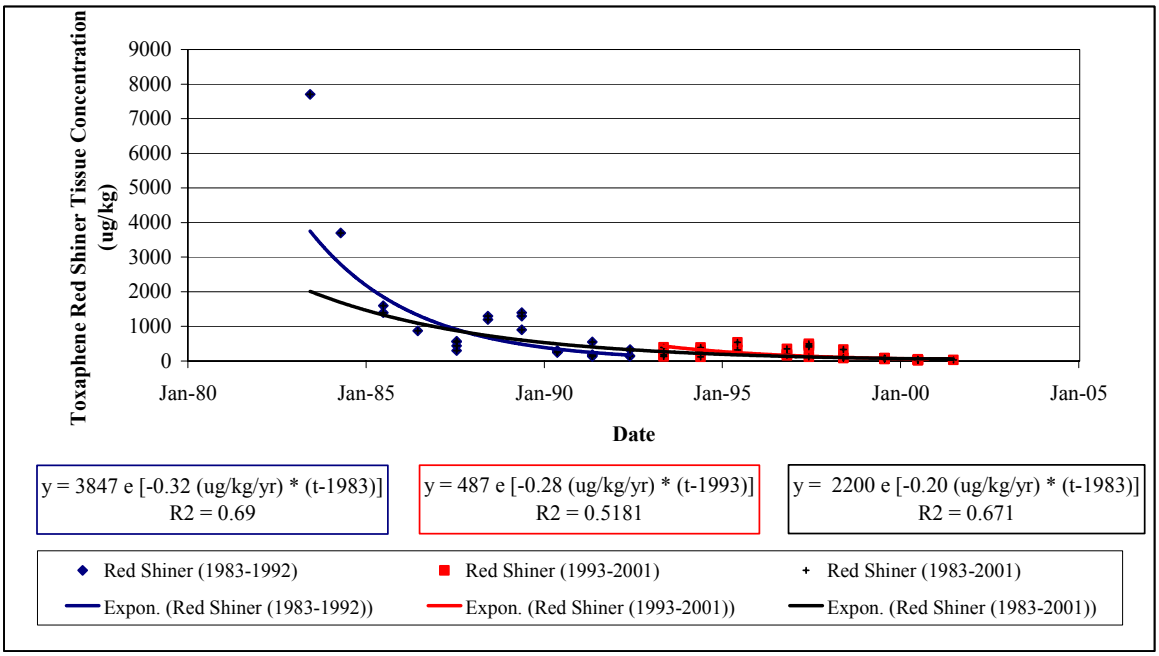
TOXAPHENE CONCENTRATIONS

The following sections present available toxaphene data for the watershed. Trends in toxaphene concentrations—particularly fish tissue concentrations—are evident in data collected for 20 years in the Newport Bay watershed.

FISH TISSUE

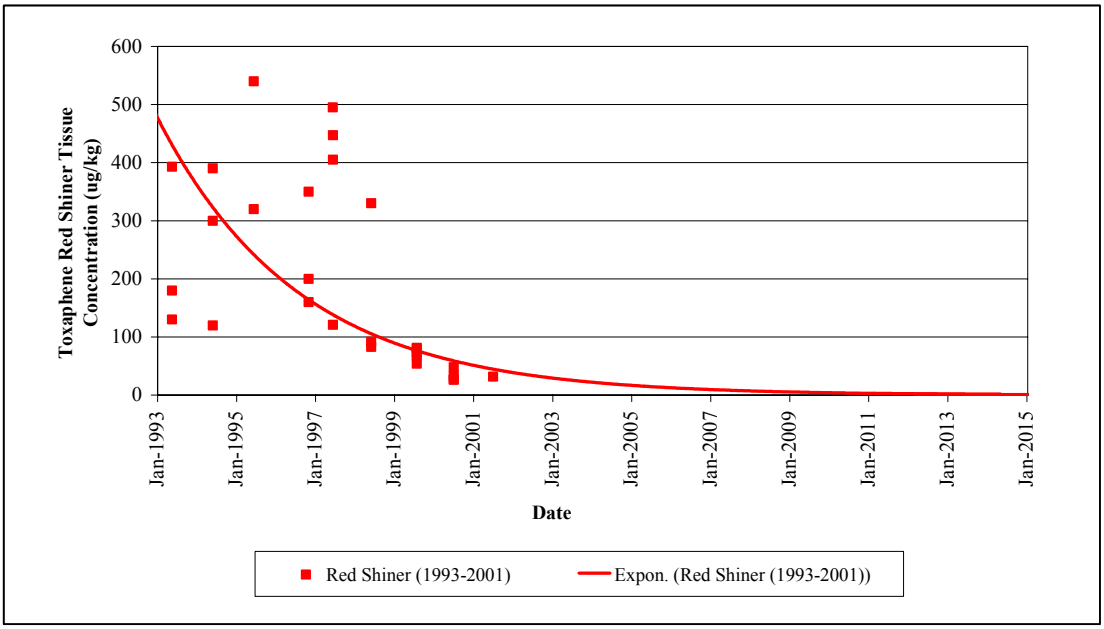
In the case of red shiner, toxaphene tissue concentration data dating from 1983 show a substantial decline (see Figures 1 and 2). As with DDT, red shiner may be taken as an indicator of toxaphene concentrations in receiving waters within the watershed, as red shiners are local, short-lived species. The primary statistical approach to establishing the declining trend in toxaphene concentrations in the watershed has been to derive first-order decay constants using historical toxaphene data for red shiner fish tissue. The equations of these curves are indicated in Figures 1 and 2.

Figure 1. Toxaphene concentrations in red shiner, San Diego Creek and Peters Canyon Wash (1983-2001)



Source: SWRCB, Toxic Substances Monitoring Program (TSMP).

Figure 2. Toxaphene concentrations in red shiner, San Diego Creek and Peters Canyon Wash, projected through 2010



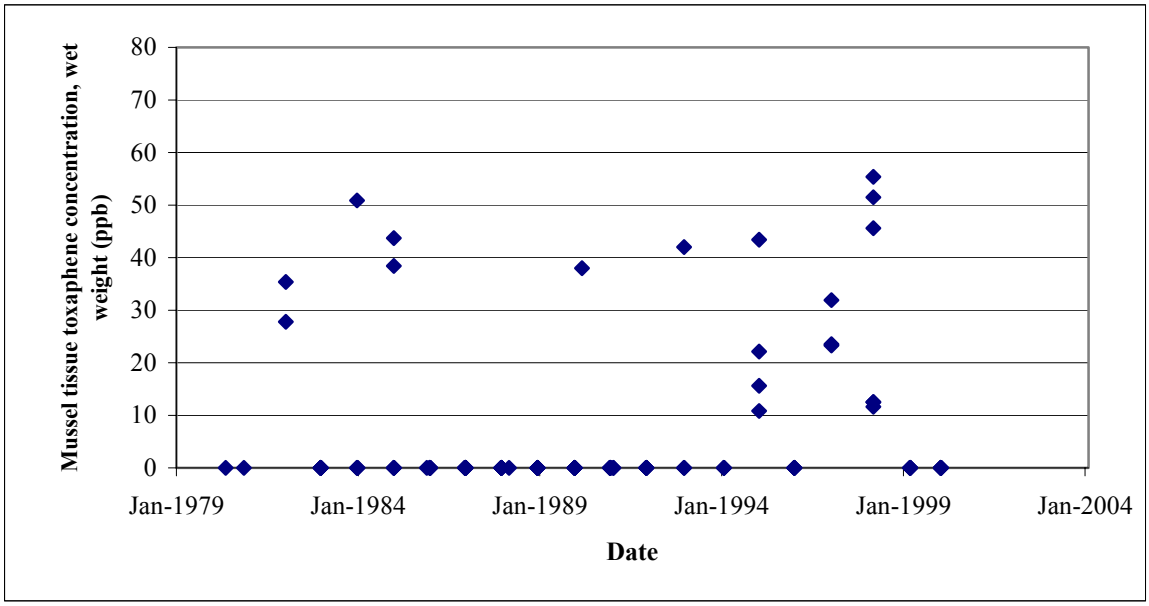
Source: SWRCB, Toxic Substances Monitoring Program (TSMP).

Two considerations suggest the robustness of the downward trend in red shiner tissue toxaphene concentrations. First, when all red shiner data are considered together, a statistically strong (R^2 value of 0.671) downward trend in toxaphene concentration is evident. Second, the statistical analysis that characterizes these trends has been confirmed by splitting the data set for red shiners into two separate sets consisting of the first ten years of data (1983-1992) and the second ten years of data (1993-2001). Linear regression analyses on the natural logarithmically transformed data sets using a 95% confidence range confirm that the calculated first-order decay rates for the red shiner toxaphene data are statistically similar for the full data set and for the sub-sampled datasets. The decay rate (-0.00055 per day or -0.20 per year) obtained for the full red shiner dataset (1983-2001) is equivalent to a half-life of 3.4 years for toxaphene in the watershed.

MUSSEL TISSUE

Mussel tissue data from Newport Bay for the period 1980 through 2000 do not show any statistically significant trends in wet weight toxaphene concentrations over time (Figure 3). However, toxaphene concentrations in 84 out of the 111 samples (76%) collected over the 21-year period were below analytical detection limits. In other words, the vast majority of samples collected were below the detection limit for toxaphene. Moreover, the frequency of non-detect results was consistent over time.

Figure 3. Mussel toxaphene concentration data, Newport Bay watershed

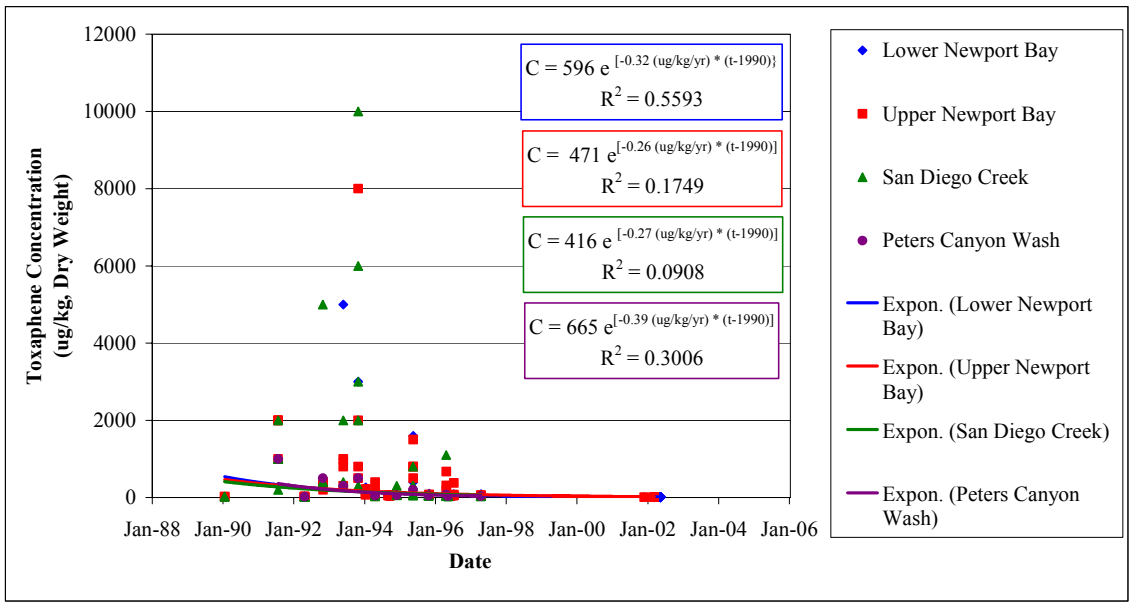


Source: State Mussel Watch (SMW) Program, 1980-2000.

BAY AND CREEK SEDIMENT

Watershed sediment data are available beginning in 1990 and also demonstrate a clear decline in toxaphene concentrations (see Figure 4). To evaluate the robustness of the downward trend, the data set for watershed sediment was divided into four geographically distinct sets representing Lower Newport Bay, Upper Newport Bay, San Diego Creek, and Peters Canyon Wash. This analysis showed that the calculated 'decay rates' for the sediment data regressions at the four locations were similar within a 95% confidence range, suggesting that the downward trend in concentrations is statistically significant. The downward trend in concentrations is particularly strong for Lower Newport Bay sediment. Sediment samples collected from Upper Newport Bay, the Rhine Channel, San Diego Creek, and Peters Canyon Channel also demonstrate downward trends in toxaphene concentrations over time, though these trends are less robust than those for Red Shiner and Lower Newport Bay sediment.

Figure 4. Sediment toxaphene concentrations, Newport Bay watershed (1990-2002)



Sources: SCCWRP database, 1990-2002; Bay and Greenstein, 2003.

Despite evidence of downward trends in sediment toxaphene concentrations in the watershed, several factors complicate assessment of the sediment data. 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 may be inappropriate. Second, there is

significant movement of sediment into, out of, and within the Bay and its watershed such that even samples taken in the same location at two different times may not represent the change in toxaphene concentration for a specific quantity of sediment. Sediment movement results both from the natural flow of water and sediment in the Bay and its watershed, as well as from periodic major dredging in the Bay, which has occurred in 1983, 1985, 1988, and 1999. Third, sediment concentrations in Newport Bay may be more indicative of toxaphene loads from years or decades past, since Bay sediments are transported from the upper watershed in a highly variable, episodic manner. Thus, toxaphene concentrations in Bay sediments reflect toxaphene 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. For all these reasons, the trends evident in the available sediment toxaphene concentration data for Newport Bay should not be weighted too heavily in overall assessment of toxaphene concentrations in the watershed.

WATER

Only ten toxaphene water concentration data points were available for the Newport Bay watershed. Table 5 summarizes these data. All data were collected in 2002, and none of the 10 data points were above detection limits, which were 10 ng/l. The CTR human health regulatory threshold for toxaphene in water is 0.00073 ug/L, or 0.73 ng/L.

Table 2. Toxaphene concentrations in water, Newport Bay

Date	Location	Sample Station	Kind of Sample	Toxaphene Concentration (ng/L)
3/12/2002	Upper Newport Bay	NB 10	Water	ND*
3/12/2002	Rhine Channel	NB 3	Water	ND*
3/7/2002	San Diego Creek	Campus Drive	Water (wet weather)	ND*
3/7/2002	San Diego Creek	Campus Drive	Water (wet weather)	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 (wet weather)	ND*
11/8/2002	San Diego Creek	Campus Drive	Water (wet weather)	ND*

* Detection limit = 10 ng/L.
Source: Bay and Greenstein, 2003.

AGRICULTURAL SOILS

For toxaphene there were fewer agricultural soil data available than for DDT. As with DDT data, samples from different years were taken in different locations since the

purpose of sampling was to assess site conditions for planning and development purposes, not to establish concentration trends over time in the watershed. Like the DDT soil data, the majority of toxaphene soil samples returned concentrations below detection limits. For example, for 2004 data, 222 of 230 six-inch depth soil samples, and 44 of 45 soil samples at depths greater than 24 inches, were below the analytical detection limit of 0.1 ppm. Although no statistically clear trends in soil toxaphene concentrations can be demonstrated from a dataset where about 96% of samples have toxaphene concentrations below detection limits, it is clear from these data that the mass of toxaphene in the watershed is currently quite small. This is consistent with expectations based upon the half-life of toxaphene, as detailed above. Table 2 summarizes available agricultural soil toxaphene concentrations.

Table 3. Historical toxaphene concentrations for agricultural soils in Newport Bay watershed

Year	0-6 inch Sample Depth			12-18 inch Sample Depth			>24 inch Sample Depth			Detection Limits (ppm)
	Average Observed Toxaphene (ppm)	Max Observed Toxaphene (ppm)	Sample Size (n)	Average Observed Toxaphene (ppm)	Max Observed Toxaphene (ppm)	Sample Size (n)	Average Observed Toxaphene (ppm)	Max Observed Toxaphene (ppm)	Sample Size (n)	
1989	0.65	0.94	3	0.55	0.55	3	ND	ND	3	0.25
1990	0.21	0.22	2	ND	ND	1	ND	ND	3	0.16
1995	ND	ND	19							0.06
2004	ND	ND	230				ND	ND	45	0.1

Sources: Byard, 1989; Byard, 1990; NMG Geotechnical, 1996; The Irvine Company, 2006.

NATURAL TOXAPHENE REMOVAL RATES

The observed decline in toxaphene concentrations in fish tissue and the low observed toxaphene concentrations in watershed soils and sediments are partly attributable to the natural removal of toxaphene from the watershed. The half-life of toxaphene in soil is reported as ranging from 1-14 years (U.S. EPA, 1999). The wide range is attributable to apparently differing degradation rates for toxaphene under aerobic and anaerobic conditions (U.S. Dept. Health and Human Services, 1996). Under anaerobic conditions the half-life of toxaphene in soil and sediment has been reported as on the order of weeks to months by the U.S. EPA (1979). However, under aerobic soil conditions, Nash and Woolson (1967) reported a half-life of 11 years. If we conservatively assume that aerobic conditions are most common in the Newport Bay watershed—suggesting a half-life on the order of 11 years—given that the use of toxaphene was banned in 1990 and excluding other loss mechanisms, the mass of toxaphene in the agricultural soils of the Newport Bay watershed would have declined by at least 63% over the past 16 years due solely to natural removal. However, if we assume anaerobic conditions—conditions typical of sediments submerged in water, such as bay sediments—the half-life of toxaphene is on

the order of weeks or months. This suggests sediments which remain consistently submerged in the watershed should currently contain very little toxaphene. The half-life for toxaphene in the watershed that was estimated using red shiner fish tissue data (3.4 years) is consistent with these estimates for the half-life of toxaphene in watershed soils.

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APPENDIX

SCIENTIFIC COMMENTARY ON
THE NEW YORK STATE SEDIMENT SCREENING LEVEL FOR
TOXAPHENE

James L. Byard, Ph.D., D.A.B.T.
October 2, 2006

SCIENTIFIC COMMENTARY ON THE NEW YORK STATE SEDIMENT SCREENING LEVEL FOR TOXAPHENE

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SUMMARY

U.S. EPA and SARWQCB are using a toxaphene TMDL sediment target of 0.1 ppb for the San Diego Creek Watershed. The target is adopted from a New York State Department of Conservation freshwater sediment screening level. The screening level is based on equilibrium partitioning, a method proposed by stakeholders and refuted by SARWQCB. Apparently, SARWQCB is unaware of the method used by New York State to derive the sediment screening level used in the toxaphene TMDL. The sediment target of 0.1 ppb would require a 99 % reduction in both the toxaphene load and in the sediment load in the San Diego Creek Watershed. New York State used a K_{ow} for toxaphene that is outdated and is 158-fold lower than the K_{ow} published by both the U.S. EPA and SARWQCB in their respective TMDL reports. Using the updated K_{ow} in the New York State method for determining the sediment screening level, yields a sediment target of 15.8 ppb. This updated target level results in a loading capacity for San Diego Creek that exceeds the existing load, negating the need for a toxaphene TMDL in the Watershed.

INTRODUCTION

On June 14, 2002, the U.S. EPA, Region IX (EPA), promulgated a total maximum daily load (TMDL) for toxaphene in the San Diego Creek 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 toxaphene 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 toxaphene TMDL and found it difficult to understand (Rose, 2005a; Rose, 2005b; Byard, 2005a; Byard, 2005b). There were many errors, wrong assumptions and contradictions. An unusually low sediment target of 0.1 ppb was cited as a screening level from the New York Sate Department of Environmental Conservation. In their most recent draft report on the organochlorine TMDLs, staff at the SARWQCB (Rose, 2006) have used the same 0.1 ppb target in sediment to achieve a toxaphene residue target in fish. This report will take a detailed look at the scientific basis for the New York State Department of Environmental Conservation sediment screening level for toxaphene. The 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 toxaphene and is applicable to the San Diego Creek Watershed. A conceptual model is also helpful in understanding the derivation of a TMDL. Figure 1 portrays a conceptual model for the fate of toxaphene in the environment relevant to a TMDL for San Diego Creek.

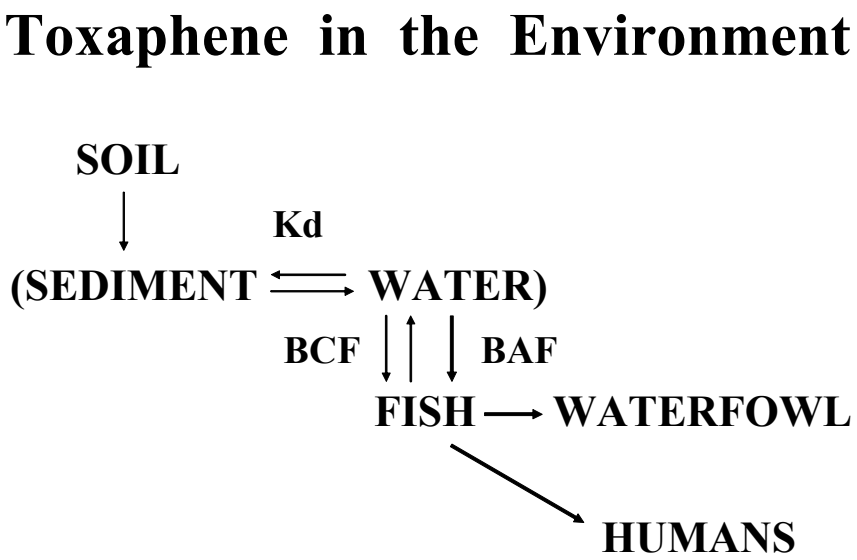

Figure 1. Conceptual model of the fate of toxaphene in the environment.

Soil residues are eroded into channels in the San Diego. A distribution coefficient, Kd, describes the equilibrium between toxaphene in sediment and toxaphene in water. Bioconcentration factors (BCF) describe the equilibrium between water and highly perfused fish tissues. Bioaccumulation factors (BAF) describe the accumulation of toxaphene 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 toxaphene in poorly perfused adipose tissue of fish, birds and humans.

The U.S. EPA used the same conceptual model to determine the loading capacity and existing loads in promulgating the DDT TMDL in 2002. Figure 2 illustrates the derivation of the loading concentration for San Diego Creek.

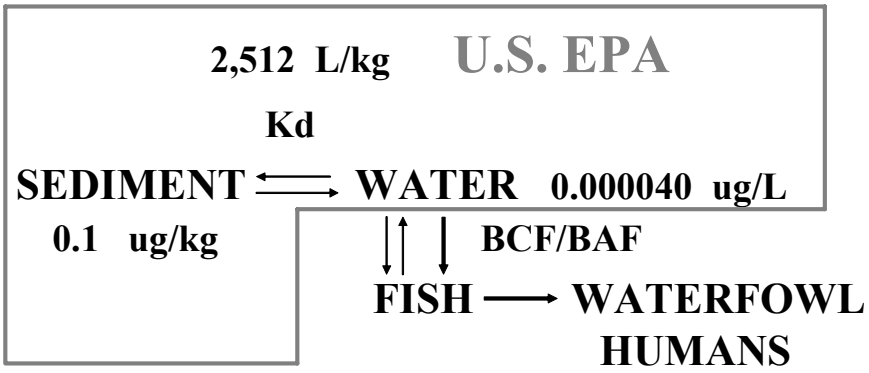

Figure 2. Toxaphene loading concentration determined from a sediment target.

