DDT, Chlordane, Toxaphene and PCB Residues in Newport Bay and Watershed: Assessment of Hazard to Wildlife and Human Health

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1 Introduction

DDT (dichlorodiphenyltrichloroethane), chlordane, toxaphene and PCBs (polychlorinated biphenyls) are persistent organochlorine chemicals that can still be widely found in soils and aquatic environments decades after use has been discontinued. Under the Clean Water Act, these chemicals are regulated by a total maximum daily load (TMDL) for each watershed to achieve levels that are not toxic to wildlife or humans. In Newport Bay and Watershed (Orange County, California), the development of TMDLs for these legacy organochlorines has been underway for more than a decade.

In 2002, the United States Environmental Protection Agency, Region IX (US EPA Region IX) promulgated TMDLs for DDT, chlordane, toxaphene and PCBs in Newport Bay and Watershed. The finding of impairment, and therefore the necessity for the TMDLs, was based on certain target concentrations for water, sediment and fish. The Santa Ana Regional Water Quality Control Board (SARWQCB or Regional Board) revised and approved these TMDLs in 2007, with the condition that an independent advisory panel (IAP) of experts review the science underlying the TMDL targets. The IAP was formed by Orange County. They met and considered the TMDLs and the underlying science. They pointed out flaws in the science supporting the TMDLs and recommended developing TMDLs based on site specific food chain bioaccumulation of the legacy organochlorines (IAP 2009). The California State Water Resources Control Board (CSWRCB or State Board) and Region IX of the USEPA have recently approved the 2007 TMDLs; implementation of the TMDLs and reconsideration of their targets based on the IAP recommendations was begun in 2013.

The regulated community has pointed out throughout the TMDL proceedings that these organochlorines are no longer in use, that residue levels are declining, and that there are no apparent effects on wildlife or human health. They have also pointed out that many of the targets to be implemented are not based on sound science. The important question for all concerned is whether the chosen targets are scientifically sound and whether current levels meet or exceed scientifically sound targets.

Technical reports that address different aspects of the TMDL process or the targets have been written by US EPA Region IX (2002), SARWQCB (2006) and scientists working for the regulated community (Flow Science et al. 2006; Byard 2011, 2012a, b). Scientists representing the regulated community and regulatory agencies have met on numerous occasions and have exchanged comment letters, including comment letters from outside scientists (Daniel Anderson, Donald MacDonald and ten others), in addition to the report from the IAP. In this review, we provide an analysis of the science underlying the organochlorine TMDLs for Newport Bay and Watershed. Since organochlorines are regulated by the TMDL process in many other locations, the analysis herein represents a case study that may have application to other watersheds.

2 Newport Bay and Watershed

2.1 Location

The Newport Bay Watershed is centrally located in Orange County, California. The 154-square mile Watershed includes portions of the cities of Newport Beach, Irvine, Laguna Hills, Lake Forest, Tustin, Orange, Santa Ana, and Costa Mesa. Runoff from the mountains that surround three sides of the watershed drains across the Tustin Plain and enters Upper Newport Bay via San Diego Creek, one of two major tributaries. Peters Canyon Wash, the other tributary, joins San Diego Creek in the City of Irvine. These combined waterways drain 75,520 acres and are major contributors of freshwater and sediment, and associated pollutants to Newport Bay, which includes both Upper and Lower Newport Bay, as shown in Fig. 1.

2.2 Climate/Hydrology

The area has a Mediterranean climate, with short, mild winters and warm dry summers. Ninety percent of the precipitation occurs during November to April with an average rainfall of approximately 13 in. per year. San Diego Creek has a wide range of water