Because most of the toxaphene in the aquatic environment is bound to sediment, the U.S. EPA used a toxaphene sediment target (New York State Department of Environmental Conservation, 1998) they asserted was necessary to achieve beneficial uses. A sediment target of 0.1 ppb was used for the San Diego Creek Watershed. The derivation of the New York State sediment target is explained in the following section.

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION FRESHWATER SEDIMENT SCREENING LEVEL FOR TOXAPHENE

New York State chose the equilibrium partition method for the derivation of a sediment screening level for toxaphene (New York State Department of Environmental Conservation, 1998). The method is explained in the excerpt below that is copied directly from the executive summary.

1. Executive Summary

The Department of Environmental Conservation originally proposed sediment criteria in 1989, as an appendix of a Cleanup Standards Task Force Report. These criteria were controversial because the proposed methodology, equilibrium partitioning, had not yet been endorsed by the U.S. Environmental Protection Agency (EPA) Science Advisory Board, and because the criteria themselves were perceived as remediation target concentrations. This revised sediment criteria document was prepared to incorporate scientific literature published since 1989, and to establish the purpose of sediment criteria for screening; that is, to identify areas of sediment contamination and to make a preliminary assessment of the risk posed by the contamination to human health and the environment. Criteria are developed for two classes of contaminants - non-polar organic contaminants and metals. Non-polar organic contaminant criteria are derived using the equilibrium partitioning approach, which has now been endorsed by the EPA Science Advisory Board. This approach estimates the biological impacts that a contaminant may cause based on it's affinity to sorb to organic carbon in the sediment. The concentration of biologically available contaminant is predicted and related to potential toxicity and bioaccumulation by using existing criteria established for the water column. New York State water quality standards and guidance values are used to derive sediment criteria. EPA water quality criteria are used only when New York State has not published a standard or guidance value for a particular compound. Water quality criteria for bioaccumulation proposed by the Divisions of Fish and Wildlife and Marine Resources are used when no New York State water quality standard or guidance value for bioaccumulation has been developed.

The details of the equilibrium partitioning method are explained in the following exerpt from pages 7 and 8, using PCBs as an example.

C. Derivation of Sediment Criteria using Equilibrium Partitioning

To derive an organic carbon normalized sediment criterion, two items of information are required:

A. An ambient water quality criterion for a particular contaminant;

B. the K_{OW} partition coefficient for the contaminant;

For example, the PCB water quality criterion (see footnote 1 on page 4) for the protection of piscivorous wildlife from bioaccumulation is 0.001 ug/l. The K_{OW} for PCB is $10^{6.14}$, or 1,380,384.3 l/kg. The organic carbon normalized PCB sediment criterion (SCoc) would be:

$$SC_{OC} = WQC * K_{OW}$$

$$PCB\ SC_{OC} = 0.001\ /ug/l * 1{,}380{,}384.3\ l/kg * 1\ kg/1{,}000\ gOC$$

$$1.38\ (\approx 1.4)\ \mu g/gOC$$

1 kg/1,000 gOC is a conversion factor.

The meaning of the criterion is: based on the equilibrium partitioning characteristic of PCBs, in order not to exceed the water quality criterion of 0.001 ug/l in the pore water, the concentration of PCB in the sediment must not exceed 1.4 μg for each gram of organic carbon in the sediment.

To apply this SC_{OC} on a site specific basis, the concentration of organic carbon in the sediment at the site must be known. If a sediment sample was known to contain 3% organic carbon, the site specific sediment criterion (SC) for PCB could be derived:

$$SC = SC_{OC} * f_{OC}$$

f_{OC} = 3% OC/kg sediment = 30 gOC/kg

PCB SC = 1.4 μg/gOC * 30 gOC/kg = 42 μg PCB/kg sediment

This criterion states that: if there are less than 42 ug PCB/kg of sediment in a sediment containing > 3% organic carbon, there is no appreciable risk to piscivorous wildlife from consuming fish or other aquatic life from the water body over the contaminated sediment.

The method assumes that the organic carbon partitioning coefficient, K_{oc} is approximately the same as the octanol-water partitioning coefficient, K_{ow}. The executive summary ends with a reference to Tables 1 and 2 which list the sediment screening levels as can be seen in the following excerpt copied directly from the executive summary.

Table 1 lists sediment criteria for 64 non-polar organic compounds or classes of compounds, and Table 2 lists sediment criteria for 12 metals.

The following excerpt of the sediment screening level for toxaphene is reproduced from Table 1.

Table 1. Sediment criteria for non-polar organic contaminants. Water quality criteria used are taken from Togs 1.1.1. If a water quality criterion was not listed in TOGS 1.1.1., then an EPA criterion was used. These are annotated with the suffix (E). EPA criteria were extracted from the "Water Quality Criteria Summary" chart (EPA, 1991). EPA water quality criteria for the protection of human health (bioaccumulation) were taken from the "Recalculated Values - Organisms Only" column. Wildlife (bioaccumulation) and Human Health (bioaccumulation) protection criteria were derived in Appendix 1, unless TOGS 1.1.1. (bioaccumulation) criteria already existed. Although these criteria are only proposed, they are useful as guidance for estimating potential human health risks. These criteria are annotated with a suffix (P), for "Proposed criteria values".

			Levels of Protection							
			Human Health Bioaccumulation		Benthic Aquatic Life Acute Toxicity		Benthic Aquatic Life Chronic Toxicity		Wildlife Bioaccumulation	
Contaminant	$LogK_{ow}$	Fresh-FW Salt -SW Both -FS	Water Qual Criteria µg/l	Sediment Criteria µg/gOC	Water Qual Criteria µg/l	Sediment Criteria µg/gOC	Water Qual Criteria µg/l	Sediment Criteria µg/gOC	Water Qual Criteria µg/l	Sediment Criteria µg/gOC
Toxaphene	3.3	FW	0.009 (P)	0.02	1.6	3.2	0.005	0.01		
		SW	0.009 (P)	0.02	0.07	0.14	0.005	0.01		

The $\log K_{ow}$ value was not specifically referenced, so one is not able to determine the source of this very important constant in the equilibrium partitioning method. The K_{ow} value appears to be more than two orders of magnitude too low (a $\log K_{ow}$ of 3.3 is equal to a K_{ow} of 1,995). For example, U.S. EPA (2002) and the SARWQCB (Rose, 2006) list a $\log K_{ow}$ for toxaphene of 5.5 (a $\log K_{ow}$ of 5.5 is equal to a K_{ow} of 316,228; 316,228/1,995 = 158). The source of the $\log K_{ow}$ value is referenced as "Southerland" EPA report, as reproduced below from the EPA 2002 TMDL report.

Newport Bay Toxics TMDLs

III. References

Agency for Toxic Substances and Disease Registry (ATSDR) 2001. http://www.atsdr.cdc.gov/

De Bruijn, J and F Busser, W Seinen, J Hermens 1989 Determination of Octanol/Water Partition Coefficients for Hydrophobic Organic Chemicals with the "slow-stirring" method. *Env. Tox. &Chem.* **8:** 499-512

EPA (Internal) Report 1995 "Summary of Measured , Calculated and Recommended Log Kow values" prepared for Elizabeth Southerland, Chief Risk Assessment and Management Branch Standards and Applied Science Division, Office of Water by S. Karickhoff and J. M. Long, Environmental Research Laboratory-Athens.

The SARWQCB draft report (Rose, 2006) cites the same $\log K_{ow}$ value and reference. If one were to apply the $\log K_{ow}$ of 5.5 to the New York State water quality criterion of 0.005 ug/l, the freshwater sediment criterion would be 1.58 ug toxaphene/ug sediment organic carbon. At 1 % organic carbon in sediment, the sediment criterion becomes 15.8 ppb instead of the 0.1 ppb value derived by New York State Department of Conservation.

Stakeholders in the San Diego Creek Watershed have previously encouraged the SARWQCB to consider using equilibrium partitioning for the derivation of sediment targets from water column criteria (Byard, 2005b). The SARWQCB has declined to use equilibrium partitioning to set sediment targets (Rose, 2006), but has unknowingly used this method by adopting the New York State sediment screening level for toxaphene. The Regional Water Board has not only created a conflict in their choice of a very low screening level, but in doing so have adopted a K_{ow} value more than two orders of magnitude different from the value that they and the U.S EPA have published in their respective TMDL documents.

ANALYSIS

The consequences of using the 0.1 ppb sediment target for toxaphene are major. The SARWQCB staff draft report (Rose, 2006) estimated a loading capacity of 5.67 grams of toxaphene per year and an existing load of 536 grams of toxaphene per year. Assuming these loads, there would have to be a 99 % reduction in the toxaphene load to meet the TMDL. Since almost all of the toxaphene is bound to sediment, the sediment load reduction in the watershed would also have to be 99 %! A sediment load reduction of this magnitude would be impractical in a major storm event. However, if we use the updated $\log K_{ow}$ of 5.5 published by both the U.S. EPA and the SARWQCB, the New York State sediment screening level goes up 158-fold. The sediment target now becomes 15.8 ppb, resulting in a loading capicity of 896 grams per year, a value greater than the estimated existing load of 536 grams per year. Therefore, a TMDL for toxaphene in the San Diego Creek Watershed is not necessary.

CONCLUSIONS

- U.S. EPA and SARWQCB are using a toxaphene TMDL sediment target of 0.1 ppb for the San Diego Creek Watershed adopted from a New York State Department of Conservation freshwater sediment screening level.

- The New York State sediment screening level is based on equilibrium partitioning, a method proposed by stakeholders, but which the SARWQCB rejected for use in the organochlorine TMDLs. Apparently, SARWQCB is unaware of the method used by New York State to derive the sediment screening level used in the toxaphene TMDL.

- The toxaphene sediment target of 0.1 ppb would require a 99 % reduction in the toxaphene load and in the sediment load in the Watershed.

- New York State used a K_{ow} for toxaphene that is outdated and is 158-fold lower than the K_{ow} published by both the U.S. EPA and SARWQCB.

- Correcting the K_{ow} to the updated value in the New York State determination of the sediment screening level, results in a sediment target

of 15.8 ppb and a loading capacity that exceeds the existing load estimated by SARWQCB.

- Because the loading capacity exceeds the existing load, a TMDL for toxaphene in the San Diego Creek Watershed is not necessary.

REFERENCES

Byard, J., TMDL for DDT in the San Diego Creek and Newport Bay. Discussion of issues for implementation, Presentation to SARWQCB staff, April 1, 2005a.

Byard, J., Observations on the DDT TMDL for San Diego Creek and Newport Bay, presentation at TMDL workshop, June 22, 2005b.

New York State Department of Environmental Conservation, Technical guidance for screening contaminated sediments. Division of Fish, Wildlife and Marine Resources, 1998.

Rose, K., SARWQCB, TMDL calculations, **http://www.waterboards.ca.gov/santaana/pdf/tmdl/oc/OCsExistingLoadsSDCreekPost.pdf**, **http://www.waterboards.ca.gov/santaana/pdf/tmdl/oc/OCsLoadingCapSDCreekPost.pdf**; **http://www.waterboards.ca.gov/santaana/pdf/tmdl/oc/OCsTMDLCalculations-NPBayPost.pdf**, 2005a.

Rose, K., Organochlorine Compounds TMDLs, Upper and Lower Newport Bay, Rhine Channel, San Diego Creek. Presentation given at TMDL workshop, June 22, 2005b.

Rose, K., Total maximum daily loads for organochlorine compounds in San Diego Creek, Upper and Lower Newport Bay, Orange County, California, draft report, August, 2006.

U.S. EPA, Total maximum daily loads for toxic pollutants, San Diego Creek and Newport Bay, California, June 14, 2002.

APPENDIX 3

FLOW SCIENCE (2006)

SUPPLEMENTAL REPORT ON ORGANOCHLORINE COMPOUNDS: CHLORDANE IN THE NEWPORT BAY WATERSHED

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Supplemental Report on Organochlorine Compounds: Chlordane in the Newport Bay Watershed

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SUMMARY

In 2002 the EPA established a Toxic Pollutants Total Maximum Daily Load (TMDL) that developed target loads for organochlorines (including DDT, chlordane, toxaphene, PCBs, and dieldrin) for portions of 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. The purpose of this report is to present data relevant to the proposed chlordane TMDL and to suggest changes to that TMDL document based on the data presented.

Dr. James L. Byard reviewed the basis for the Regional Board's claim in the draft organochlorine TMDL document that chlordane is responsible for sediment toxicity in Newport Bay and found it to be baseless (see Appendix for details). First, the Regional Board took chlordane concentrations in sediments to be toxic since they exceeded the effects range median (ERM). However, Dr. Byard found that the applicable ERM was based on faulty data, and that if correct data had been used the ERM would have been well above observed sediment chlordane concentrations. Second, where the Regional Board concluded that chlordane was the cause of observed amphipod toxicity, Dr. Byard pointed out that the correlation between chlordane concentrations and toxicity does not demonstrate a cause-effect relationship between the two factors, and that in fact dose-response bioassays in amphipods and more sensitive aquatic species indicate that chlordane concentration thresholds that result in toxicity to the most sensitive aquatic species are well above the highest levels of chlordane reported by the Regional Board for Newport Bay sediments. These considerations indicate that a chlordane TMDL is unnecessary for the sake of addressing observed sediment toxicity in the Bay.

Of the several chlordane concentration targets proposed by the Regional Board for application in the Newport Bay watershed draft organochlorine TMDL document (see Table 1), one has been evaluated in this report—the OEHHA fish tissue guidance value of 30 ppb. Use of the OEHHA screening value to establish TMDL targets would be problematic for several reasons. First, OEHHA values were intended to identify locations for further study, and were never intended to be regulatory endpoints. Additionally, OEHHA is in the process of revising their guidance value for chlordane from 30 ppb to 200 ppb. No fish tissue sample from any species since 1987—prior to the ban of chlordane—has exceeded 200 ppb.

Moreover, Newport Bay watershed chlordane concentrations in red shiner fish tissue and mussel tissue exhibit a strong decline over time, which is expected to continue into the future. Also, 100% of available water samples and all of the most recent agricultural

soil data (from 2004) exhibit chlordane concentrations below analytical detection limits. Together, these data further suggest that a chlordane TMDL is unnecessary.

Finally, a review of relevant literature indicates that the half-life of chlordane in soil ranges from 1 to 10 years (Hornsby et al., 1996). Given that the use of chlordane was banned in 1988, and assuming a very conservative half-life of 10 years and no other loss mechanisms, the mass of chlordane in the agricultural soils of the Newport Bay watershed would have declined by almost 75% over the past 18 years due solely to natural removal processes. However, if the half-life is in fact one year, the decline would effectively be 100%. These substantial natural rates of toxaphene removal and degradation likely account in some measure for the decline observed in red shiner and mussel tissue concentrations over time, and for the low concentrations observed in other media.

INTRODUCTION

In 2002 the EPA established a Toxic Pollutants Total Maximum Daily Load (TMDL) that developed target loads for organochlorines (including DDT, chlordane, toxaphene, PCBs, and dieldrin) for portions of 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. Chlordane was banned in 1988. 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 – rather, these studies have found that other compounds are more 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).

This report supplements the report "DDT Analysis for the Newport Bay Watershed" (Flow Science et al., 2006). The primary purposes of this report are as follows:

1. To present analysis to date of the chlordane standards proposed for use by the Regional Board in their draft organochlorine TMDL document. Specifically, analysis covers the OEHHA fish tissue value of 30 ppb, and examines the Regional Board's claim that chlordane is responsible for sediment toxicity in the Bay.

2. To summarize data from the Newport Bay watershed on chlordane concentrations in various media, including fish and mussel tissue, creek and bay sediment, water, and agricultural soils. Comparable data for DDT were previously presented in Flow Science et al. (2006).

3. To summarize data on natural removal rates for chlordane from watershed soils and sediments.

4. To discuss the relevance of these data to the proposed TMDL for organochlorines in portions of the Newport Bay watershed. Specifically, this

report aims to recommend changes to the recently issued TMDL staff report and implementation plan.

PROPOSED CHLORDANE TARGETS

In their forthcoming TMDL, the Santa Ana Regional Board proposes to apply several standards for chlordane to levels of the pesticide in different media. 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.

SEDIMENT TOXICITY THRESHOLDS

In the Draft Organochlorine TMDL document, the Regional Board claims that chlordane is causing acute toxicity in Newport Bay. As evidence, they point to the fact that sediment chlordane concentrations exceed the effects range median (ERM) for chlordane in sediments, and to the fact that chlordane is present in sediments that are toxic to benthic organisms. However, as Dr. James L. Byard points out, there are several problems with this evidence which undermine the Regional Board's claim that chlordane is responsible for toxicity in the Bay (details of Dr. Byard's assessment are provided in the Appendix).

First, the data set used in formulating the chlordane ERM is flawed, resulting in an ERM that greatly underestimates the toxicity threshold for benthic organisms due to chlordane in sediments. For example, several data points were calculated using outdated partition coefficients, and other data were gathered using techniques that are inappropriate for assessing sediment toxicity. If valid data were used, the ERM would be considerably higher and would be greater than the observed chlordane concentrations in Newport Bay sediments.

Second, the SA RWQCB's claim that chlordane is causing toxicity to benthic organisms is partially based on a study by the Bay Protection and Toxic Cleanup Program (BPTCP). Based on this study, the SA RWQCB concluded that there is a correlation between sediment chlordane residues and both the inhibition of larval development in purple sea urchins and the disruption of benthic communities in the Bay. However, contrary to the SA RWQCB's conclusion, the BPTCP study did not in fact document these findings, and thus that report does not support the SA RWQCB's claim that chlordane is causing benthic toxicity.

Finally, as noted, the SA RWQCB cites the fact that chlordane is present in Bay sediments found to be toxic to amphipods as evidence that chlordane is causing the observed toxicity. However, as Dr. Byard points out, the observed correlation between chlordane concentration and toxicity does not necessarily indicate a cause-effect relationship between the two factors. In fact, dose-response bioassays in amphipods and more sensitive aquatic species indicate that chlordane concentration thresholds that result in toxicity to the most sensitive aquatic species are well above the highest levels of chlordane reported by SA RWQCB for Newport Bay sediments. Moreover, several recent studies of acute toxicity in the Bay have concluded that it is unlikely that organochlorine compounds are responsible for observed toxicity, and that organophosphate pesticides, such as diazinon and chlorpyrifos, and possibly pyrethroids are more likely the toxic agents (Lee and Taylor, 2001; Bay et al., 2004).

Thus, insofar as observed chlordane concentrations in Newport Bay do not exceed a scientifically defensible ERM value, and insofar as it is highly unlikely that chlordane is responsible for observed sediment toxicity, there is no basis in sediment toxicity for the proposed chlordane TMDL.

OEHHA SPORT FISH GUIDANCE LEVEL

In the draft organochlorine TMDL report, the SA RWQCB proposes to use the guidance tissue level for chlordane in California sport fish published by the Office of Environmental Health Hazard Assessment (OEHHA) as the governing standard for fish tissue concentrations in the watershed. The OEHHA value for chlordane is currently 30 ppb. However, OEHHA is in the process of revising this value. The OEHHA draft report entitled "Development of Guidance Tissue Levels and Screening Values for Common Contaminants in California Sport Fish: Chlordane, DDTs, Dieldrin, Methylmercury, PCBs, Selenium, and Toxaphene" (February 2006) lists a revised guidance level of 200 ppb for chlordane. This revised value of 200 ppb represents OEHHA's most up-to-date assessment of human health hazards related to fish tissue consumption. If an OEHHA value is to be used to regulate fish tissue concentrations in the watershed—a regulatory role which OEHHA values were never intended to play—200 ppb should be used, not 30 ppb as the draft organochlorine TMDL proposes.

Moreover, since 1987 not a single fish tissue sample from the Bay for any species has exceeded a chlordane concentration of 200 ppb. In fact, in the entire record of fish tissue chlordane concentration data, only three red shiner samples from San Diego Creek (out of 169 total samples for all species) exceeded 200 ppb, and these exceedances occurred in 1984 and 1987, prior to the ban on chlordane. Furthermore, if it is considered that red shiner are not generally consumed by humans—they are generally a bait fish—this means that data for typically-consumed fish species have never exceeded the revised OEHHA value during the period of record in the Newport Bay watershed, including samples before chlordane's ban. Finally, it is notable that the vast majority of the most recent fish tissue samples yielded chlordane concentrations below analytical detection limits. Together, these considerations suggest that a TMDL is not necessary for the purposes of reducing chlordane concentrations in fish species inhabiting the Newport Bay watershed.

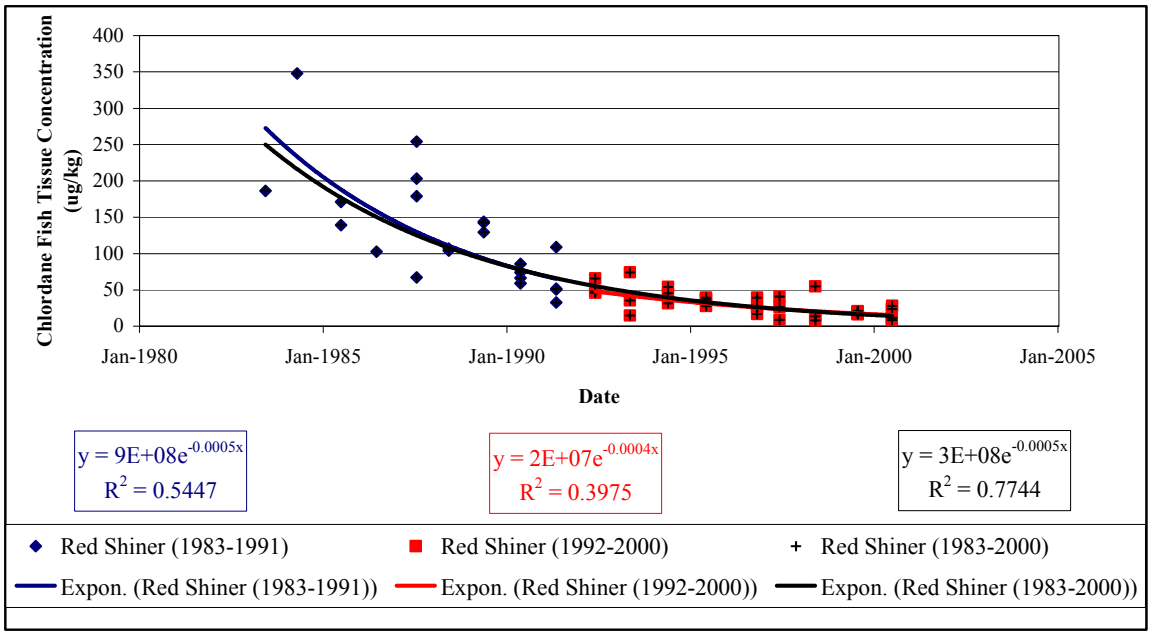
CHLORDANE CONCENTRATIONS

The following sections present available chlordane data for the watershed. Trends in chlordane concentrations—particularly in fish tissue and mussel tissue—are evident in data collected for almost 20 years.