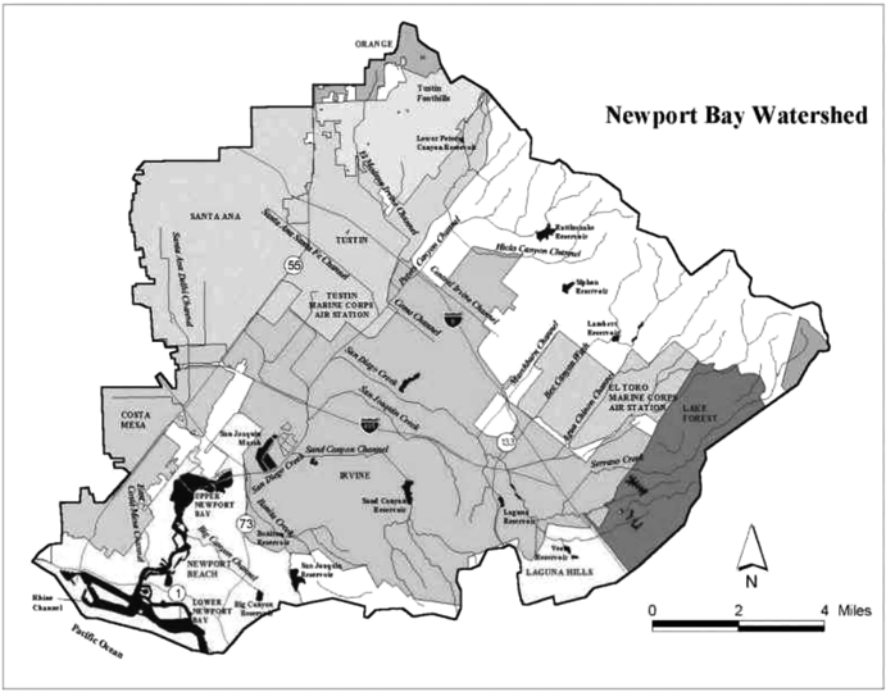

Fig. 1 Newport Bay and Watershed. Figure 1-1 reproduced from U.S. EPA Region IX (2002)

hardness and is influenced by the slightly saline water table (less than 2% salinity). Prior to the 1960s, San Diego Creek was not connected directly to Newport Bay, but an engineered flood control system was constructed within the watershed making the connection permanent. Flows now enter Newport Bay through San Diego Creek throughout the year. San Diego Creek currently has a mean base flow rate of approximately 12 cubic feet second (cfs). Storm events can increase this daily flow to over 9,000 cfs. The Upper Bay's estuary has saline water conditions during dry weather, yet experiences a heavy influx of freshwater from San Diego Creek and other tributaries during major storms. Water quality in the Lower Bay is intermediate between the Upper Bay and the Pacific Ocean.

2.3 Land Use

Land within the Newport Bay Watershed was first settled during the late nineteenth century, leading to the development of agriculture on a large portion of the inland areas. The end of World War II brought urbanization to the area, and land use changed significantly in the past 60 years from agricultural to residential and commercial uses. In 1983, agricultural and urban uses accounted for 22% and 48% of the Newport Bay Watershed, respectively, but by 1993, the proportions were 12% and 64% (US EPA Region IX 1998). As of 2000, agricultural uses had dropped to approximately 7% of the watershed area (US EPA Region IX 2002). Based on plans for development of agricultural lands, this trend is expected to continue.

2.4 Water Quality

Both Upper and Lower Newport Bay and San Diego Creek have been listed as impaired for possessing "unknown toxicity." The toxics TMDL promulgated in 2002 by US EPA Region IX was intended to address the unknown toxicants responsible and develop limits for selenium, metals, organophosphates and organochlorines. Other TMDLs are in place for nutrients (US EPA Region IX 1998), fecal coliforms (SARWQCB 1999), and sediment (SARWQCB 1998).

Researchers have observed acute toxicity in San Diego Creek and Newport Bay. Acute toxicity was observed in urban storm water runoff and in agricultural drainage from some types of crops in the watershed (Lee and Taylor 2001). Bay et al. (2004) collected sediment samples in 2000 and 2001 and found toxicity at multiple locations in both Upper and Lower Newport Bay. It was concluded in both studies that the acute toxicity was not caused by organochlorine compounds, but more likely was attributable to organophosphate (diazinon and chlorpyrifos), carbamate, and pyrethroid pesticides (Lee and Taylor 2001; Bay et al. 2004). Although uses of diazinon and chlorpyrifos have been phased out by the US EPA, other organophosphates, carbamates and pyrethroid pesticides are still used in residential, agricultural and commercial

applications, such as the commercial nurseries formerly located in the upper part of the Watershed. Recent studies indicate that the acute toxicity of sediments from Upper Newport Bay is diminishing (Orange County Watersheds 2008–2011).