FISH TISSUE

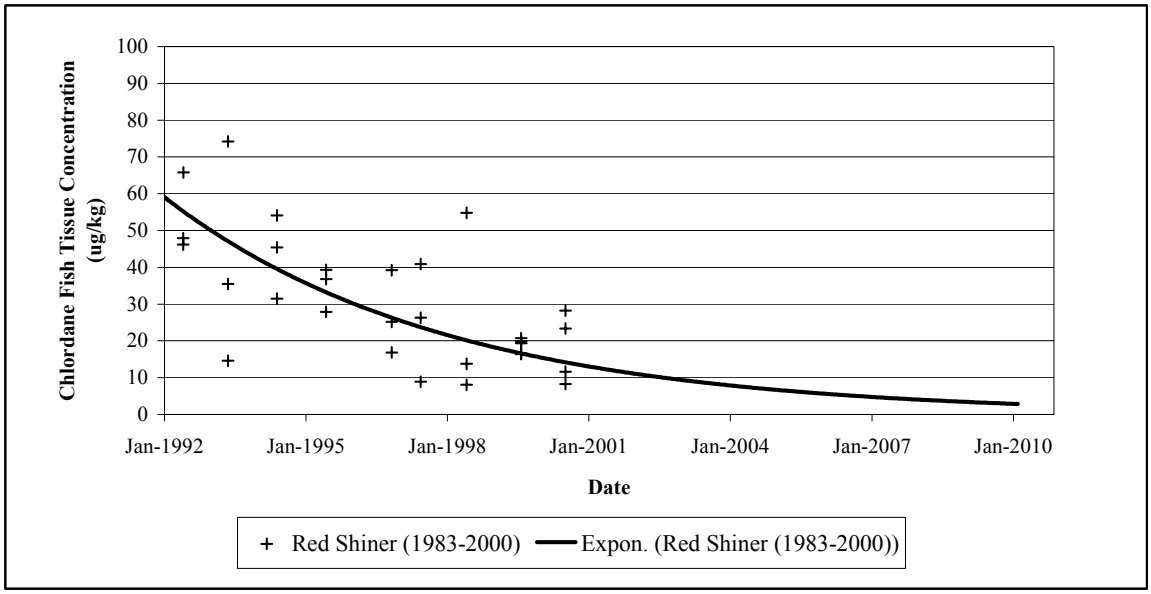
In the case of red shiner, chlordane tissue concentrations dating from 1983 show a substantial decline over time (see Figures 1 and 2). The primary statistical approach to establishing the declining trend has been to derive first-order decay constants using historical red shiner tissue data. The equations of these decay curves are indicated in Figures 1 and 2. Red shiner tissue concentrations may be taken as an indicator of chlordane concentrations in the watershed, as red shiners are local, short-lived species.

Figure 1. Chlordane concentrations in red shiner, San Diego Creek and Peters Canyon Wash (1983-2000)



Sources: California Toxic Substances Monitoring Program (TSMP), 1983-2000.

Figure 2. Chlordane concentrations in red shiner, San Diego Creek and Peters Canyon Wash, projected through 2010



Sources: California Toxic Substances Monitoring Program (TSMP), 1983-2000.

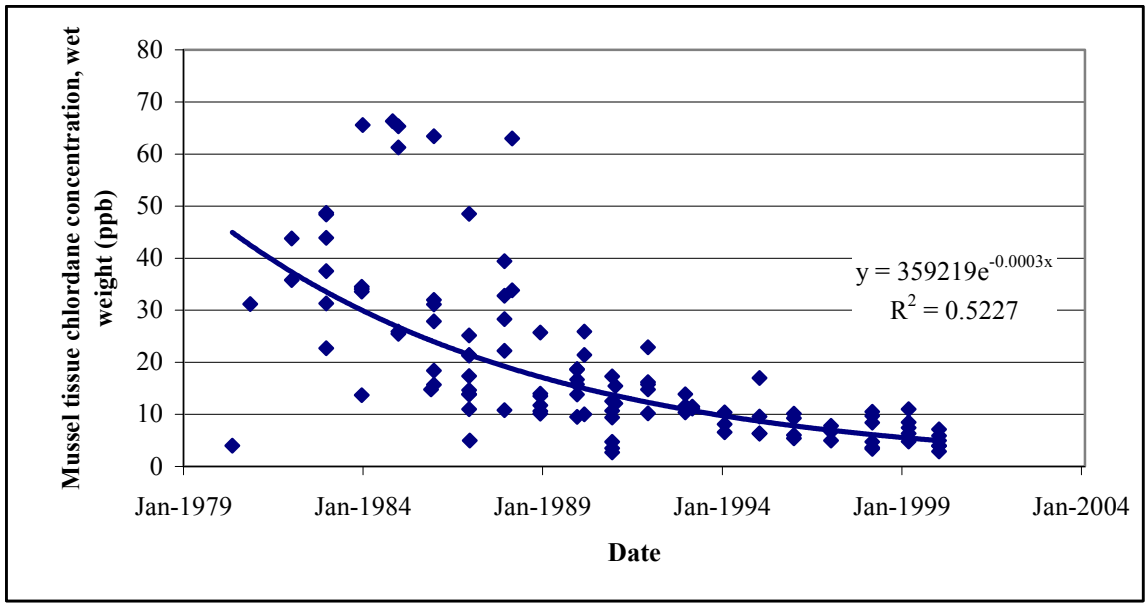
Two considerations suggest the robustness of the downward trend in red shiner tissue chlordane concentrations. First, when all red shiner data are considered together, a statistically strong (R^2 value of 0.774) downward trend in chlordane concentration is evident. Second, the statistical analysis that characterizes these trends is confirmed by splitting the data set for red shiners into two separate sets consisting of the first nine years of data (1983-1991) and the second nine years of data (1992-2000). Exponential decay curves fit to these two subsets of data revealed consistent downward trends during both periods. Therefore, the downward trend observed in the complete data set (1983-2000) is not simply the result of a temporally localized effect, but rather is an accurate portrayal of declines in chlordane concentrations over the entire period. The decay rate (-0.00046 per day or -0.17 per year) obtained for the full red shiner dataset (1983-2000) is equivalent to a half-life of 4.1 years.

As noted previously, only three (of 54) red shiner samples have ever exceeded the revised OEHHA value of 200 ppb, and these exceedances were from 1984 and 1987, prior to the ban of chlordane. Moreover, as previously noted, the vast majority of the most recent data (e.g., from 2000 to 2002) for other fish species have shown chlordane tissue concentrations below an analytical detection limit of 5 ppb.

MUSSEL TISSUE

Like red shiner data, mussel tissue data from Newport Bay show decreasing chlordane concentrations dating to 1980 (Figure 3). An exponential regression analysis of mussel data (by wet weight) for the period of record (1980-2000) showed a reasonably strong chlordane concentration decline rate in mussels (R^2 = 0.5227). A split analysis was also performed on mussel data for the two periods 1980 to 1989 and 1990 to 2000. This split analysis revealed that neither the earlier (R^2 = 0.1176) nor the later period (R^2 = 0.2968) evidence as statistically strong a decline as the complete period. Nevertheless, the most important conclusion is that when the entire mussel data set (1980-2000) is considered, it reflects a statistically significant decline in chlordane tissue concentrations, and is equivalent to a half-life for chlordane of 6.2 years (decay rate of -0.00031 per day or -0.11 per year).

Figure 3. Mussel chlordane concentration data, Newport Bay watershed



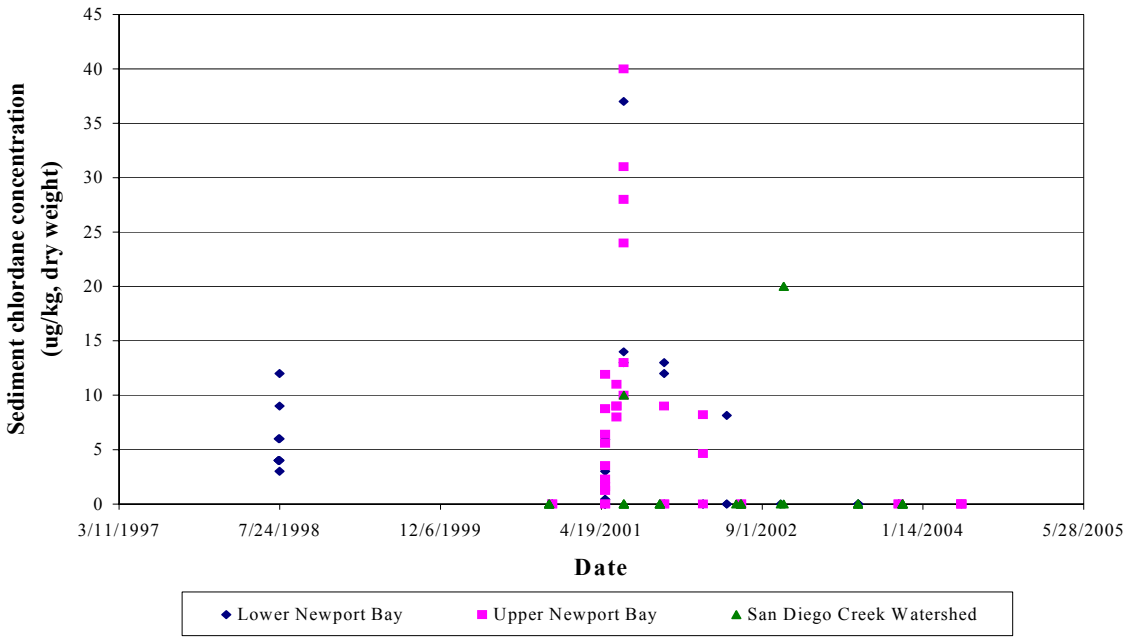
Source: State Mussel Watch (SMW) Program, 1980-2000.

BAY AND CREEK SEDIMENT

Bay and creek sediment chlordane concentration data from lower Newport Bay, upper Newport Bay, and the Newport Bay watershed are available for the period 1998 through 2004 (Figure 4). However, it is difficult to infer Bay-wide trends in sediment chlordane 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 and its watershed such that even samples taken in the same location at two different times may not represent the change in chlordane concentration for a specific quantity of sediment. Sediment movement results both from the natural flow of water and sediment in the Bay and its watershed, as well as from periodic dredging in the Bay, which has occurred in 1983, 1985, 1988, and 1999. Third, sediment concentrations in Newport Bay may be more indicative of chlordane loads from years or decades past, since Bay sediments are transported from the upper watershed in a highly variable, episodic manner. Thus, chlordane concentrations in Bay sediments reflect chlordane 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. For all these reasons, the available sediment data for Newport Bay are not the most reliable indicators of bioavailable chlordane concentration trends in the watershed. However, it is still notable that since 2002 all 22 Bay and creek sediment samples have exhibited chlordane concentrations below an analytical detection limits of 25 ppb (or ug/kg).

Figure 4. Sediment chlordane concentrations, Newport Bay watershed (1990-2002)



Sources: SCCWRP BIGHT 98 Survey (1998); Bay and Greenstein (2003); Bay, Greenstein, and Brown (2004); The Irvine Company (2000-2004); Santa Ana RWQCB (2002).

WATER

Only 12 chlordane water concentration data points were available for the Newport Bay watershed. Table 3 summarizes these data. All data were collected in 2001 or 2002, and none of the data points were above the analytical detection limit of 1 ng/l. The CTR human health regulatory threshold for chlordane in water is 0.00057 ug/L, or 0.57 ng/L.

Table 2. Chlordane concentrations in water, Newport Bay

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

* Detection limit = 1 ng/L.
Sources: Bay and Greenstein (2003); Bay, Greenstein, and Brown (2004).

AGRICULTURAL SOILS

Agricultural soil chlordane data were available for the Newport Bay watershed for the years 1989, 1990, 1995, and 2004. Samples from different years were taken in different locations since the purpose of sampling was generally to assess site conditions for planning and development, not to establish concentration trends over time in the watershed. Like toxaphene soil data, the majority of chlordane soil samples returned concentrations below detection limits. For example, in 2004, 230 samples were collected at a depth of zero to six inches, and 45 soil samples were collected at depths greater than 24 inches. All 2004 samples were below detection limits. From these data, it is clear that nearly all chlordane in agricultural soils within the watershed has been removed by volatilization (see discussion below), and the mass of chlordane in watershed soils is so low as to be uniformly below detection limits. Table 6 summarizes available agricultural soil chlordane concentrations and sample detection limits.

Table 3. Historical chlordane concentrations for agricultural soils in Newport Bay watershed

Year	0-6 inch Sample Depth			12-18 inch Sample Depth			>24 inch Sample Depth			Detection Limits (ppm)
	Average Observed Chlordane (ppm)	Max Observed Chlordane (ppm)	Sample Size (n)	Average Observed Chlordane (ppm)	Max Observed Chlordane (ppm)	Sample Size (n)	Average Observed Chlordane (ppm)	Max Observed Chlordane (ppm)	Sample Size (n)	
1989	0.24	0.24	3	ND	ND	1	0.16	0.19	5	0.25
1990	0.17	0.17	2	0.21	0.21	1	0.19	0.19	3	0.16
1995	0.05	0.06	19							0.06
2004	ND	ND	230				ND	ND	45	0.1

Sources: Byard, 1989; Byard, 1990; NMG Geotechnical, 1996; The Irvine Company, 2006.

NATURAL CHLORDANE REMOVAL RATES

The half-life of chlordane in soil is estimated at 350 days (or approximately 1 year), but can range from 37 days to 3500 days (approximately 10 years) (Hornsby et al., 1996). Chlordane is persistent in soils and volatilization is believed to be the only major removal mechanism (U.S. Dept. of Health and Human Services, 1994). Given that the use of chlordane was banned in 1988, and assuming a very conservative half-life of 10 years and no other loss mechanisms, the mass of chlordane in the agricultural soils of the Newport Bay watershed would have declined by almost 75% over the past 18 years due solely to volatilization. However, note that this number results when assuming the highest estimated half-life for chlordane. If the half-life is in fact one year, the decline would effectively be 100%. As noted above, the red shiner and mussel data can be used to estimate the half-life of chlordane in the watershed, as chlordane concentrations in their tissues are direct measures of their exposures. These data indicate a chlordane half-life in the watershed of between 4.1 years (obtained from red shiner data) and 6.2 years (obtained from mussel data), well within the range of published data on the half-life of chlordane in the environment.

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APPENDIX

SCIENTIFIC COMMENTARY ON THE
CHLORDANE TMDL FOR NEWPORT BAY

James L. Byard, Ph.D., D.A.B.T.
October 19, 2006

SCIENTIFIC COMMENTARY ON THE CHLORDANE TMDL FOR NEWPORT BAY

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

October 19, 2006

SUMMARY

The SARWQCB is claiming that chlordane is impairing benthic organisms in Newport Bay because sediment concentrations exceed the effects range median (ERM) and because chlordane is present in sediments that are toxic to benthic organisms. The ERM data set for chlordane is flawed, resulting in an ERM that greatly underestimates the toxicity threshold for benthic organisms due to chlordane in sediments. Contrary to statements in the SARWQCB draft Staff Report, the Bay Protection and Toxic Cleanup Program did <u>not</u> find a correlation between chlordane residues in sediment and inhibition of larval development in purple sea urchins or disruption of benthic communities in Newport Bay. The observed correlation of chlordane residues in sediment with amphipod toxicity in Newport Bay does not indicate that chlordane is responsible for this toxic effect. In fact, dose-response bioassays in amphipods and more sensitive aquatic species demonstrate that thresholds for toxicity are well above the highest levels of chlordane reported by SARWQCB for Newport Bay sediments.

INTRODUCTION

In their most recent draft report on the organochlorine total maximum daily loads (TMDLs), staff at the Santa Ana Regional Water Quality Control Board (SARWQCB) have decided to require a TMDL for chlordane in Newport Bay based on results of the triad of sediment chemistry, sediment toxicity to benthic organisms and disruption of benthic communities (Rose, 2006). This report will take a detailed look at the science underlying the triad to assess whether the results indicate impairment to benthic organisms in Newport Bay. The starting point is a discussion of the assays that make up the triad.

SEDIMENT TRIAD

The sediment triad for chlordane has three components. The first component is the concentration of chlordane in sediments from Newport Bay. The draft Organochlorine Staff Report compiled chlordane levels in sediments from several reports (Appendix A-2 to Rose, 2006). Levels of chlordane in sediments ranged from < 1 ppb to 55 ppb in the time period 1994-2004. The overall average appears to be about 10 ppb. Chlordane levels are declining in the watershed as a result of degradation, immobilization and past cancellation of all uses.

The second component in the triad is toxicity of sediments to benthic organisms. Two bioassays, used extensively in Newport Bay studies, are mortality to amphipods and sediment pore water inhibition of fertilization and larval development in purple sea urchins. Chlordane was negatively correlated with amphipod survival for 20 sampling sites in Newport Bay as shown in Table 24, which is reproduced from the Bay Protection and Toxic Cleanup Program (BPTCP) report (State Water Resources Control Board [SWRCB], 1998).

Table 24. Spearman Rank Correlation results for selected toxicants significantly correlated with amphipod toxicity (*Eohaustorius* and *Rhepoxynius*) results from specific water bodies.

Water Body	Chemical	N	Spearman Rho	Significance
Anaheim Bay	Selenium	22	-0.453	0.025
Huntington Harbor	Antimony	15	-0.757	0.001
Huntington Harbor	Lead	15	-0.629	0.01
Huntington Harbor	Tin	15	-0.842	0.0005
Newport Bay	Percent Fines	20	-0.649	0.0025
Newport Bay	TOC	20	-0.422	0.05
Newport Bay	Antimony	20	-0.458	0.025
Newport Bay	Chromium	20	-0.598	0.005
Newport Bay	Copper	20	-0.542	0.01
Newport Bay	Lead	20	-0.392	0.05
Newport Bay	Mercury	20	-0.444	0.05
Newport Bay	Nickel	20	-0.633	0.0025
Newport Bay	Tin	20	-0.495	0.025
Newport Bay	Zinc	20	-0.497	0.025
Newport Bay	Total Chlordane	20	-0.380	0.05
Newport Bay	Total PCB	20	-0.408	0.05

The correlation coefficient was -0.38, significant at the 0.05 level. Higher correlations were observed in the same samples with percent fines, total organic carbon and 8 metals. Chlordane was not correlated with inhibition of purple sea urchin fertilization or larval development in sediment pore water samples from Newport Bay (SWRCB, 1998). Chlordane was correlated with inhibition of purple sea urchin larval development in data collected in the entirety of Region 8, but the report did not indicate whether this correlation applied to Newport

Bay. Many of the sediment samples from Newport Bay contained levels of ammonia and sulfide that were toxic in the amphipod and purple sea urchin bioassays. Hence, toxicity measured in many of the sediments was due to ammonia and sulfide.

The third component of the triad is benthic community degradation. Crustaceans are generally the most sensitive species in the benthos and are given extra weight in the benthic index. The benthic index was not correlated with chlordane concentrations in sediments from Newport Bay as can be seen by the absence of chlordane in Table 29 reproduced below from the BPTCP report (SWRCB, 1998).

Table 29. Spearman Rank Correlation results for selected toxicants significantly correlated with benthic indices.

Water Body	Chemical	N	Rho	Significance
All	Cadmium	28	-0.329	0.05
All	Chromium	28	-0.392	0.025
All	Copper	28	-0.369	0.05
All	Iron	28	-0.431	0.025
All	Nickel	28	-0.383	0.025
All	p,p'-DDD	28	-0.332	0.05
All	p,p'DDE	28	-0.409	0.025
All	Total DDT	28	-0.322	0.05
All	Fines	36	-0.392	0.01
All	TOC	36	-0.362	0.025
Newport Bay	Chromium	20	-0.480	0.025
Newport Bay	Copper	20	-0.380	0.05
Newport Bay	Iron	20	-0.570	0.005
Newport Bay	Nickel	20	-0.459	0.025
Newport Bay	o,p'DDE	20	-0.407	0.05
Newport Bay	p,p'DDE	20	-0.481	0.025
Newport Bay	Fines	21	-0.638	0.0025

Additional correlations were performed between separate components of the benthic index and different toxicity test results. Analyses demonstrated significant relationships between normal urchin development at 25 and 50% porewater and total crustacean species ($p < 0.0025$ and $p < 0.01$, respectively).

Overall, chlordane concentrations in sediments from Newport Bay are weakly correlated with toxicity to organisms in the benthos. This correlation is not by itself an indication of causation. The toxicity correlated with chlordane could well be explained by the metals that also correlated with toxicity or by the hundreds of other chemicals that could be present but were not measured. Other investigators (Bay et al, 2004) have suggested that amphipod toxicity is associated with unmeasured organic compounds, possibly organophosphorus or pyrethroid insecticides. The authors of the BPTCP report (SWRCB, 1998) also noted that unknown chemicals could be causing sediment toxicity as stated in the segment reproduced below from the text of the report.

For example, our characterization of organic chemical contamination is constrained by the limited number of contaminants measured. Samples often contained un-identified organic compounds that were not further characterized due to the limited scope of the program; these might have contributed to the toxicity of the samples.

Let us look further at what is know about toxicity thresholds for chlordane to amphipods and other benthic organisms to gain an understanding as to whether the levels of chlodane in sediments in Newport Bay are high enough to cause toxicity to these organisms. Since the SWRCB (1998) compared chlordane to the effects range median (ERM) sediment quality guideline (Long and Morgan, 1990), let us begin by looking at the data underlying the ERM for chlordane.

CHLORDANE ERM

The ERM is calculated from 12 data points reproduced below as Table 33 from Long and Morgan (1990).

Table 33. Effects range-low and effects range-median values for chlordane and 12 concentrations used to determine these values arranged in ascending order.

Concentrations (ppb)	End Point
0.3	EP 99 percentile chronic marine
0.5	ER-L
0.6	EP 95 percentile chronic marine
2.0	San Francisco Bay, California, AET
3.5	San Francisco Bay, California, bioassay COA
3.5	San Francisco Bay, California, bioassay COA
4.1	San Francisco Bay, California, bioassay COA
6.0	ER-M
6.4	San Francisco Bay, California bioassay COA
17.4	EP freshwater lethal threshold
25.0	DuPage River, Illinois, benthos COA
31.3	Trinity River, Texas, bioassay COA
120.0	SSB LC50 for *C. septemspinosa*
<5800.0	SSB LC50 for *N. virens*

The first two data points (0.3 and 0.6 ppb chlordane) are derived by equilibrium partitioning from the chronic marine CTR standard for water to sediment using the lower 95th and 99th percentile of the variability in Koc values (Pavlou et al., 1987). These two data points are in error for two reasons. The first reason is that the Koc values are outdated since they are not based on the superior slow-stir technique (DeBruijn et al., 1989). Second, the high variability in the

outdated Koc values is not seen in those derived by the slow-stir method, precluding the necessity of using the lower 95th and 99th percentile of Koc values. Multiplying the Koc value for chlordane published by U.S. EPA (2002) and the SARWQCB (Rose, 2006) times the chronic marine CTR standard gives a single data point of 65 ppb chlordane in sediment containing 1 % organic carbon.

The last two data points in Table 33 are based on spiked sediment bioassays (McLeese and Metcalfe, 1980; McLeese et al., 1982). These two bioassays did not use valid techniques for assessing sediment toxicity. The first study was done with sand schrimp (*Crangon septemspinosa*) and involved adding an unreported amount of chlordane to a beaker, drying off the solvent, adding water and coarse sand (0.28 % organic matter; 0.5-2 mm diameter particles). The sand was allowed to settle and the shrimp were added. The authors concluded that chlordane dissolved in the water phase was the primary cause of toxicity. Chlordane bound to sediments contributed little to toxicity. For these reasons and the fact that the chlordane moved from water to sediment, this bioassay is primarily a water bioassay. The same is true of the second study (McLeese et al., 1982). The difference in the two studies being that in the second study the organism was a polychaete worm (*Nereis virens*) and the sediment was sandy silt with 2 % organic carbon.

In a true sediment bioassay, all of the chlordane would be picked up off of the glass by the sediment. The sediment would then be transferred to a clean container and equilibrated with water. Samples of water and sediment would be analyzed periodically until an equilibrium was reached. Only after equilibrium is reached, would the test organism be added. Long and Morgan (1990) misinterpreted these data points (McLeese and Metcalfe, 1980; McLeese et al., 1982) as spiked sediment bioassays, which they are not. If one applies equilibrium partitioning (using the Koc published by SARWQCB and U.S. EPA) to the water only bioassays reported by McLeese et al., the estimated sediment LC_{50}s are 9,000 ppb for the sand shrimp and 7,100,000 ppb for the polychaete worm.

The remaining 8 data points in the ERM table are based on the presence of chlordane (along with hundreds of other chemicals) in toxic sediments. None of these eight data points provide dose-response information. The flaws in the ERM data set make the ERM value useless as an indication of the threshold for benthic toxicity due to chlordane in sediments. The true threshold appears to be orders of magnitude greater than the ERM. This conclusion is further supported by other bioassay data.

CHLORDANE TOXICITY TO AQUATIC ORGANISMS

Cardwell et al. (1977) studied the chronic toxicity of chlordane in the amphipod, *Hyallela azteca*. Table 23 from the Cardwell et al. (1977) study is reproduced below.

TABLE 23. RELATIVE SURVIVAL AND GROWTH OF HYALLELA AZTECA EXPOSED TO TECHNICAL CHLORDANE

Parameter	Measured concentration of technical chlordane, µg/l					
	Control	1.4	2.6	5.3	11.5	20.5
Replicate 1						
No. survivors[a]	27	23	23	24	3	0
% survivors	108	92	92	96	12	0
Wet body weight, mg[b]	6.3 ±1.3	6.2 ±1.5	6.4 ±1.2	5.1 ±0.9	3.8 ±0.7	... ...
Dry weight, mg[b]	1.58	1.49	1.57	1.37	0.87	...
Replicate II						
No. survivors	22	25	25	24	9	0
% survivors	88	100	100	96	36	0
Wet body weight, mg[b]	7.5 ±1.3	5.8 ±1.3	5.8 ±1.6	5.5 ±1.6	5.3 ±1.0	... ...
Dry weight, mg[b]	1.92	1.55	1.53	1.35	1.33	...

[a] 25 individuals introduced initially per chamber.

[b] Average calculated weight per individual.

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In a 65 day study of mortality and weight gain, the no-observed-effect level (NOEL) appears to be 2.6 ppb in water. Using equilibrium partitioning to estimate the sediment concentration of chlordane required to reach 2.6 ppb in water, gives a sediment level at 1 % organic carbon of 42,172 ppb (2.6 ug/L x 1,622,000 L/kg x 0.01 ug organic carbon/ug sediment = 42,172 ug/kg).

The U.S. Fish and Wildlife Service (Eisler, 1990) reviewed the aquatic toxicity of chlordane. They reported an LC_{50} of 40 ppb in water for the amphipod *Gammarus fasciatus*. The equivalent LC_{50} for sediment at equilibrium would be 649,000 ppb. Other sensitive aquatic species include the pink shrimp (LC_{10} of 0.24 ppb in water), planarian (5 day NOEL of 0.2 ppb in water), and dungeness crab survival and molting (37 day NOEL of 0.015 ppb in water). The dungeness crab bioassay appears to be the most sensitive; the equilibrium NOEL in sediment calculated out to be at least 243 ppb chlordane.

ANALYSIS

Relying on the mere presence of chlordane along with hundreds of other chemicals in toxic sediments is an incomplete weight of evidence analysis. One must also consider the results of dose-response bioassays. Valid spiked sediment bioassays could not be found for chlordane. Therefore, one has to rely on spiked water bioassays and equilibrium partitioning to estimate the chlordane levels in sediments. The available aquatic toxicity bioassay data do not support an effect of chlordane on benthic organisms at levels of ~ 10 (< 1-55) ppb in sediment reported for Newport Bay. The lowest effect level exceeds 1,000 ppb and the NOEL in the most sensitive species is at least 243 ppb chlordane in sediment.

CONCLUSIONS

- The SARWQCB is claiming that chlordane in sediments is impairing benthic organisms in Newport Bay based on exceedance of the effects range median (ERM) and the presence of chlordane in sediments that are toxic to benthic organisms.

- The ERM data set for chlordane in sediments is flawed, resulting in an ERM that greatly underestimates the toxicity threshold for benthic organisms.

- Contrary to statements in the SARWQCB draft Staff Report, the Bay Protection and Toxic Cleanup Program did not find a correlation between chlordane residues in sediment and inhibition of larval development in purple sea urchins or disruption of benthic communities in Newport Bay.

- The finding of a correlation between chlordane concentrations in sediment and amphipod toxicity in Newport Bay does not by itself indicate causation; causation is not supported by dose-response bioassays. Thresholds for toxicity in amphipods and more sensitive aquatic species are well above the highest levels of chlordane reported by SARWQCB for Newport Bay sediments.

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APPENDIX 4

STEVEN BAY, SCCWRP (2006); PETER KOZELKA, EPA (2006); KEITH MARUYA, SCCWRP (2006); JAMES L. BYARD (2006); RONALD S. TJEERDEMA (2006)

COMMENTS ON THE PROPOSED TMDL FOR DDT, PCBs, CHLORDANE, AND TOXAPHENE, FOR THE NEWPORT BAY WATERSHED

Comments on Draft OC TMDL Report

October 12, 2006

Steven Bay, SCCWRP

Section 6.0 TMDLs, Load Allocations and Margin of Safety.
This section generally provides an adequate description of the assumptions involved in the calculation of the load allocations. The description of the calculation of the land use allocation percentages in Table 6-3 is not sufficient to allow the reader to follow the process. An explanation and justification for the discharge source ranking and relative ranking terms in the calculation is needed.

Section 7. Seasonal Variation and Critical Conditions
This section provides an adequate description of the key conditions affecting seasonal patterns in discharge into Newport Bay and I agree that an annual basis for implementing the TMDL is more appropriate than a daily basis. This section could be strengthened by including a figure that demonstrates the seasonal variation in contaminant loads entering the bay.

Section 8. Implementation.
The recommendations for a phased implementation plan, with substantial effort directed towards acquiring additional data for revision of the TMDL, is supported. There is much uncertainty associated with the proposed sediment targets for this TMDL and more information is needed in order to develop TMDL targets that are defensible from a scientific perspective. The special studies and monitoring that are listed are appropriate and will provide useful information for consideration when the TMDL is reopened.

There are two critical aspects of the TMDL that should be the focus of special studies: listing of specific chemicals as the cause of impairment and the calculation of sediment targets. The identification of DDTs, PCBs, and chlordane as causes of impairment were based on the interpretation of incomplete data using practices consistent with other agencies. While this approach may be reasonable to initiate the TMDL process, greater confidence in the determination of the cause is needed. Most problematic is the identification of chemicals based on the sediment toxicity and chemistry data cited in the document. The available data from Newport Bay indicates an association between sediment contamination and toxicity, which supports the need for a toxics TMDL, but it does not provide conclusive data to support the identification of DDT, PCB, or chlordane as the cause. The exceedance of SQGs and presence of statistical correlations for individual contaminants is not sufficient to establish the cause, as these findings could be due to the effects of other contaminants that were unmeasured or cross-correlated with the target chemicals. For example, recent data from other organizations (unpublished) indicates that pyrethroid pesticides may be a significant cause of sediment toxicity in upper Newport Bay.

Similarly, the use of low-range SQGs to establish the TMDL target concentrations is problematic on two levels. First, as is the case with their use for causal inference, these SQGs are based on

empirical associations using data from diverse locations and are not intended for use as cleanup targets. Second, the use of a toxicity-based SQG to establish a TMDL target for DDTs, where the impairment is related to indirect effects, is inappropriate.

Two types of special studies are needed to address the above deficiencies: First, additional sediment TIE studies are needed to determine the specific chemical contaminants responsible for sediment toxicity in Newport Bay. These studies should include newer methods developed for pesticides, attempt to fractionate the sediments to distinguish among different classes of organic chemicals, and should be applied to diverse locations within Newport Bay.

Second, directed studies to establish scientifically robust sediment targets for the chemicals are needed. For chemicals of concern for indirect effects, the target concentrations should be based on bioaccumulation data/models and appropriate risk assessment principles. The draft SQO case study conducted for Newport Bay provides a starting point in this process. The Regional Board should examine this study during Phase 1 and conduct any additional analyses needed to develop targets based on bioaccumulation and risk to humans or wildlife. This case study was conducted to explore the utility of the SQO framework in a realistic application, but the assumptions used for portions of the case study do not necessarily represent SWRCB policy. A similar effort is needed for chemicals that have been identified as a cause of impairment for direct effects to the benthos. Site-specific studies and data analyses are needed to determine the protective concentrations of contaminants that have been identified as the cause of direct effects. These studies should include field and laboratory experiments to determine the bioavailability of the contaminants and the dose-response relationship between the target sediment contaminants and biological effects for representative sediment types.

For Meeting 3 – October 12, 2006

here are some preliminary comments from my individual perspective as member of TAC for SDCk/NBay OCs TMDLs:

-selected numeric targets are consistent with long term goal of restoring all beneficial uses
-TMDLs should be expressed in both annual and daily mass units, thus 2 tables belong in section 6
-separate WLAs should be defined for MS4 (which equals urban runoff), Gen.Construction, Caltrans, Commercial Nurseries
-perhaps as you move downstream, the LA for channels & streams should decrease in %, since it is largest in freshwatershed and much smaller contribution in Lower Bay
-RB has discrection regarding decision on MOS
-support notion of existing loads (if lower than loading capacity) to be set as TMDL
-section 7, "extensive sediment transport only primarily occurs during the extreme storm events"
-Impl.Plan recognizes many on-going or planned studies to help understand impacts or pollutant transfer in these waters,
suggest Plan to include some description of easy to accomplish and reasonably cost-effective BMPs to occur in early stages, also include some monitoring guidelines that if still exceeded would trigger more aggressive (and possibly expensive) BMPs that would further reduce pollutant loads to the system.

EPA will submit official comments once the draftTMDL is complete.

Peter Kozelka

Organochlorine Compounds TMDLs for Newport Bay Watershed, Secs 6-8
Comments by Keith Maruya (SCCWRP Chemistry Dept.)

Sec 6.0

1. p.67, 4th para, 3rd sentence. Setting TMDLs at existing load when it is < loading capacity is a conservative decision, one that is not identified as such on p.70. For example in Table 6-1, setting the TMDL for PCBs @ 0.70 g/d (existing load) for San Diego Creek when estimated loading capacity is 5.30 g/d is extremely conservative. Setting TMDLs in these cases to a fixed percentage (20%? 50%?) of the loading capacity seems more reasonable and/or defensible.
2. p.69, Table 6-2. Proposed TMDL allocations for urban runoff (36%) relative to construction (28%) and agriculture (5%) may be appropriate for urban/industrial pollutants (e.g. PAH and PCBs). Stormwater concentrations (not loadings) of DDTs and chlordanes, however, may be highest in construction and agricultural site drainages (SCCWRP PRISM prelim data), perhaps suggesting that contaminant specific allocations may be warranted.
3. p.70, table 6-3. What is the *discharge source ranking* based on? Loading? Concentration or flow only?

Sec 7.0

None

Sec 8.0

1. p.80, study (6). The indirect effects study using Newport Bay as a case study is a good first step, but the complexities of this undertaking warrant a much larger effort in developing and validating a suitable framework, particularly since study outputs like sensitive wildlife receptors will be site specific and have a large impact on selection of sentinel species for TMDL/BMP compliance monitoring.
2. p.84, 1st para. Are there high quality data to indicate commercial nurseries are a significant source of legacy OCs? If not, does this need to be a special study?
3. p.84, 2nd para, (1). Although passive methods like SPME and SPMDs can measure very low dissolved concentrations, their utility is limited to relatively benign environments and/or certain flow regimes. One of the biggest "OC loadings" measurement challenges is stormwater particulates.

Sec 8.4 Monitoring Program Requirements

1. A consistent set of analytical measures that best represent the regulated OCs (e.g. total PCBs) in each matrix of interest (sediment, tissue, ambient water) needs to be established (if not already existing). These parameters should include a comprehensive and/or representative list of compounds (e.g. PCB congeners) to minimize the degree of unmeasured and/or unrecognized contributions.

2. Data quality requirements should be uniform and reflect the goals of the monitoring effort. Demonstration of sufficient analytical performance (detection limits, accuracy/precision) by those providing monitoring data should be required.
3. Because bioaccumulation/biomagnification (especially in top predators) probably will not respond immediately to load reductions, appropriate time scales and schedules for monitoring need to be established and supported by empirical data and/or modeling. Sampling frequency for sediments (source/sink) should be linked so that temporal comparisons with biota (receptor) could be made.
4. Sentinel species (particularly fish) for monitoring should be selected based on home range, life history (e.g. when are lipid levels high?) and size/age. If appropriate sentinels cannot be identified, transplanted or caged surrogates and/or biomimetic devices (SPME, SPMD) should be considered.

Additional Data Needs

I. Contaminant loadings and source (land use) contributions into the Upper Bay and San Diego Creek during wet weather

Rationale – High uncertainty in OC suspended particulate concerntrations in stormwater. Actual loadings may differ across OC classes for given land use category (e.g. agriculture vs. construction). Accurate apportionment of stormwater loads by land use will result in more efficient implementation of BMPs.

Implications – Inaccurate WLAs/LAs may result in unattainable TMDLs.

Approach – Synthesize results of PRISM studies (3) and (4) to determine if present WLAs/LAs are reasonable, with a focus on loading/concentrations differences among OCs. If substantially different, design special studies to better quantify these differences.

II. Toxaphene in San Diego Creek

Rationale -- Toxaphene listing solely due to tissue levels with little/no tenable link to bedded sediments (most nondetect). This is problematic since TMDL targets are strongly focused on sediment. Because of its complexity, there is an inherent large degree of uncertainty with analysis of toxaphene in environmental samples using standard regulatory methods (e.g. EPA Method 8081a), especially at low levels.

Implications -- Incorrect determination of toxaphene could lead to false positives or negatives, resulting in an incorrect impairment listing.

Approach – analyze existing database and methods used. If suspect, perform confirmation of toxaphene residues in San Diego Creek (and select Bay) samples (sediment, tissues) using GC-ECNI-MS or MS/MS. Possible to use properly archived samples, less so on organic extracts (no pre QA). Best to do on freshly collected samples.

JAMES L. BYARD, PH.D., D.A.B.T.

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October 10, 2006

Dr. Kathy Rose
Santa Ana Regional Water Quality Control Board
3737 Main Street
Riverside, California 92501

Re: Second set of detailed comments on the draft Organochlorine TMDL Staff Report.

Dear Kathy:

Thank you again for having me as a member of the TAC and for providing an opportunity to make informal comments on the draft Staff Report. My previous two comment letters provided both general and detailed comments on the problem statement and numeric target chapters.

At this point, you are expecting comments on the more recently completed parts of the draft Staff Report, including the implementation plan. However, my comments will remain focused on the numeric targets. The reason is that the targets in the draft Staff Report are outdated, misinterpreted or in error. To discuss other aspects of the draft Staff Report - including the implementation plan - when the targets are wrong, would be inappropriate. Some of the problems with the targets have been discussed in my two previous comment letters. In addition, new information has been learned about the targets since the first two comment letters were written. This additional information shows clearly that the proposed numerical targets were misinterpreted and/or just plain wrong. A detailed presentation and analysis of this new information will be provided to the SARWQCB within a few weeks. This letter will summarize some of the important new findings.

The draft Staff Report has adopted the U.S. EPA Region IX toxaphene sediment target of 0.1 ppb for the San Diego Creek Watershed. The target is cited as a New York State Department of Conservation freshwater sediment screening level. The screening level is based on equilibrium partitioning, a method proposed by stakeholders and refuted by SARWQCB (pages 32-34 of the draft Staff Report). Apparently, SARWQCB is unaware of the method used by New York State to derive the sediment screening level

used in the toxaphene TMDL. The sediment target of 0.1 ppb would require a 99 % reduction in both the toxaphene load and in the sediment load in the Watershed. New York State used a K_{ow} for toxaphene that is outdated and is 158-fold lower than the K_{ow} published by both the U.S. EPA and SARWQCB in their respective TMDL reports. Using the updated K_{ow} in the New York State method for determining the sediment screening level, yields a sediment target of 15.8 ppb. This target level results in the loading capacity for San Diego Creek to exceed the existing load as determined by SARWQCB (Table 4-7 in the draft Staff Report).

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 flawed 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 have been considerably 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 the total DDT 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. The total DDT TELs are so flawed that they should not be used for any purpose.

The U.S. EPA, SWQCB and SARWQCB have misinterpreted the OEHHA fish guidance for total DDT to claim impairment of sport fishing in Newport Bay. The OEHHA guidance states that the 100 ppb level is an internal screening level used to indicate when additional risk assessment might be needed. The OEHHA authors caution against using the 100 ppb level as a standard. The objective of the OEHHA guidance , as stated in the 1991 base document, was to achieve a potential cancer risk of less than 1/10,000 at each site. This objective is met in Newport Bay. OEHHA has not issued a sport fish advisory for 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, an action level that is still in effect today. OEHHA has recently 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. DDTs are not impairing sport fishing in Newport Bay.

The SWQCB does not recommend the use of TELs or the NAS marine fish guidance of 50 ppb as targets for TMDLs, yet the SARWQCB insists upon using these outdated and scientifically flawed guidance numbers. Ignoring the potency of chlordane to benthic organisms is ignoring a critical line of evidence in the weight of evidence approach to using sediment triad data. Ignoring this line of evidence and concluding that chlordane is impacting benthic organisms merely on occurrence (along with hundreds of other chemicals) in toxic sediments is unscientific. Dose-response bioassays in benthic organisms indicate that the toxicity thresholds for chlordane are several orders of magnitude higher than chlordane levels measured in sediments from Newport Bay.

The failure to understand and use the science behind the proposed targets has resulted in a proposed rule making that is incorrect and, therefore, unfair. If the TMDL targets are adopted as they now stand, resources would be wasted on a nonproblem, resources that could be used to address known toxicity in the Watershed.

I would be happy to respond to any comments concerning the above.

Sincerely,

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

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October 10, 2006

Dr. Kathy Rose
Santa Ana Regional Water Quality Control Board
3737 Main Street
Riverside, CA 92501

Re: Comments on the OC TMDL Staff Report – Sections 4 through 8

Dear Kathy:

Thanks again for the opportunity to both serve on the TAC and provide input on the OC TMDL. As an academic I try to challenge preconceived notions that are out of step and encourage change when either new information or improved understanding comes about. While many of the legacy pesticides have been convenient pariahs for many years, it can take unusual courage to recognize current realities and use current science to effectively manage them. In the case of OCs in the Newport Bay Region, *current* science indicates that they are no longer a toxicological concern.

I hope you find my comments helpful, and please do not hesitate to contact me if you would like additional information.

Best regards,

Ronald S. Tjeerdema, Ph.D.
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Organochlorine Compound TMDLs: Draft Staff Report – Sections 4 through 8

R. S. Tjeerdema
Review and Comments

October 10, 2006

A. Overall Document

Similar to the first three sections, the remaining sections are well organized and written, and clearly summarize the information considered in establishing TMDLs for the various organochlorine compounds in San Diego Creek, Newport Bay, and the Rhine Channel. However, I still have a number of concerns in regards to the rationale and approach utilized for developing the TMDLs, including:

a. The document contains an inappropriate reliance on historical data and fails to undertake a residue trends analysis which would accurately characterize current (or future) chemical conditions.
b. Implementation measures will be required by the agency to address compounds that are not of toxicological concern and which, therefore, are not scientifically justified.
c. Because receiving waters are in a state of attainment, there is real potential for unnecessary agency, municipal, and private resource expenditures – which could potentially be put to better use addressing real problems elsewhere.
d. The need of certain TMDLs for San Diego Creek and its tributaries is not clearly justified – in fact, good science argues against them.

Below are more specific comments.

B. Section 4.0 – Source Analysis and Existing Loads

Page 1, paragraph 6, line 1: Water solubility is *always* inversely proportional to *K*ow – water concentration constitutes the denominator of the *K*ow equation.

Page 3, Table 4.1: Why not report measured *K*oc values, as they are inherently more representative (and accurate) than those estimated from regressions involving *K*ow? In 2002 the USEPA insisted on using a *K*ow value to estimate the *K*oc for DDT, even when informed that measured *K*oc values for DDT exist in the scientific literature.