Understanding sediment loads is important to understanding the fate of organochlorines, because these chemicals bind tightly to soil and sediment particles. Annual sediment loads discharged from the Watershed were estimated at approximately 250,000–275,000 t during the rapid urbanization period of the 1980s and 1990s (US EPA Region IX 1998). Much of the sediment load resulted from in-stream erosion (Trimble 1997). Because of the volume of sediments deposited within the Bay, the Upper Bay was dredged in 1983, 1985, 1988, 1999 and 2010 (Newport Bay Conservancy 2013). Implementation of the sediment TMDL (SARWQCB 1998) has led to reduced sediment loads.

Stream stabilization and other measures have reduced sediment loads to the Bay.[1] Flow rate and suspended sediment discharge samples collected at San Diego Creek showed that although average annual flow volume for the years 2000–2005 was roughly equivalent to the average annual flow volume for 1983–1999, average annual sediment discharge for the latter period was only 42% of the average annual sediment discharge for 1983–1999 (see footnote 1). Orange County's consultant attributed this reduction in sediment load to land development, effectively capping soils, and to erosion control measures in the watershed. Moreover, this consultant found that "[a]s the San Diego Creek watershed becomes further developed, less and less watershed supply of sediment is released during storm events (see footnote 1)."

In the next section, we address the fate of DDT in the Watershed and the science underlying the DDT TMDL targets.

3 DDT

3.1 Levels in the Environment

DDT was first used as an insecticide in California around 1944 and was in wide use by 1947. In 1963, the California Department of Food and Agriculture declared it a restricted material, and 1972 was the last year that DDT was applied to crops in the state (Mischke et al. 1985).

According to the United States Department of Health and Human Services (US DHHS 2002), commercial DDT is a mixture of several congeners, and typically has a composition of 65–80% *p*,*p*′-DDT and 15–21% *o*,*p*′-DDT. In the environment, DDE and DDD are the major degradation products of DDT. DDT and its congeners are persistent in the environment and have been found in various animal species, in water, in soil, and in sediment (US DHHS 2002). As indicated below and except as otherwise indicated, DDT is used to represent the sum of all measured DDT congeners.

[1] County of Orange Resources and Development Department 2006.

DDT is strongly hydrophobic; for example, *p*,*p*′-DDE, the main metabolite of concern, has a K_{ow} of 6.956 (de Bruijn et al. 1989). After application to soils, DDT may be lost through both volatilization and biodegradation. Volatilization tends to be the more important removal mechanism initially, while biodegradation is more important later in the removal process (US DHHS 2002). As a result of both these processes, DDT removal from soils tends to be non-linear, and thus the first 50% of DDT tends to be removed from soil more quickly than subsequent halves, such that the half-life of DDT in soil may increase over time (US DHHS 2002).

A variety of studies have been conducted to characterize the half-life of DDT and its metabolites. In temperate climates, the half-life of DDT in soil has been reported to range from 2.3 to 16.7 years (Lichtenstein and Schultz 1959; Racke et al. 1997; Stewart and Chisholm 1971).

Although it has been suggested that other non-organochlorine pesticides, such as dicofol, continue to be used in the watershed and may include small amounts of DDT, the SARWQCB (2006) concluded that dicofol contains minimal levels of DDT and is therefore an "inconsequential continuing source in the watershed." Mischke et al. (1985) concluded that DDT levels in dicofol were too low to account for the DDT soil residues found in their 1985 study of agricultural residues in California soils.

As discussed below, recent data from Newport Bay and Watershed indicate that DDT concentrations are declining in all media where historical and recent data are available, and these data have been used to estimate the half-life of DDT in the local watershed.