Page 9, Figure 4.3: Use of DDT trends data (1995 – 2004) to calculate mean values for the 10-year time period is an inappropriate use of the data. It completely obliterates the clearly declining trends, but also is misleading (as inaccurately summarized by the statement of page 10, paragraph 1, line 3). The appropriate approach would have been to semi-logarithmically graph the declining concentration trends for the 10-y time period and regress them to estimate the concentrations present in the region today. The TMDLs should be based on current conditions (or those estimated to exist at implementation), not those that may have occurred years ago.

Page 14, paragraphs 2 and 3; page 15, Table 4.4: The information presented is nearly 10 years old, and its validity in representing current conditions is highly suspect. For instance, the statement "...the levels shown in Table 4.4 reflect a reservoir of OC pollutants that *likely* exists

at these sites..." is speculative and unsupported by data. There appears to be a real resistance to estimate current or future chemical concentrations in the region – as they may not support the development of TMDLs. This is a clear example of not applying the best science to the project.

Page 16, paragraph 1, last two sentences; Table 4.5: Normally I would support the idea of confirming data by the use of potentially more sensitive methods. In this case the nurseries clearly used an established EPA method, which they were most likely directed to do and thus should be acceptable. However, in this case it appears that since the data do not support the establishment of TMDLs, doubt is being cast upon its validity.

Page 21, paragraph 2, line 2: As stated, "Existing loads were estimated using the same process as was used by USEPA (2002). That procedure utilized the geometric mean of measured tissue concentrations in *Cyprinella lutrensis* (red shiner) collected June 9, 1998..." As stated, existing loads were based on decade-old data? In reality, they represent 1998 loads, not existing ones.

C. Section 5.0 – Linkage Analysis and Loading Capacity

Page 4, paragraph 1, line 12: For accuracy in determining the current sediment deposition patterns in Newport Bay, the RMA model should be re-run, as suggested in the document. The substitution of "professional judgment" due to a lack of time is not appropriate when considering the resources that may be allocated based upon the results of the TMDL process.

D. Section 6.0 – TMDLs, Load Allocations, and Margin of Safety

Page 67, paragraph 4; Table 6.1: Based upon my assessment above regarding the estimation of current chemical concentrations, I would strongly question the use of "existing" when describing loads. Current loads have not been determined by estimating current concentrations.

Page 69, list of conservative assumptions and uncertainties: By not regressing chemical data, one *very* conservative ("worst case") assumption that should be mentioned is that OC concentrations in the region have not declined significantly in the past decade.

Page 71, second bullet: Most likely, by using outdated information the sediment loads and deposition patterns have been overestimated (please see comment under Section 5.0, above). Failure to estimate both current (or future) sediment loads and OC concentrations place serious doubt as to the accuracy and usefulness of the TMDLs.

Page 71, fifth bullet: Use of a limited data set (red shiner collected in 1998) instead of regressing ten years of trend data represents poor science, not estimation of a "worst case scenario."

E. Section 7.0 – Seasonal Variation and Critical Conditions

No specific comments.

F. Section 8.0 – Proposed Implementation Plan

Page 77, paragraph 3: The phased implementation approach seems reasonable for the benefits as stated in the paragraph. In particular, and as described later in the section, it provides time for

additional monitoring and other assessments which may lead to modification of the TMDLs over time as well as the delisting of certain water body-pollutant combinations. However, if the proposed TMDL implementation is scheduled to occur by 2015 – nearly ten years from now – for greatest accuracy the TMDLs should be developed using the conditions estimated to exist in 2015. In all likelihood they would not support TMDL development or implementation.

G. Summary:

My main concern with the development and implementation of TMDLs for OCs such as DDT is that their use is rationalized by historical conditions, and not what they currently are. Again, long-term trends clearly indicate that OCs are in decline in the region, as would be anticipated as:

a. They have not been used for many years (DDT use was discontinued in 1972).
b. They are subject to biodegradation.
c. Agricultural lands in the region continue to be converted to other uses, many of which sequester residues on site (also reducing sediment transport).

As mentioned above, the mathematical regression of temporal data can with reasonable accuracy estimate current environmental concentrations. Better yet, with an implementation target of 2015, they should be regressed to estimate conditions that would be operable then. In all likelihood the TMDLs would not be needed. Without such regression analysis there is a real risk that the implementation of TMDLs will be erroneously based on historical conditions in the watershed.

APPENDIX 5

WRC CONSULTING SERVICES (2006)

HISTORICAL SEDIMENT LOAD EXAMINATION, SAN DIEGO CREEK WATERSHED

Historical Sediment Load Examination
San Diego Creek Watershed

Prepared for:

County of Orange
Resources and Development Management Department

Prepared by:

WRC Consulting Services
Santa Ana, California

June 28, 2006

Table of Contents

Introduction

The sediment monitoring program within the San Diego Creek watershed has provided a wealth of data concerning the quantities of sediment load that runoff flows have historically carried. These data have been measured as the watershed has undergone change – changes associated with development, and changes associated with establishment of erosion management facilities and strategies. The purpose of this study is to determine the effects these watershed changes have had on the rate of sediment transport within the watershed.

Data Presentation – Long Term Monitoring Locations

Data is available for the three long term monitoring stations from the 1982/83 monitoring period through the 2004/05 monitoring period. Annual runoff volumes and estimated sediment loads over this period are summarized in Table 1.

Table 1 Historical Data Summary, Long Term Monitoring Locations

	Annual Flow in acre-feet			Annual Sediment Discharge in tons		
Year	San Diego Creek at Campus Drive	Peters Canyon at Barranca Parkway	San Diego Creek at Culver Drive	San Diego Creek at Campus Drive	Peters Canyon at Barranca Parkway	San Diego Creek at Culver Drive
1983	58,952	24,323	16,504	534,035	178,507	158,651
1984	29,425	15,774	5,657	64,455	26,897	24,599
1985	26,987	11,831	5,879	32,236	18,331	27,904
1986	29,746	12,453	6,654	37,760	NR	NR
1987	21,423	12,112	NR	20,060	9,800	NR
1988	22,089	10,797	3,751	34,186	21,037	12,408
1989	17,359	10,489	3,666	19,810	16,264	13,163
1990	19,154	NR	NR	24,855	NR	NR
1991	28,935	NR	NR	83,924	NR	NR
1992	37,186	14,697	11,676	173,212	47,845	103,516
1993	62,510	29,170	22,140	355,208	116,283	228,309
1994	20,000	9,910	4,190	33,027	15,075	12,705
1995	61,182	19,493	NR	347,579	82,633	NR
1996	23,501	8,453	6,323	49,438	8,716	32,064
1997	33,946	13,392	10,240	92,181	30,529	68,266
1998	92,345	34,072	35,555	611,461	179,579	404,085
1999	17,334	8,703	5,499	16,439	6,908	11,957
2000	17,780	7,400	6,960	28,864	13,639	26,205
2001	27,320	11,180	10,280	75,686	33,301	49,592
2002	10,610	5,520	2,630	5,640	1,392	3,354
2003	30,090	13,910	9,230	64,740	31,835	19,039
2004	18,690	7,380	7,020	30,464	15,265	18,065
2005	75,860	27,040	27,790	165,810	41,108	91,862
1983-99 average	35,416	15,711	10,595	148,816	54,172	91,469
2000-05 average	30,058	12,072	10,652	61,867	22,757	34,686
ratio of 2000-05 average to 1983-99 average	84.9%	76.8%	100.5%	41.6%	42.0%	37.9%
ratio of 2005 value to 1983-99 average	2.14	1.72	2.62	1.11	0.76	1.00

As indicated above, the 2005 period (July 2004-June 2005) had above normal runoff at all three long term monitoring locations, with annual runoff totals equaling 1.72 to 2.62 times the average experienced over the pre-TMDL period (1983-1999). However, the sediment load estimated at these three locations was less than would be expected based on relative runoff rates, with total loads of only 0.76 to 1.11 times the average annual load estimated over the pre-TMDL period. The data in Table 1 also indicate that the average annual flows during the 2000-2005 period were comparable to the average

annual flows during the 1983-1999 period. The average annual sediment load during the 2000-2005 period, however, was less than half as large as the average annual sediment load during the 1983-1999 period. These data indicate that sediment concentrations in the runoff flow in the recent period are less than had been observed in the past. This conclusion differs from the conclusions derived in a December 2003 examination of the available data (see Reference 1). The available data and factors contributing to this decline in sediment concentration are examined in further detail in a later section of this report.

Data Presentation – Recently Added Monitoring Locations

At the remaining monitoring locations within the San Diego Creek watershed the period of available record is less extensive. Measured and approximated data for these locations are summarized in Table 2.

Table 2 Annual Runoff and Sediment Discharge Summary, New Monitoring Locations

	Annual Flow in acre-feet					Annual Sediment Discharge in tons				
Year	Sand Canyon at University Dive	Bonita Canyon at MacArthur Blvd	Santa Ana Delhi at Irvine Blvd.	Marshburn Channel	Agua Chinon	Sand Canyon at University Dive	Bonita Canyon at MacArthur Blvd	Santa Ana Delhi at Irvine Blvd.	Marshburn Channel	Agua Chinon
2000	548	2,457	3,570	162	85	15	212	406	52	731
2001	764	2,773	6,690	309	166	24	316	983	138	1,982
2002	385	2,210	4,080	59	27	9	157	246	11	128
2003	827	2,539	3,236	384	215	27	356	896	178	2,570
2004	310	960	3,690	225	25	19	61	464	97	190
2005	3,600	5,340	11,140	1,060	540	493	934	1,889	641	10,856
2000-05 average	1,072	2,713	5,401	367	176	98	339	814	186	2,743

Table 2 contains many approximations. Data not provided directly through the County's monitoring reports or the data summarization provided in the 2003 URS report (see Ref. 1) were extrapolated using available measured sediment transport data and trends indicated by these data. Recent sediment transport measurements for the Marshburn Channel, Agua Chinon, Bonita Canyon and Sand Canyon monitoring locations are shown in Figures 1 through 4. The regression equations indicated in these figures were applied to the available flow record to estimate sediment transport quantities during the unmeasured period. For the Agua Chinon and Marshburn Channel locations, flows for the 2000-2003 were approximated using relations developed through comparison of same day flows on San Diego Creek during the 2004/05 monitoring period. These relationships are shown in Figures 5 and 6.

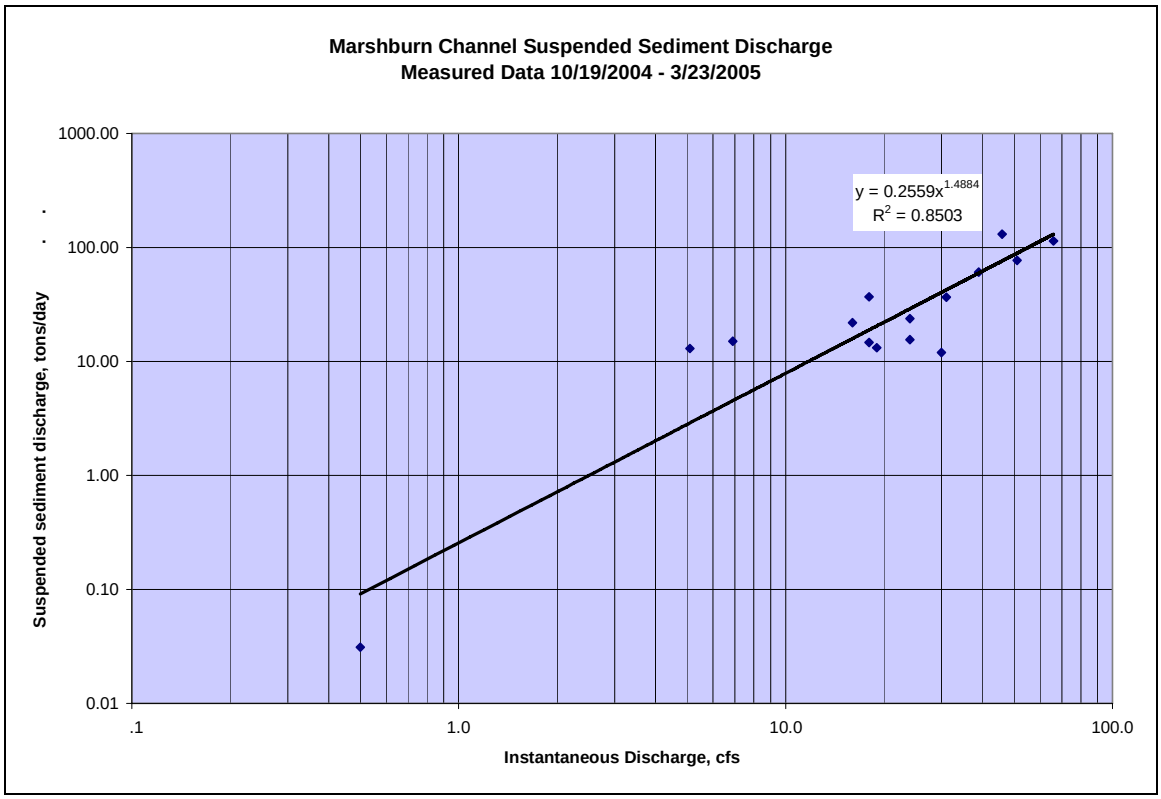

Figure 1

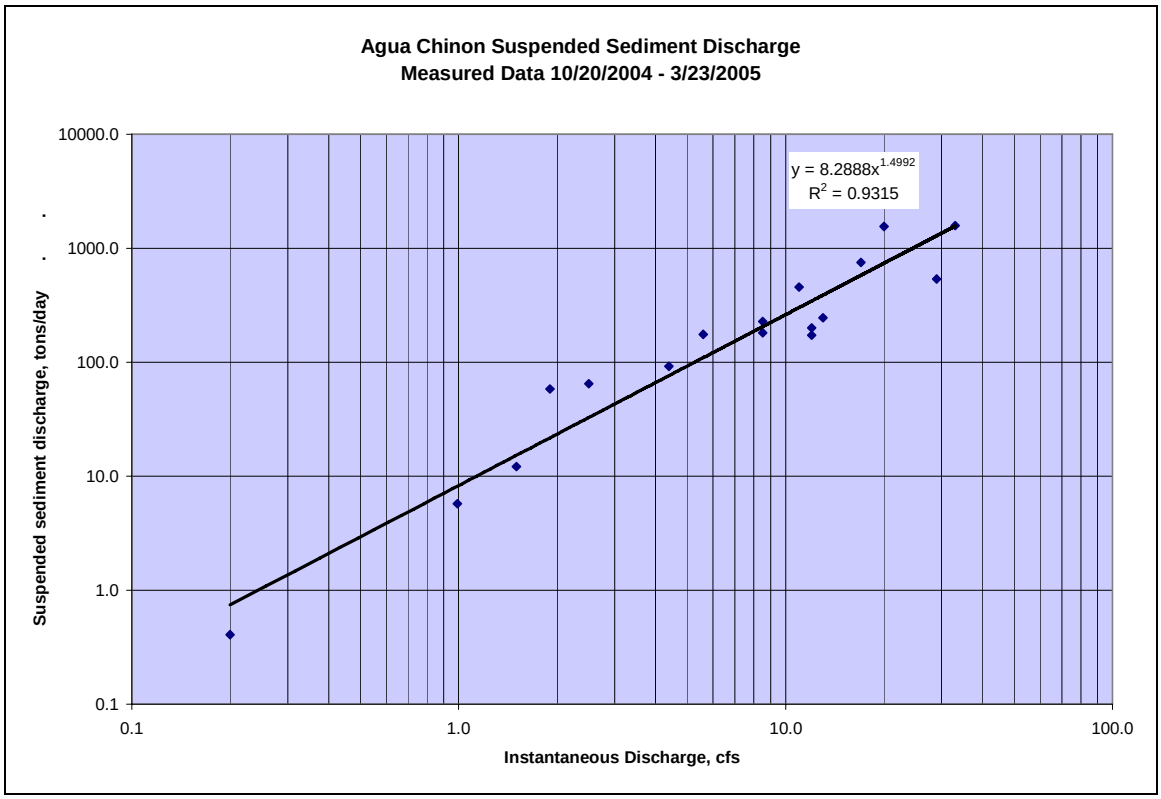

Figure 2

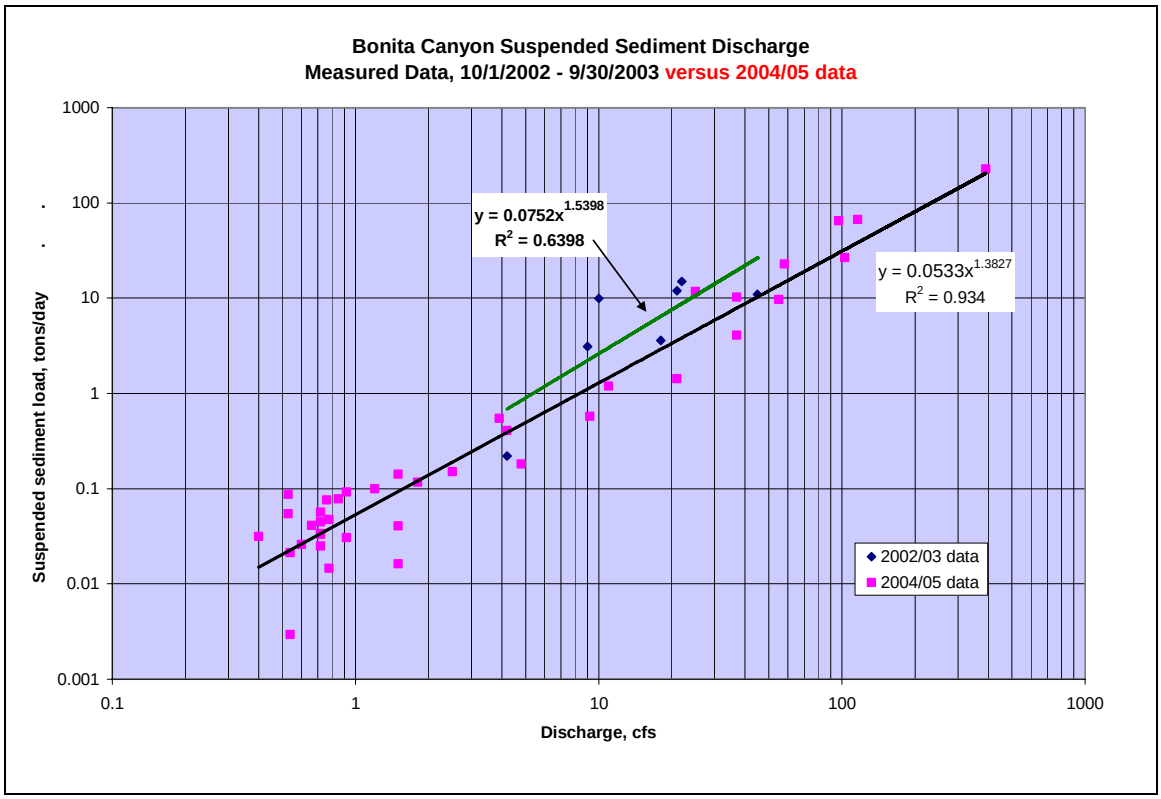

Figure 3

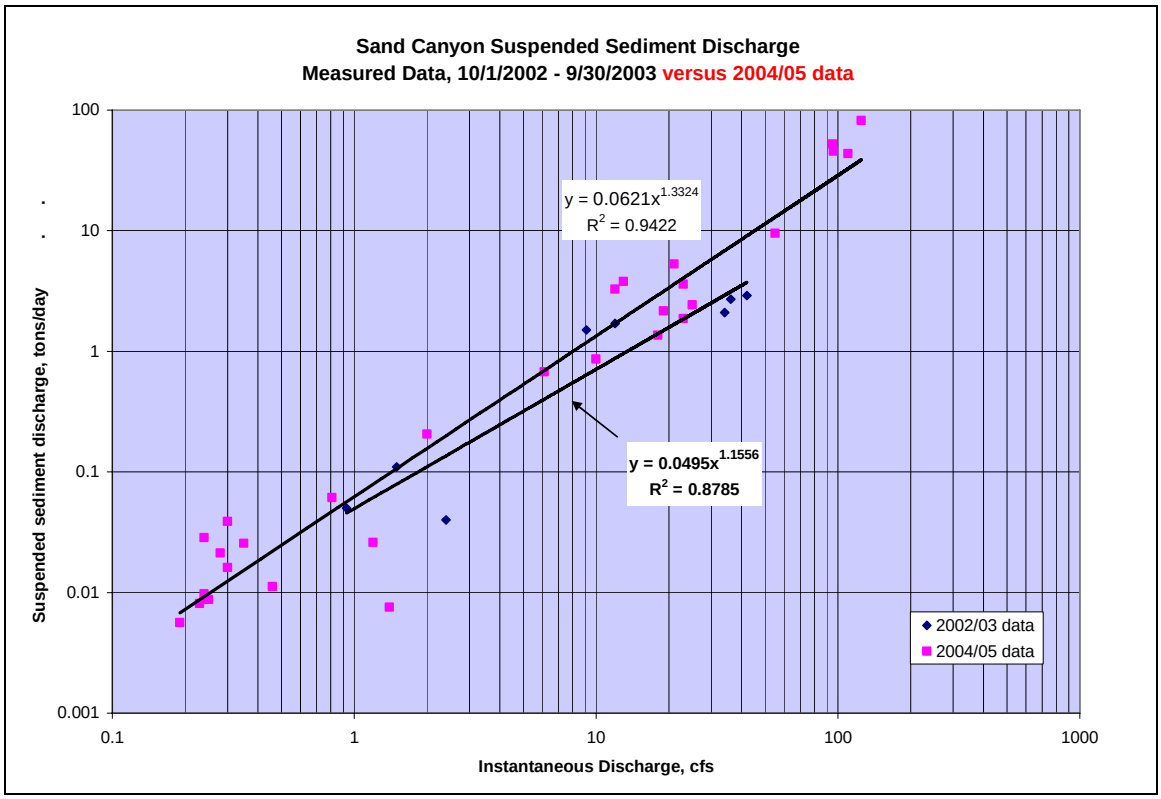

Figure 4

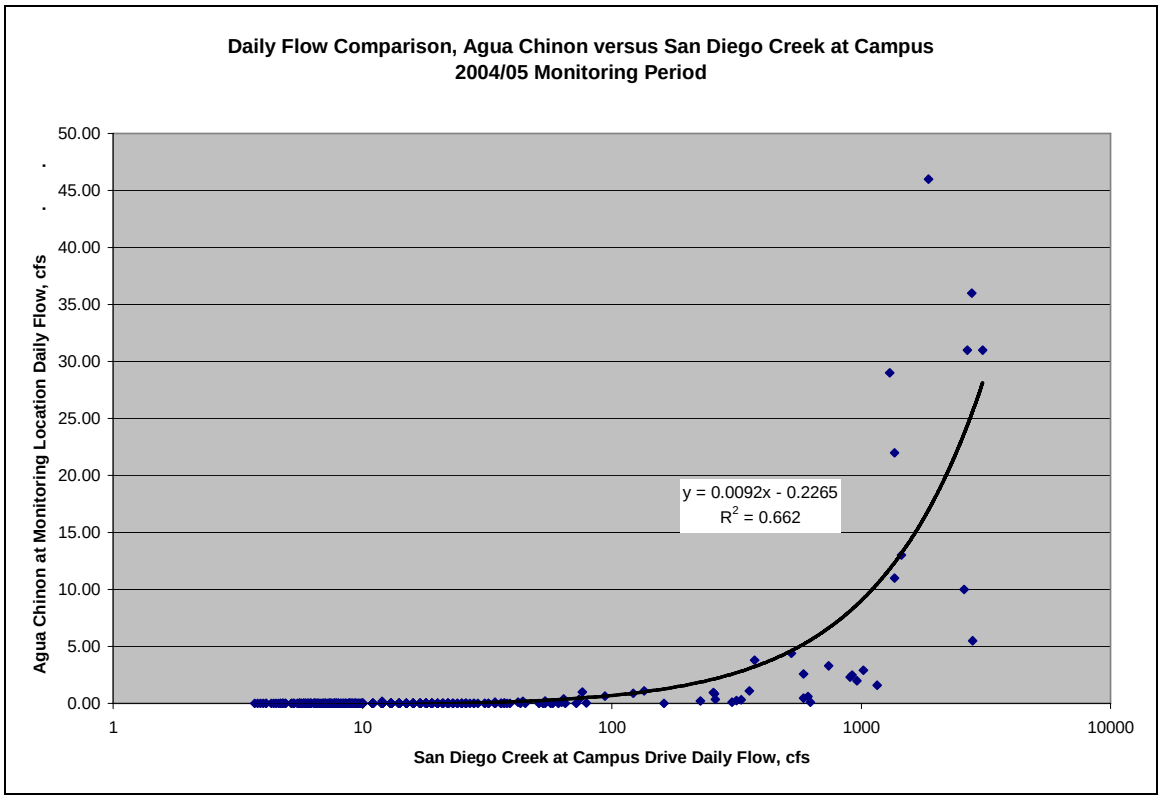

Figure 5

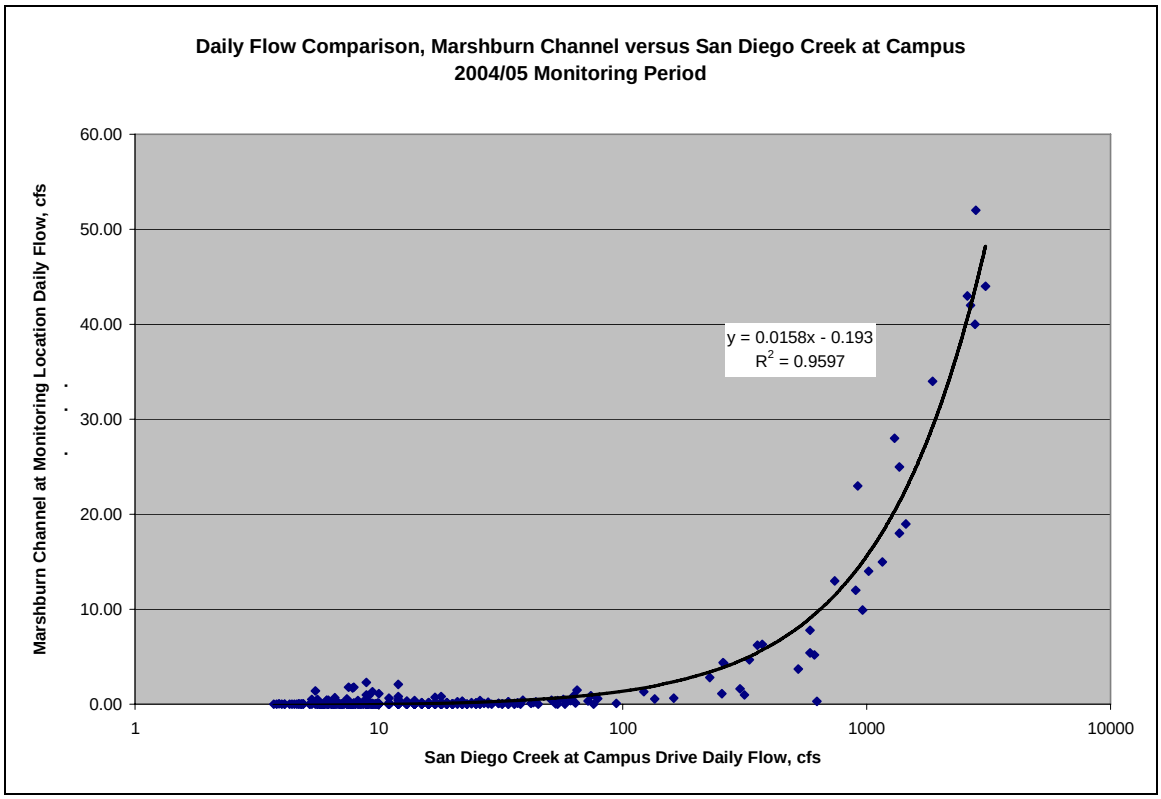

Figure 6

Comparison with Expected Loading

Tables 1 and 2 include average annual sediment loading quantities for each of the monitoring locations, corresponding to the post TMDL period (2000-2005). As mentioned above, the 2000-2005 experienced average annual runoff quantities that were comparable to the average experienced over the 1983-1999 period (76.8 percent to 100.5 percent of the longer period average, see Table 1). Thus, the average sediment loadings from the 2000-2005 period provide an initial estimate of the average annual loading for the current condition of the watershed. In Table 3, these quantities are compared to the expected average annual loadings that were used in developing the sediment monitoring plan for the watershed (see Reference 3). As indicated in this Table, the recent period sediment loads are significantly lower than the expected (pre-TMDL) quantities.

Table 3

Measured versus Expected Average Annual Sediment Load at Monitoring Locations within the Study Watershed

Location	A 2000-2005 average annual tons	B expected (pre-TMDL) average annual tons	A/B measured/expected
San Diego Creek at Campus Drive	61,867	104,400	59.3%
Peters Canyon at Barranca Parkway	22,757	34,600	65.8%
San Diego Creek at Culver Drive	34,686	56,100	61.8%
Sand Canyon at University Dive	98	8,195	1.2%
Bonita Canyon at MacArthur Blvd	339	77,332	0.4%
Santa Ana Delhi at Irvine Blvd.	814	2,022	40.3%
Marshburn Channel	186	1,715	10.9%
Agua Chinon	2,743	8,106	33.8%

The sediment loadings measured during the 2004/05 monitoring period are also compared to the pre-TMDL average annual loadings in Figure 7. The watershed experienced the second highest recorded rainfall totals during the 2004/05 monitoring period, and runoff totals 1.72 to 2.65 times the average annual rates of the 1983-99 period (see Table 1). As shown in Table 3 and Figure 7, loadings from Sand Canyon, Bonita Canyon, Santa Ana Delhi Channel and Marshburn Channel have been significantly lower than expected. Agua Chinon appears low on an average annual basis (Table 3), but was in line with the other major channels (Peters Canyon and San Diego Creek) in its 2004/05 performance.

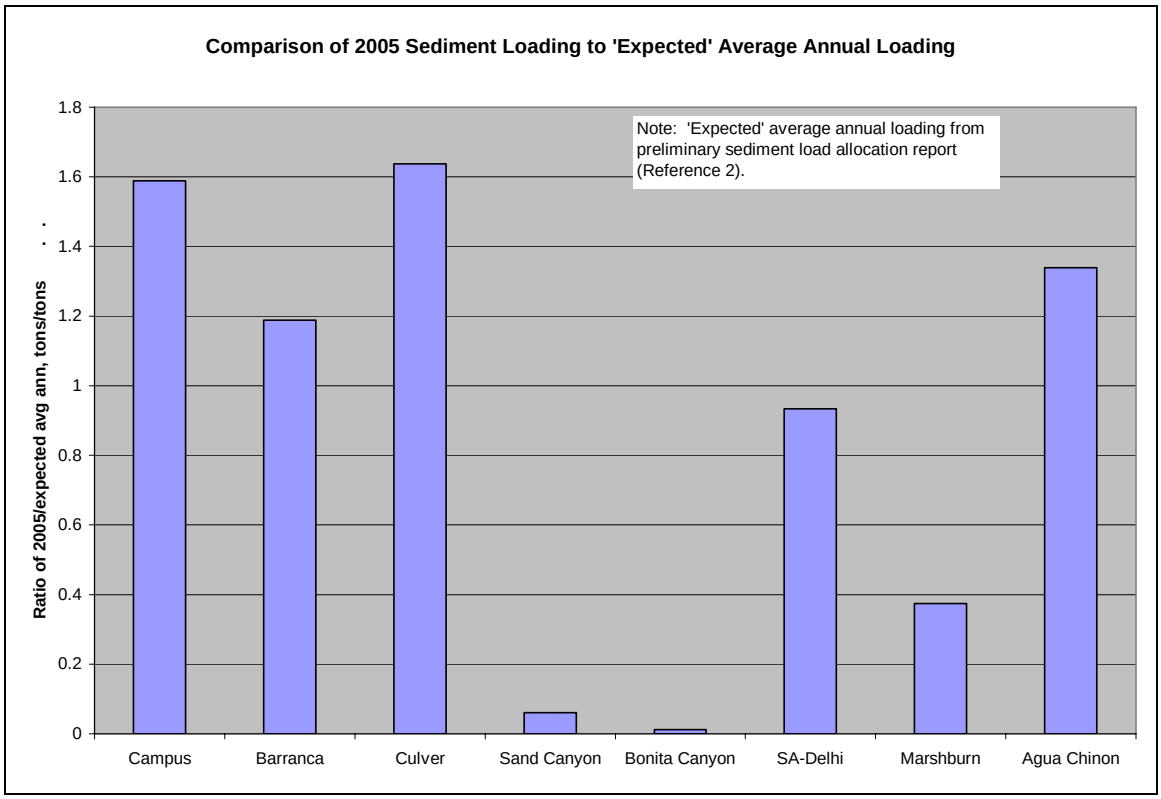

Figure 7

Discussion of Recent Results

The recent data indicates that sediment concentrations in recent flows are lower than had been estimated previously. This finding is illustrated in Figures 8 through 10, which compare average sediment concentrations to annual runoff volume for the period of record at the Campus, Culver, and Barranca monitoring locations. The 2005 data highlighted in these figures plot significantly off the trend of the prior years data, including the data from recent years (2000-2004). Potential reasons for the departures evident with the recent data are discussed below.

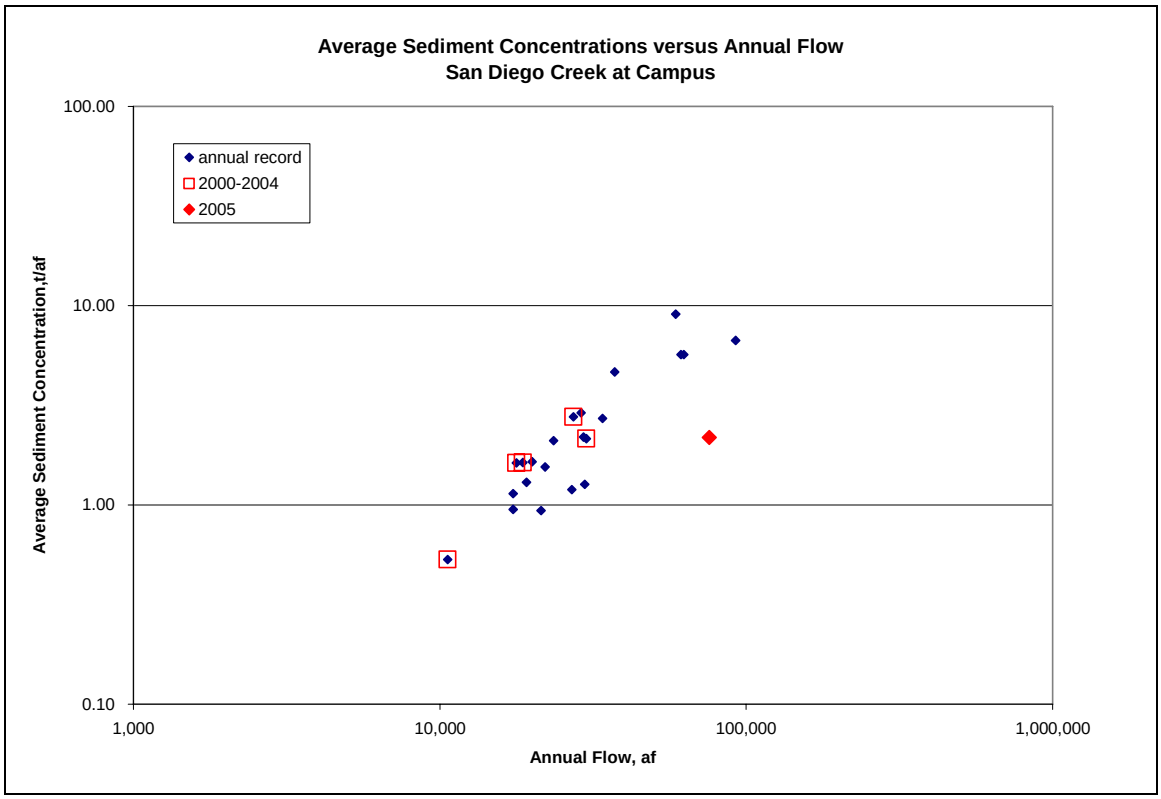

Figure 8

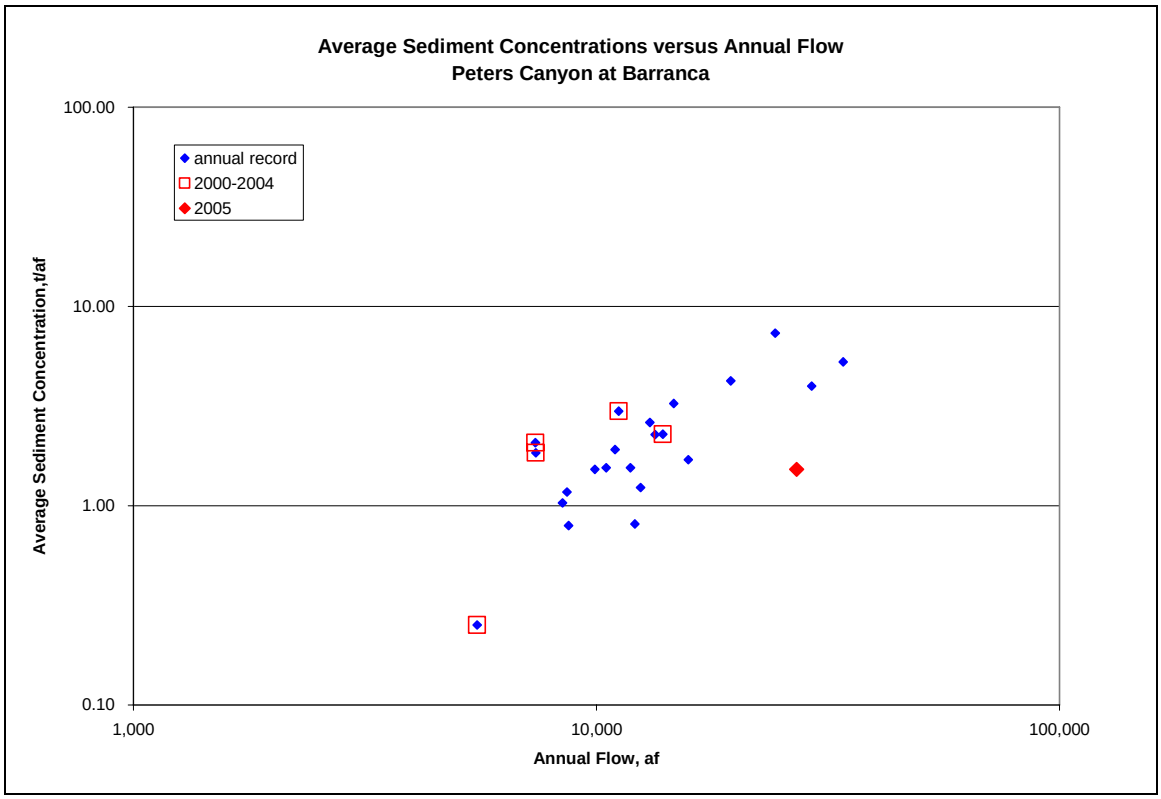

Figure 9

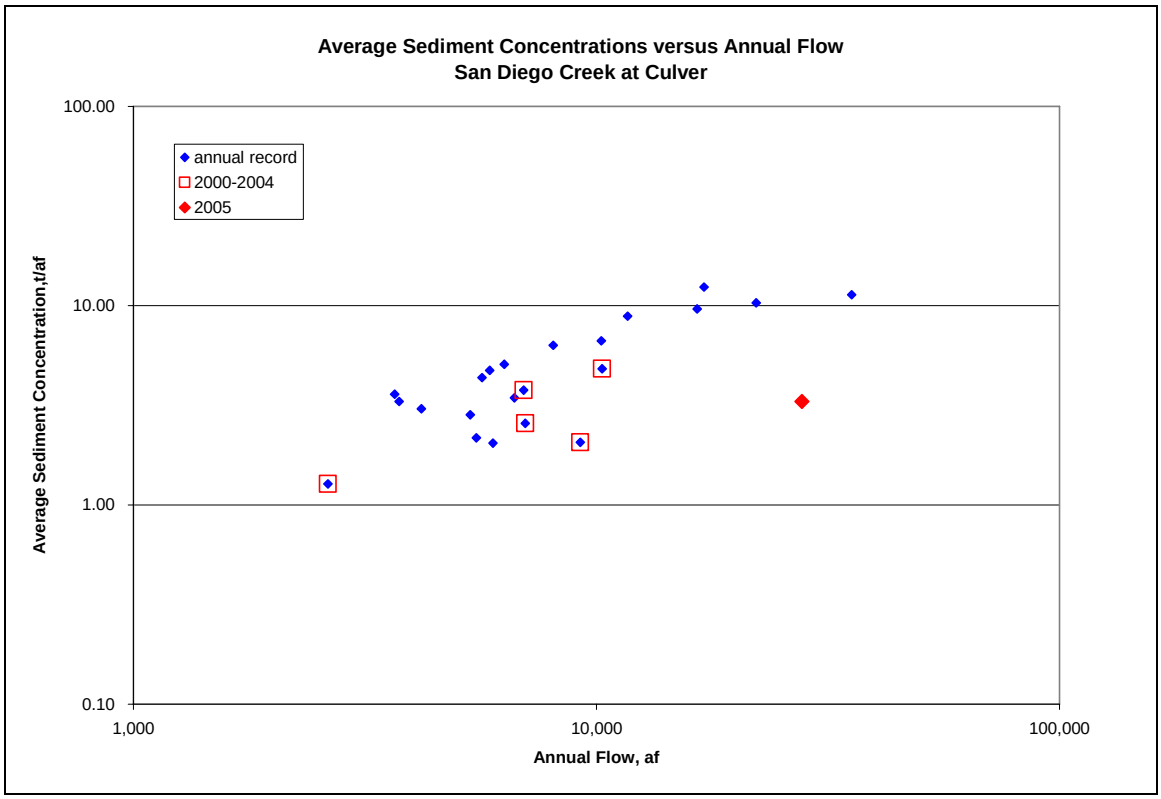

Figure 10

The 2005 data appears anomalous in contrast to prior recorded data. However, the available data presented below indicates that the changes in sediment concentration had been occurring over a period of time, rather than abruptly, as Figure 8 through 10 would indicate.

As shown in Figures 11, 12, and 13, there has been notable reduction in the sediment discharge versus flow discharge relationship at each of the long term gages over recent years. The County's practice has been to use multiple years of data in computation of the flow versus sediment transport relationship used in computation of sediment flows during unmeasured periods. Multiple years of data are used to describe this relationship to avoid over-reliance on a single year's data. The County has waited to update the relationship until this year – sufficient high flow measurements had not been available for better estimation of the current transport rates until the 2005 data had been produced. The recent update to the flow versus sediment transport relationship reflects several years of accumulation of changes rather than changes that occurred in 2005 alone. Thus, sediment discharges reported for the years between 1998 and 2005 have likely been overestimated.