3.1.1 Agricultural Soils

Table **1** presents historical DDT concentrations for agricultural soils in the Newport Bay watershed. In general, these soils seem to exhibit a downward trend in DDT concentrations over time, which is expected given a DDT half-life of less than

Table 1 DDT concentrations in agricultural soils in the Newport Bay Watershed

Year	0-12 inch Sample Depth			12-24 inch Sample Depth			>24 inch Sample Depth			Detection Limits (ppm)
	Range of Detected Total DDT (ppm)	Total Samples	Total Non-detect Samples	Range of Detected Total DDT (ppm)	Total Samples	Total Non-detect Samples	Range of Detected Total DDT (ppm)	Total Samples	Total Non-detect Samples	
1985	0.001 - 1.750	12	0							--
1987	0.034 - 1.500	10	0	0.025 - 2.140	10	4	--	10	10	0.016
1988	0.027 - 1.090	10	1	0.095 - 0.150	10	8	0.550 - 0.550	10	9	<0.027 - 0.064
1989	0.024 - 0.791	15	6	0.052 - 0.707	10	5	0.016 - 0.333	19	13	0.016
1990	0.102 - 0.900	4	1	0.110 - 0.910	2	0	0.020 - 0.197	7	3	0.016
1991	0.019 - 0.488	34	4	0.010 - 0.490	32	14				--
1995	0.085 - 0.806	19	1							--
2000	0.007 - 0.132	28	0							0.005
2002	0.005 - 1.620	174	34				0.020 - 0.073	27	17	--
2004	0.002 - 2.000	230	167				0.002 - 0.300	45	36	0.002 - 0.2
2006	0.013 - 0.157	6	1				0.005 - 0.005	6	5	0.005

Sources: Unpublished technical report provided by the SARWQCB (1985); unpublished technical reports provided by The Irvine Company (1985–2006); no data were available for shaded areas and for entries noted with '--'

20 years (Lichtenstein and Schultz 1959; Racke et al. 1997; Stewart and Chisholm 1971) and the fact that DDT use was discontinued in the early 1970s. However, the data reported in Table **1** were not sampled from soils at the same locations. Given that no data were available showing the amounts of DDT historically applied to different areas of the watershed, the DDT data on agricultural soils cannot be used to assess trends over time or local DDT half-life values.

Several agricultural soil DDT data points have also been reported in Mischke et al. (1985) for Orange County. Total DDT concentrations in that report ranged from 0.32 to 2.96 ppm for three different sample locations. However, the precise locations of these samples could not be identified from the report, and thus the data were not useful for establishing trends in agricultural soil DDT concentrations in the Newport Bay watershed.

Peak DDT concentrations at the 12–24 in. depth were generally comparable to concentrations in the top 12 in., while peak DDT concentrations in samples collected from a depth of 24 or more inches were roughly two to sixfold lower than concentrations at the surface. Sampling locations for several sampling years are presented in Fig. 2; note that exact locations for samples collected in 1988, 1991, and 1995 are not known. Data from those years are shown using the average concentration in the approximate location of sample collection.

If one conservatively assumes a half-life of 20 years for DDT in soil, that DDT was banned in 1972, and if other losses or removal mechanisms are excluded, the mass of DDT in the agricultural soils of the Newport Bay watershed would have declined by approximately 71% over the past 36 years solely from soil degradation.

Because DDT adsorbs strongly to soil particles, the predominant pathway for movement in the watershed is via soil erosion. Two related changes within the watershed have served to minimize the transport of DDT to the waters within the Newport Bay watershed. First, urban development initially has led to a conversion of land use away from agricultural use and toward residential, commercial, and industrial development, which increases the impervious land and minimizes direct erosion from land surfaces. Much of this land use conversion has occurred on land that was in agricultural production prior to 1972, when DDT was in use, and much of the land currently in agricultural production was first farmed after 1972, indicating that DDT would not have been applied to these areas. Second, several measures, including channelization and construction of and improvements to the flood control system, have resulted in decreased sediment loads being delivered to the Bay, and therefore, decreased sediment yield of the watershed over time.[2] Development in the watershed has and will continue to reduce the amount of DDT available to biota in the watershed.

[2] WRC Consulting Services Inc, Historical Sediment Load Examination, San Diego Creek Watershed. Report prepared for County of Orange, Resources and Development Management Department. June 28, 2006.