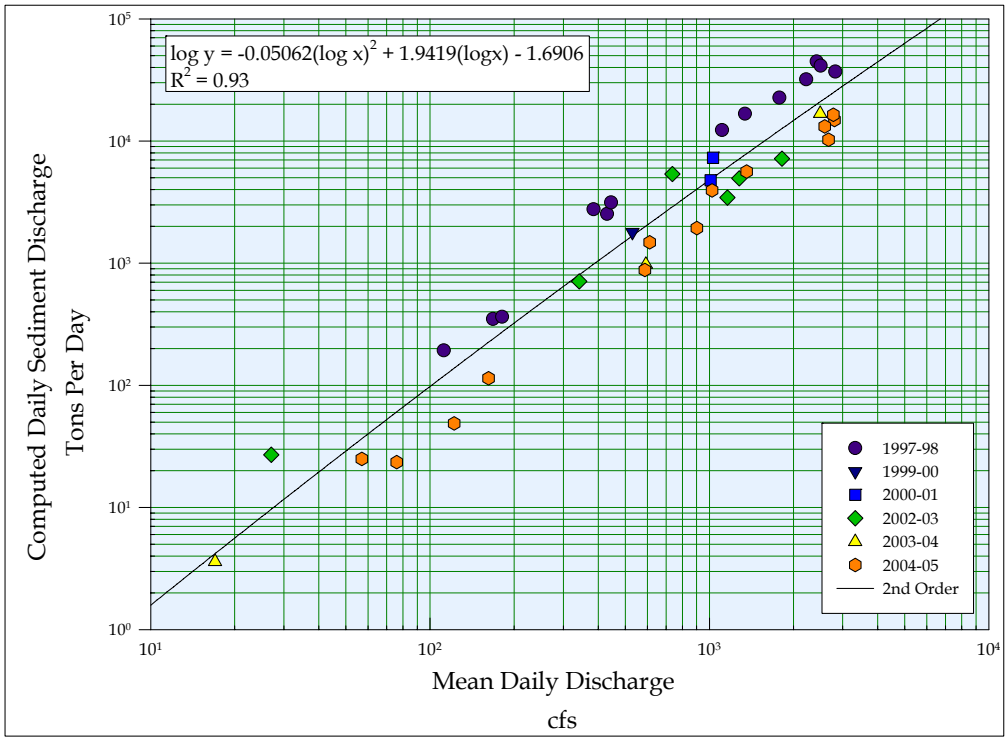

Figure 11 Sediment Transport Curve: San Diego Creek at Campus Drive 1998-2005

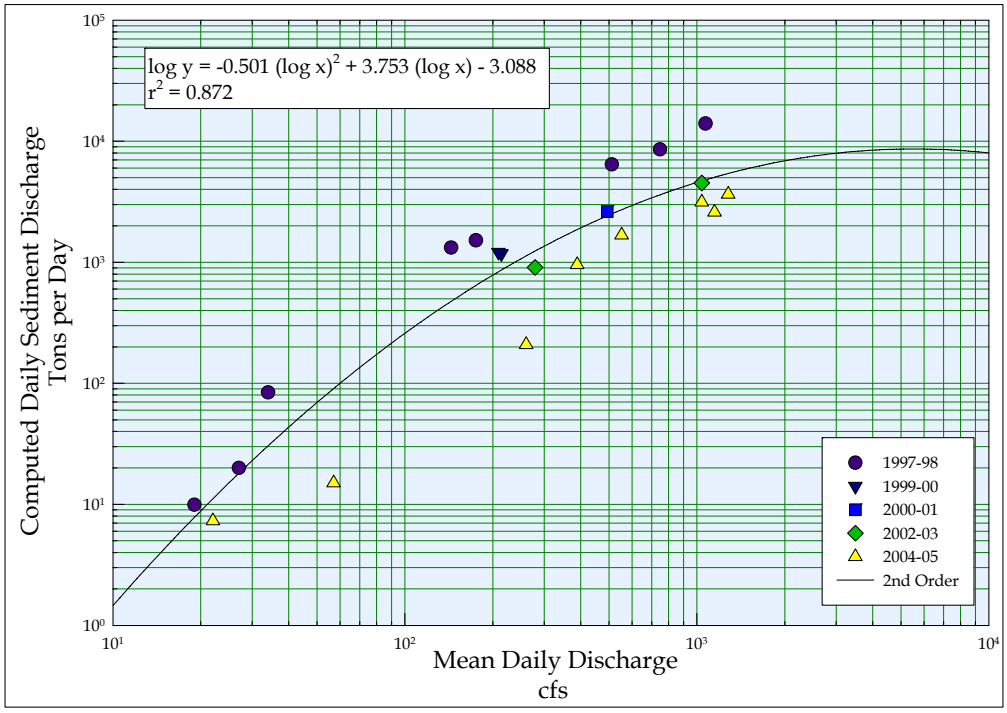

Figure 12 Sediment Transport Curve: Peters Canyon Wash at Barranca Parkway 1998-2005

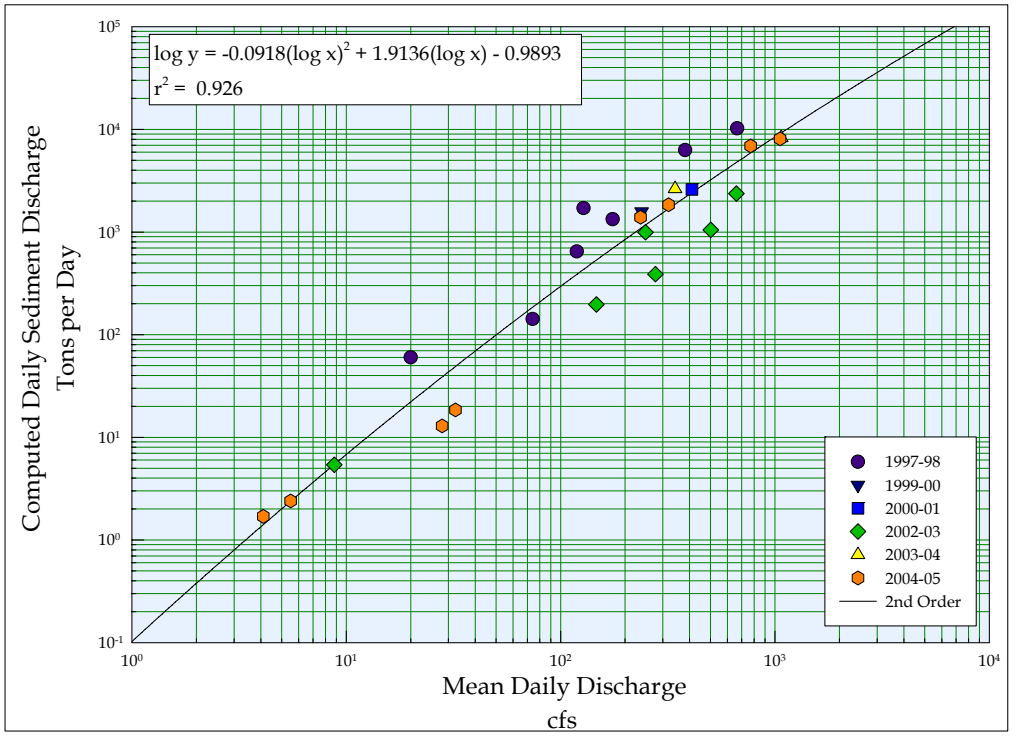

Figure 13 Sediment Transport Curve: San Diego Creek at Culver Drive 1998-2005

Sediment Load Components

The reduction in sediment load at the Campus Drive and Culver Drive locations has been predominantly associated in a reduction in the load of fine materials, as demonstrated in Figures 14 through 19.

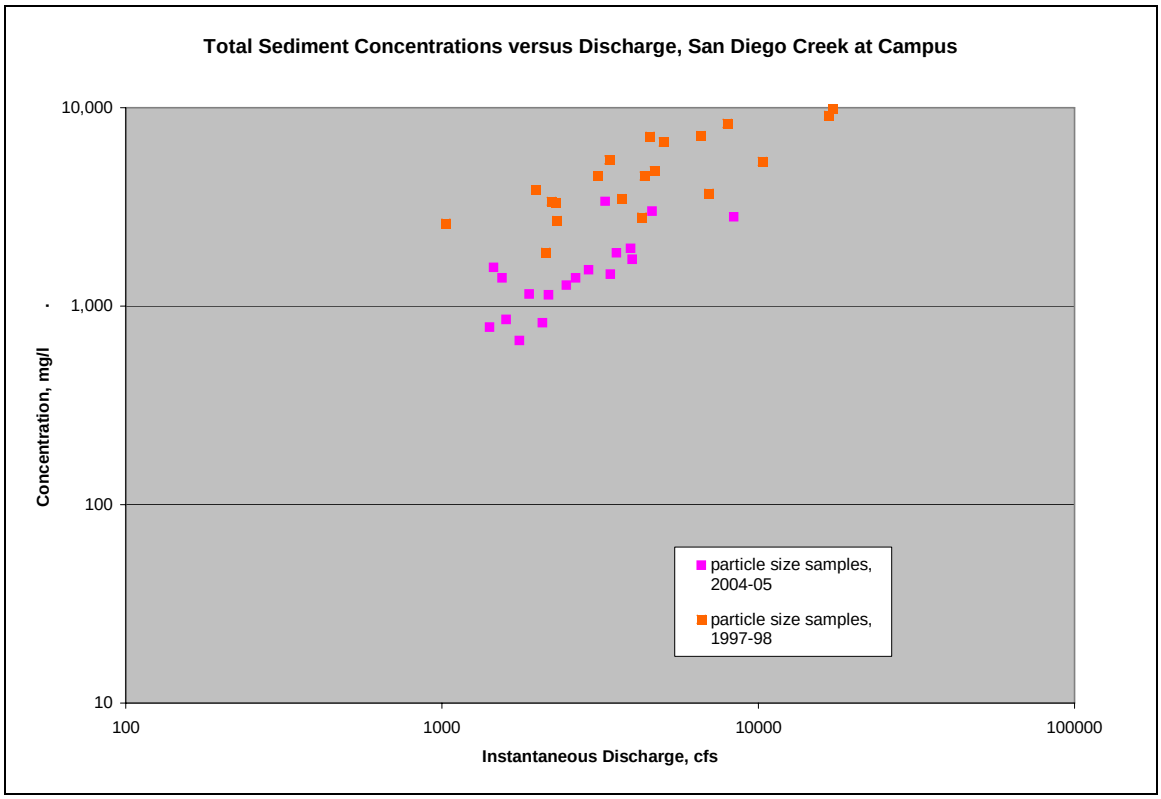

Figure 14

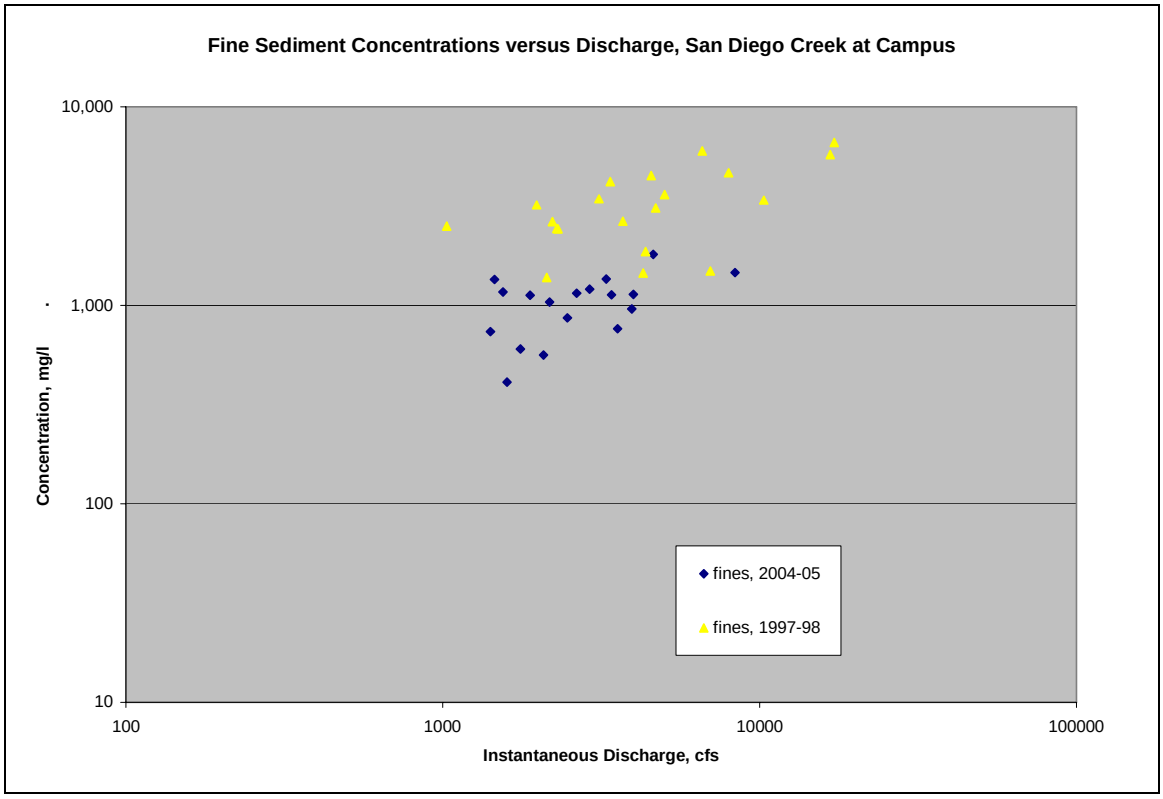

Figure 15

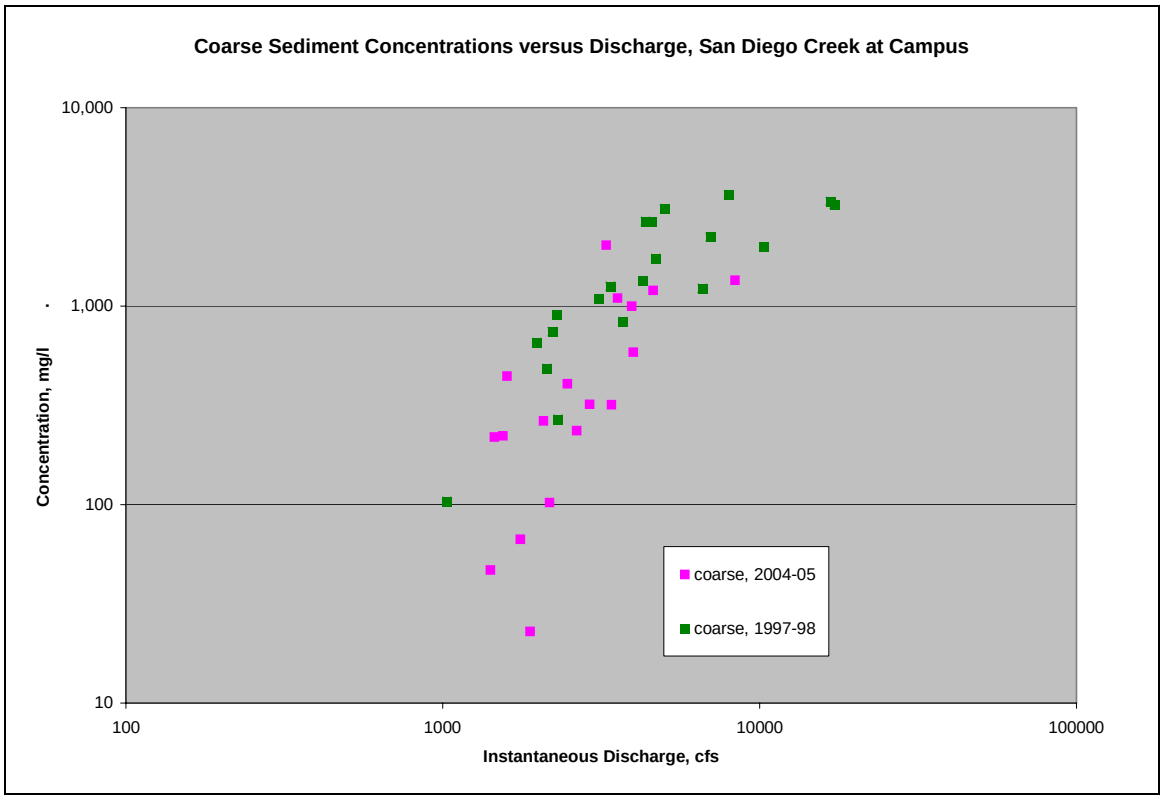

Figure 16

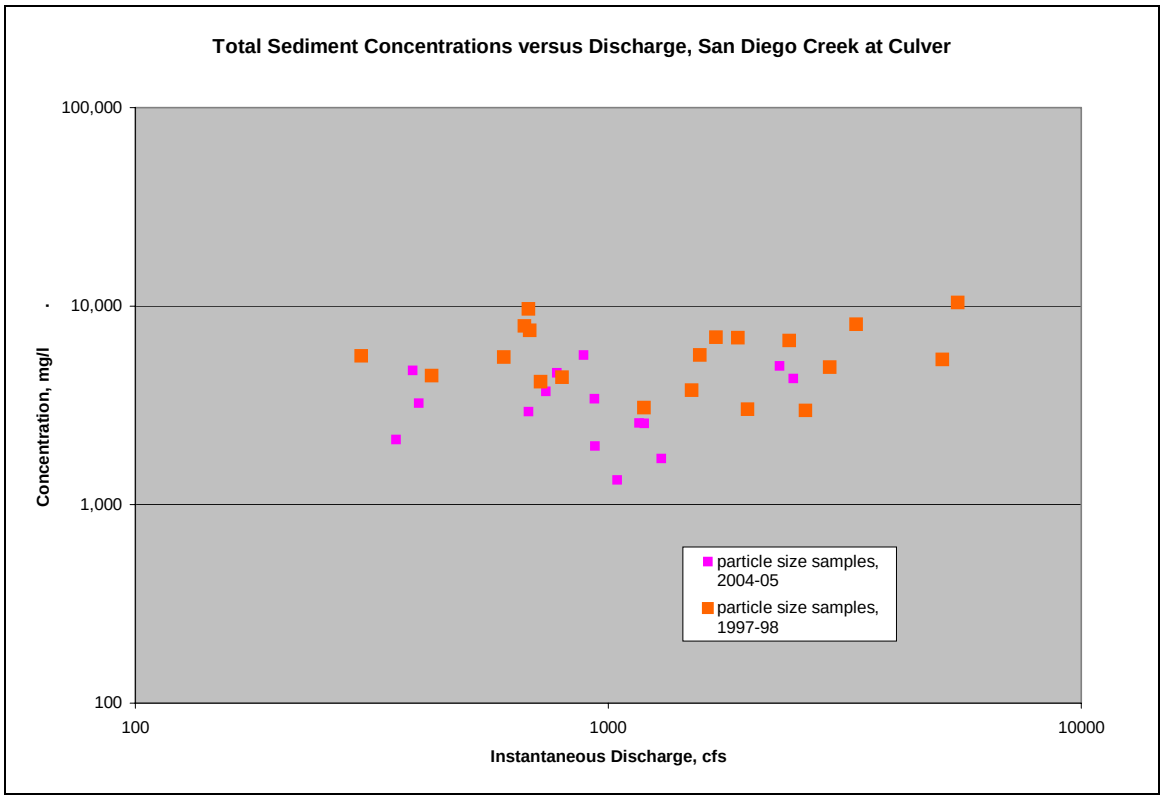

Figure 17

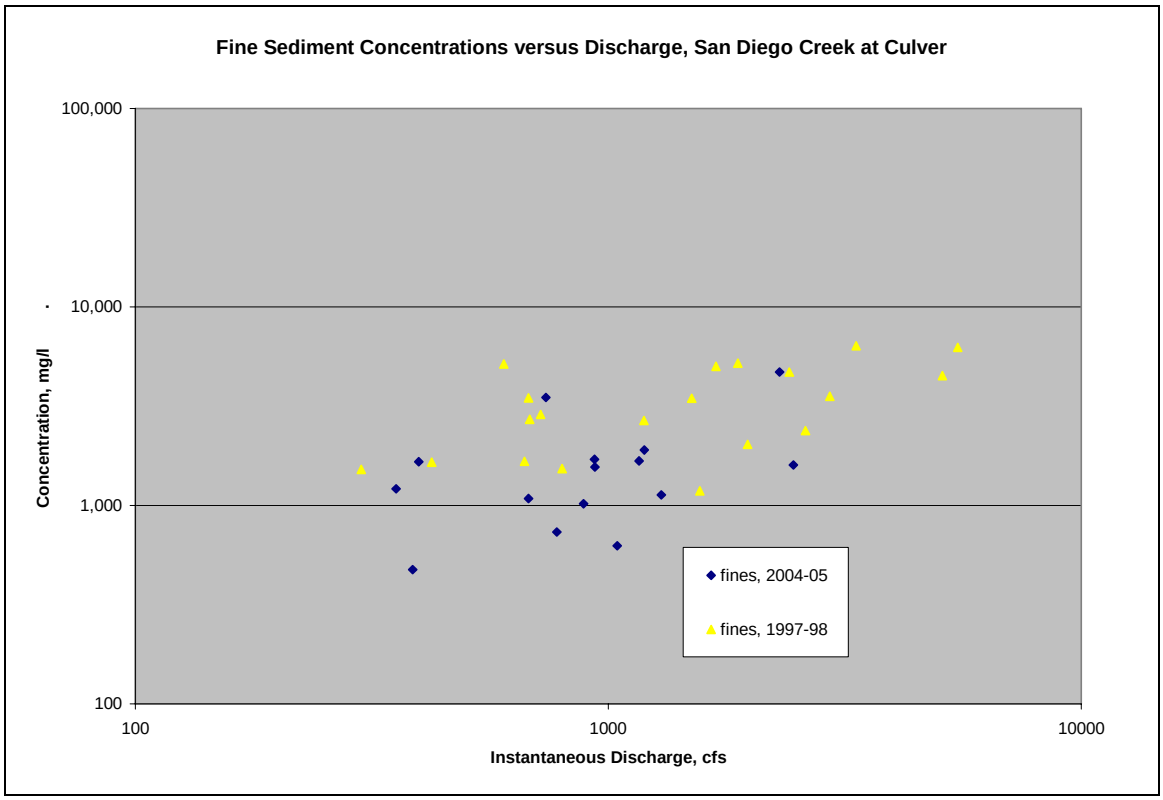

Figure 18

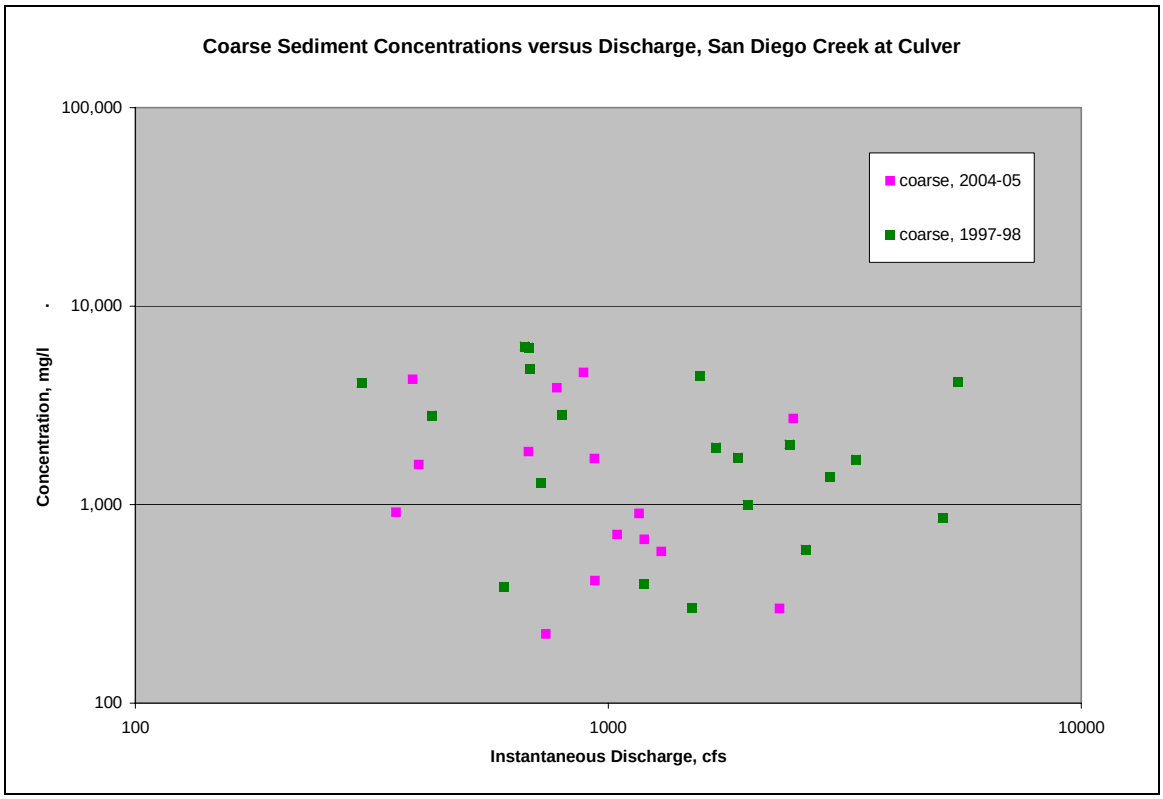

Figure 19

At the Peters Canyon monitoring location, reduction in both fine and coarse material loads have been observed, as indicated in Figures 20 through 22.

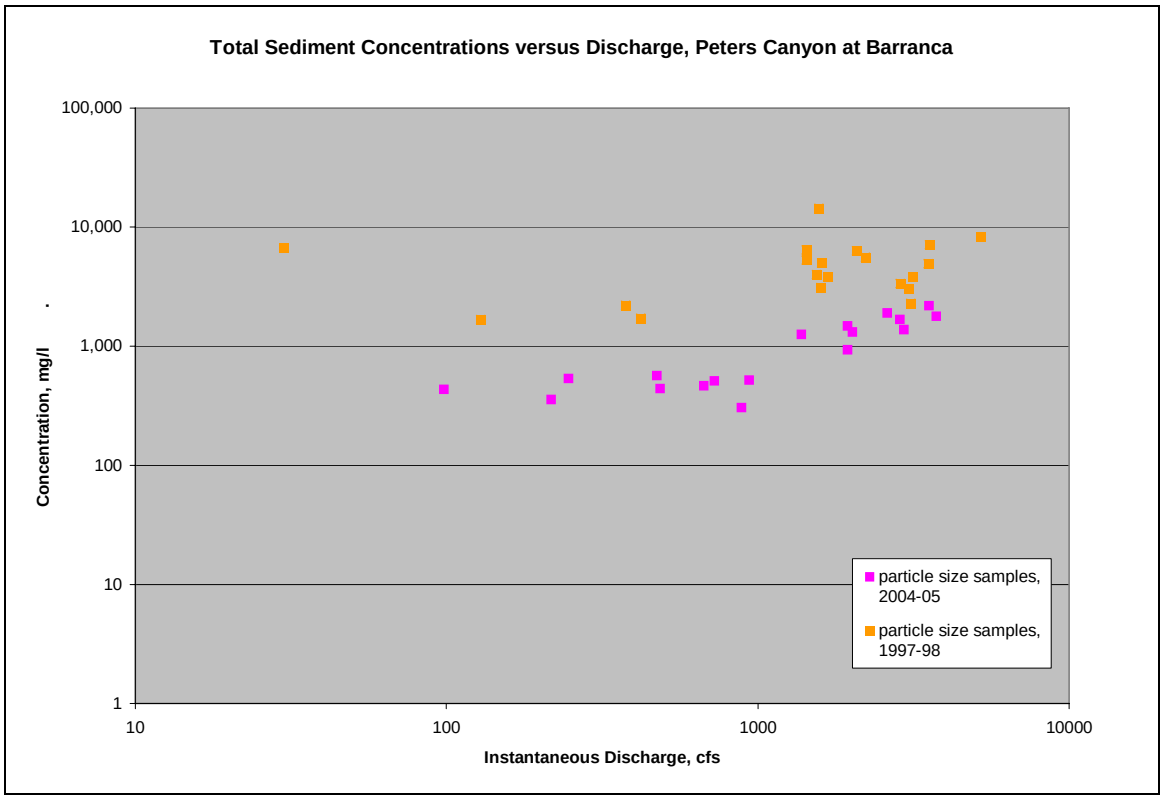

Figure 20

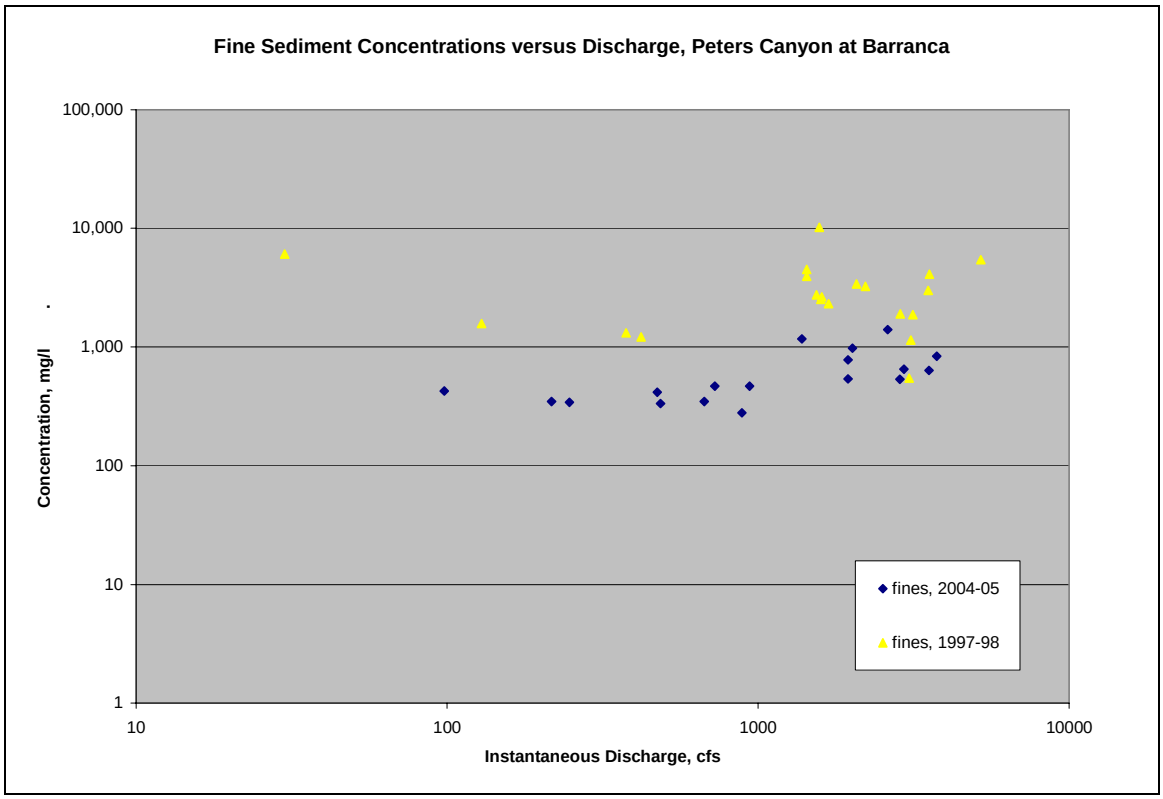

Figure 21

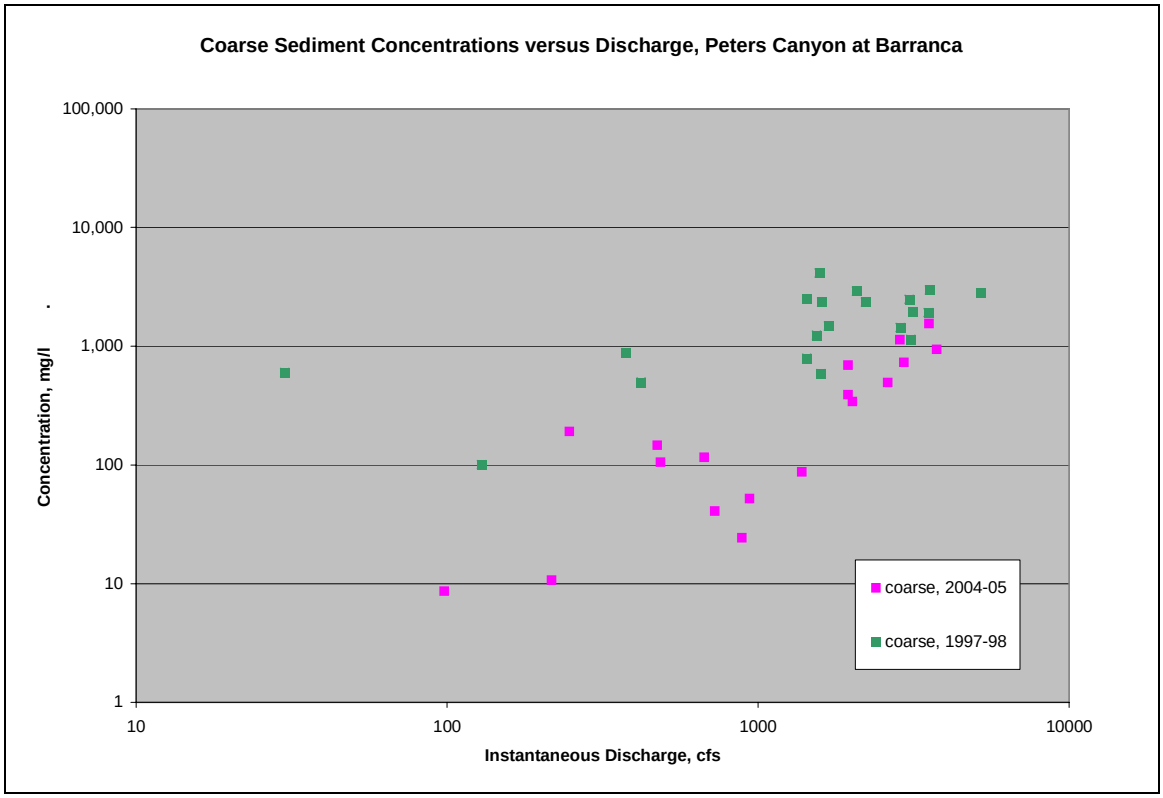

Figure 22

There is a distinct difference in the mechanisms controlling the amount of fine and coarse sediments carried by flows in San Diego Creek. The capacity for flows to carry fine sediments is very high, and is controlled only by the amount supplied from the watershed. In contrast, the capacity for flows to carry coarse sediments is controlled by the makeup of the channel bed (the bed material size distribution) and the hydraulic characteristics of the flow (particularly velocity and depth). As the San Diego Creek watershed becomes further developed, less and less watershed supply of sediment is released during storm events. This results in less sediment load entering the channels. In fully lined or otherwise armored channels with high sediment transport capacity, the sediment load the flow carries will be controlled by the amount of sediment supplied by the watershed. In channels composed of transportable material, flows will pick up additional sediment if the watershed supply is less than the flow is capable of carrying. The channel bed material is typically composed of the coarse sizes.

The above mechanisms explain the changes in sediment transport being observed at the Campus Drive, Barranca Parkway and Campus Drive locations. At each of these locations, changes have been observed in the amount of fine sediment load the flow is carrying at a particular flow rate – the transport curve has dropped. This reduction is likely associated with development within the watershed and measures being applied to reduce watershed erosion.

At the Campus Drive and Culver Drive locations, little change has been observed in the amount of coarse sediment load the flow is carrying at a particular flow rate, while at Barranca Parkway, the coarse sediment load has decreased. At both Campus Drive and Culver Drive, the San Diego Creek channel has an abundance of material for transport, and the channel properties are relatively constant – thus the mechanisms controlling bed material transport capacity have not changed. At Barranca Parkway, however, the channel shows evidence of incisement and the bed material is coarsening. Thus, the coarse material transport has shown reduction since the bed material size has increased. Over time, as the coarse material load from watershed supply to Peters Canyon continues to decline, the bed material will continue to coarsen. The process will continue until the reach becomes naturally armored, which may occur simultaneously with channel slope adjustment.

Figure 23 is an aerial view of the San Diego Creek/Newport Bay watershed with areas of recent development change noted. Areas of recent development were determined through comparison of 1994 and 2004 aerial photos. The 1994 data was limited to the western portion of the watershed (the lighter underlying photo area in Figure 23). However, the limited coverage of the 1994 data is believed to extend over the areas of recent development.

Much of recent development has been focused in the Peters Canyon watershed. Recent development within the Peters Canyon drainage basin extended over about 6 square miles, or 14 percent of the total subwatershed area. This recent development activity

modified approximately 50 percent of the portion of the Peters Canyon drainage basin that had not been developed prior to 1994.

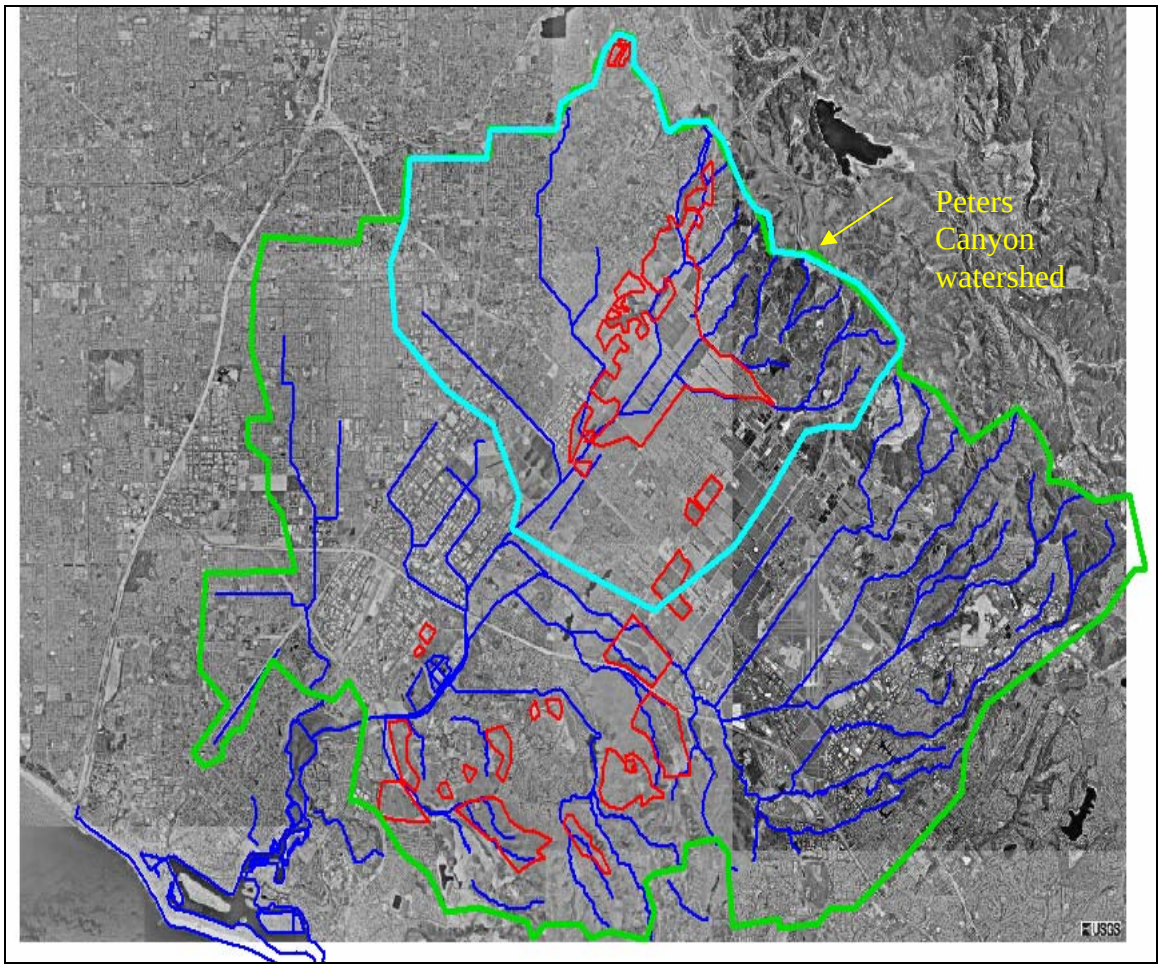

Figure 23 Newport Bay Watershed with Recent Development Areas Highlighted in Red

Very few data points are available for assessment of changes in sediment load in the Santa Ana-Dehli channel (see Figure 24). The earliest data was measured in the 2000-01 period, and shows quite a bit of scatter. The 2004-05 data points are few, though located at the low end of the previous scatter. Not enough measured data is available to assess any long term trends for this channel, though given its low historical loading and the fact that the contributing watershed is predominantly developed, little change here would be expected.

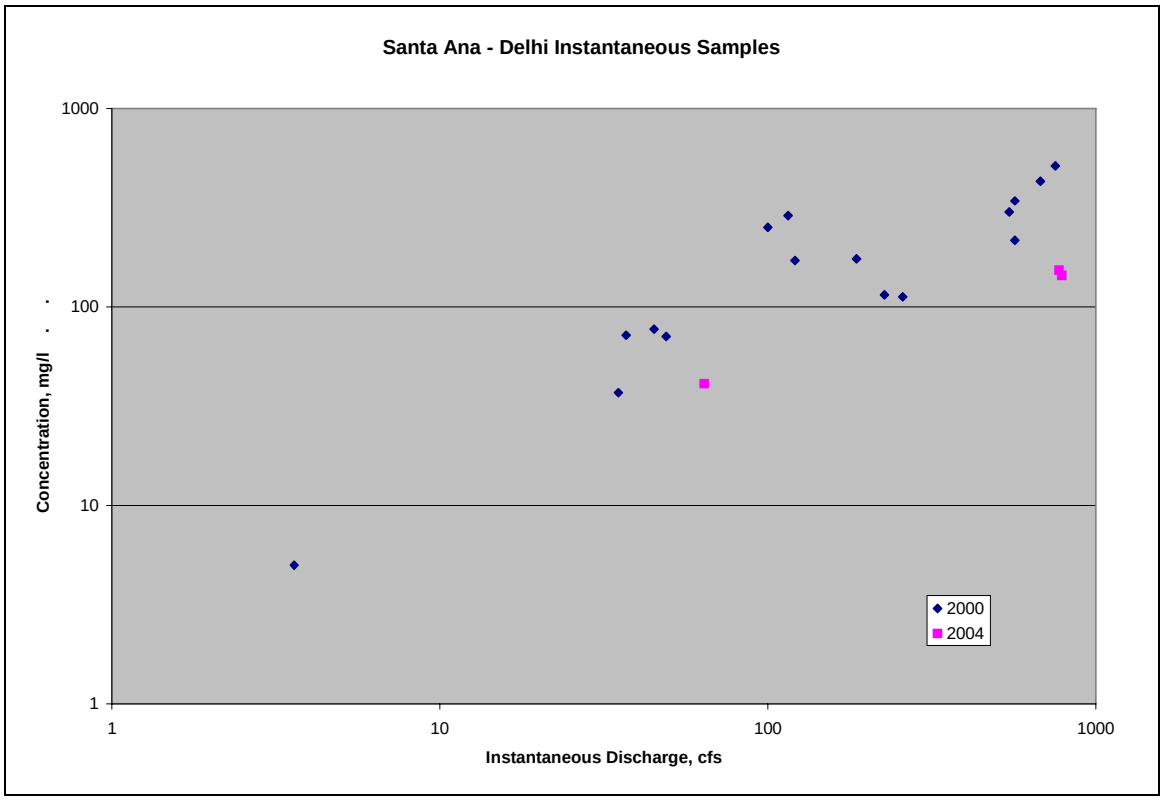

Figure 24

Conclusions

The available data indicates that sediment loads in the San Diego Creek watershed are reduced significantly from rates recorded in the pre-TMDL period.

At the Campus Drive monitoring location on San Diego Creek, sediment discharges measured during the 2004-05 period are lower than had previously been measured for the entire range of measured flow rates. There has been a progressive reduction in sediment discharge versus flow discharge relationship in recent years. The reduction in sediment load has been predominantly associated in a reduction in the load of fine materials.

At the Culver Drive monitoring location on San Diego Creek, the trends are less clear. The sediment discharge versus flow relationship for 2004-05 is lower than the 1997-98 period, but generally higher than measurements from the 2002-03 period. Any general reduction in sediment load since 1997-98 has been associated with decrease in fine sediment load – the coarse sediment load relationship appears to have remained constant.

At the Barranca Parkway monitoring location on Peters Canyon, the sediment discharges measured during the 2004-05 period are lower than had previously been measured for the entire range of measured flow rates. There has been a progressive reduction in sediment discharge versus flow discharge relationship in recent years. Both coarse and fine sediment load curves have significantly lowered since the 1997-98 monitoring period.

As the San Diego Creek watershed becomes further developed, less and less watershed supply of sediment is released during storm events. This results in less sediment load entering the channels.

At the Campus Drive and Culver Drive locations, little change has been observed in the amount of coarse sediment load the flow is carrying at a particular flow rate, while at Barranca Parkway, the coarse sediment load has decreased. At both Campus Drive and Culver Drive, the San Diego Creek channel has an abundance of material for transport, and the channel properties are relatively constant – thus the mechanisms controlling bed material (coarse) transport capacity have not changed. At Barranca Parkway, however, the channel shows evidence of incisement and the bed material is coarsening. Thus, the coarse material transport has shown reduction since the bed material size has increased. Over time, as the coarse material load from watershed supply to Peters Canyon continues to decline, the bed material will continue to coarsen. The process will continue until the reach becomes naturally armored, which may occur simultaneously with slope adjustment.

The remaining monitoring gages within the San Diego Creek watershed have record periods that are too short to assess any long-term trends. The loading estimated during the recent major precipitation period (2004/05) can be contrasted with the expected average annual loading computed for the preliminary load allocation and monitoring plan reports to provide more indication of current trends. This comparison indicates that contributions from the urban, construction, and agriculture land use portions of the watershed (predominantly represented by monitoring gages at the Santa Ana-Dehli, Bonita Canyon and Marshburn Channel, respectively) are less than had been estimated in the load allocation and monitoring plan study. These data indicate that open space land use areas are becoming more significant as contributors to the total sediment yield from the watershed.

As development continues in the watershed, the open space portions of the watershed will be increasingly the dominant land use type providing watershed sediment yield to the channel and Bay, particularly the fine sediment fraction of the total load. The non-armored channels will adjust in response to the coarse material supply available from these areas, and the channel beds will coarsen and/or flatten in response to reduced loading, a trend that is now observable in Peters Canyon. The coarser and/or flatter channels will ultimately transport sediment loads at rates in balance with the watershed supply.

References

(1) *Review of the Sediment TMDL Monitoring Program for the San Diego Creek Watershed; Examination of the 1999-00 through 2002-03 Monitoring Periods.* Prepared by URS for County of Orange Public Facilities and Resources Department, December 18, 2003.

(2) *Preliminary Sediment Load Allocation Analysis for San Diego Creek and Newport Bay*. Prepared by Tetra Tech, Inc., for County of Orange Public Facilities and Resources Department, October 1999.

(3) *Sediment Load Allocation Monitoring Plan, San Diego Creek and Newport Bay.* Prepared by Tetra Tech, Inc., for County of Orange Public Facilities and Resources Department, June 2000.

(4) *Upper Newport Bay/San Diego Creek Watershed TMDL 2004-05 Report.* Prepared by County of Orange Resources and Development Management Department, January 2006.