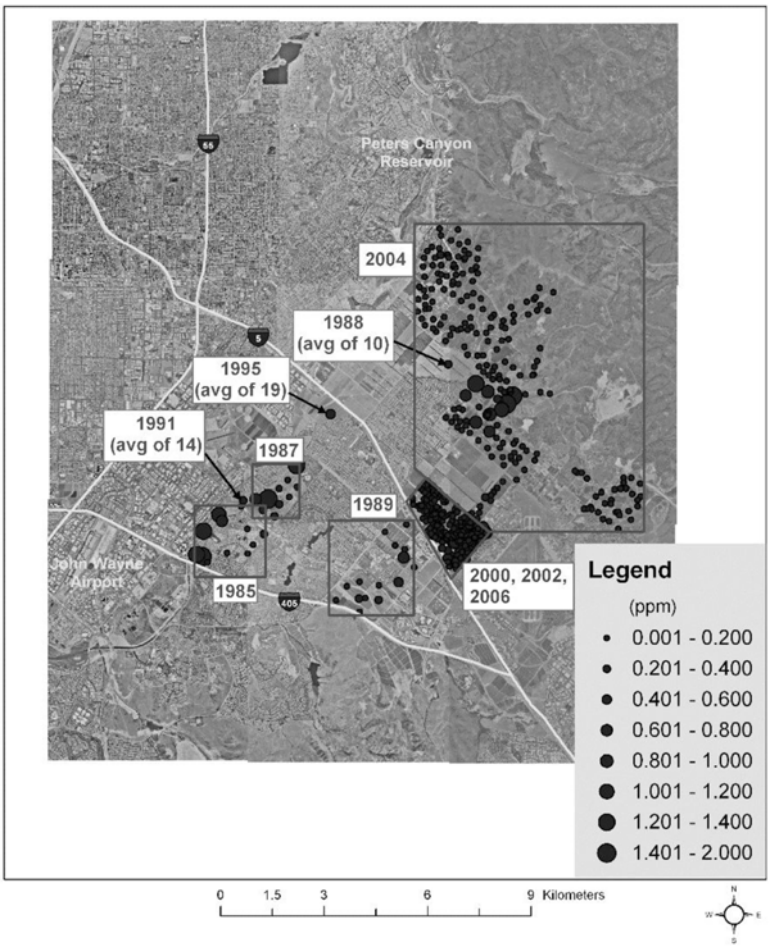

Fig. 2 Locations of agricultural soil samples analyzed for DDT. Data were from unpublished technical reports provided by The Irvine Company (1985–2006). The aerial photo is a composite of photos taken in 1994 and 1995

3.1.2 Sediments

Sediment data are plotted in Fig. 3 for Lower and Upper Newport Bay for the period 1980 through 2011. There are no data available for the period from 1987 to 1995.

Bay-wide trends in sediment DDT concentration over time are difficult to infer from these data for several reasons. First, sampling was conducted by multiple agencies, using multiple methodologies, at varying locations and sample depths. Given this diversity in sampling approach and location, direct comparisons between data from year to year are inappropriate. Second, there is significant movement of sediment into, out of, and within the Bay such that even samples taken in the same

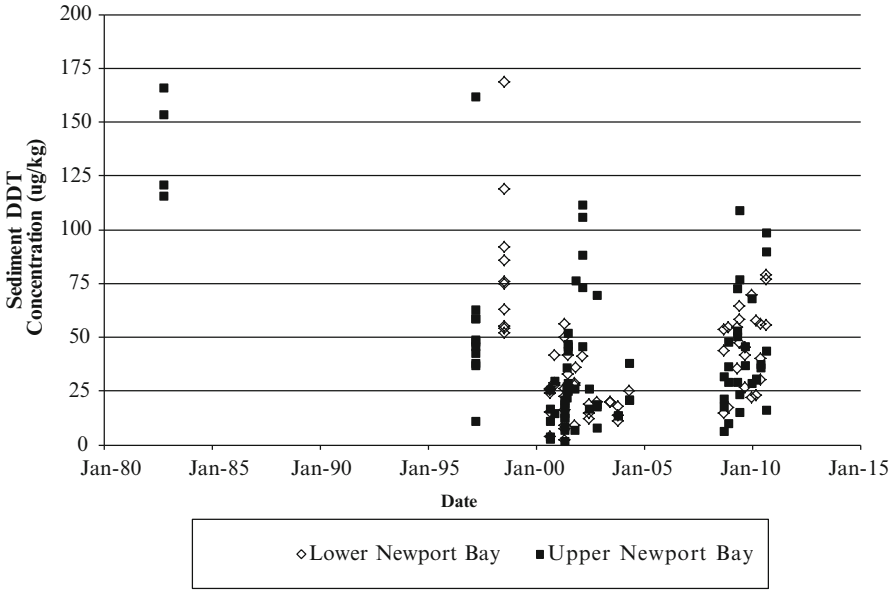

Fig. 3 DDT concentrations in sediment from Newport Bay. Values above detection limits are shown. *Sources*: Orange County PFRD (1980–1986) (Personal communication from Bruce Moore of the Orange County Public Facilities and Resources Department (OCPFRD). Unpublished sediment data for 1980–1986); SCCWRP (1998); Bay et al. (2004); unpublished reports provided by The Irvine Company (2000–2004); US EPA Region IX (2002); Masters and Inman (2000); Orange County Watersheds (2008–2011)

location at two different times may not represent the change in DDT concentration for a specific quantity of sediment. Sediment movement results both from tidal flows and storm flows, as well as from periodic major dredging projects in the Upper Bay, which have occurred in years previously noted. Third, sediment concentrations in Newport Bay may be more indicative of DDT loads from years or decades past, since Bay sediments are transported from the upper watershed in a highly variable, episodic manner, correlated with storm events and wetter-than-average rainfall years.

Thus, DDT concentrations in Bay sediments reflect DDT that was applied many years ago in the upper watershed, and then sorbed to sediments in that location, which were subsequently eroded into a creek channel and transported to the Bay. Finally, Bay sediment DDT concentrations found are not necessarily bioavailable. This is especially true of samples collected from deeper sediment cores. While sample depths were not available for all data plotted in Fig. 3, the Orange County PFRD data from 1980 through 1986 reflect sample depths between 2 and 25 ft, with an average of 11 ft, well below the biologically active layer, which extends only to a depth of approximately 6 in. Thus, these early sediment samples are not indicative of concentrations available to biota in the Bay. For all these reasons, the available sediment data for Newport Bay are not reliable indicators of bioavailable DDT concentration trends in the watershed and should not be used independent of other available data.

Table 2 DDT concentrations in the water column in Newport Bay and Watershed

Date	Water body	Sample location	Fresh water flows	Total DDT (ng/L)[a]
4/23/2001	Lower Bay	Turning Basin	Unspecified	1.29
4/23/2001	Lower Bay	PCH Bridge	Unspecified	1.04
3/12/2002	Rhine Channel	NB3	Unspecified	ND
3/13/2002	Upper Bay	NB10	Unspecified	ND
3/7/2002	San Diego Creek	Campus Drive	Dry weather	ND
3/7/2002	San Diego Creek	Campus Drive	Storm	ND
5/2/2002	San Diego Creek	Campus Drive	Dry weather	ND
5/2/2002	San Diego Creek	Campus Drive	Dry weather	ND
8/12/2002	San Diego Creek	Campus Drive	Dry weather	ND
8/12/2002	San Diego Creek	Campus Drive	Dry weather	ND
11/8/2002	San Diego Creek	Campus Drive	Storm	3
11/8/2002	San Diego Creek	Campus Drive	Storm	ND

[a]Detection limit = 1.0 ng/L
ND is not detected
Bay et al. (2004) and Bay and Greenstein (2003)

3.1.3 Water Column

Twelve water samples have been collected that characterize DDT concentrations in the waters of the Newport Bay and Watershed, as shown in Table 2. Accurate measurement of the very low levels at which DDT is present in water in the Bay is difficult, and only 3 of 12 data points, which were all collected in 2001 and 2002, were above detection limits. For these reasons, no meaningful trend analysis could be performed on concentrations of DDT in water. The California Toxic Rule (CTR) human health regulatory threshold for DDT in water is 0.00059 µg/L, or 0.59 ng/L (US EPA Region IX 2000).

3.1.4 Fish and Mussels

A rigorous statistical analysis of DDT concentration data was conducted for three different media: fish tissue, mussel tissue, and sediment. This analysis demonstrated that DDT concentrations in red shiners and in mussels collected from San Diego Creek, Upper Newport Bay, and Lower Newport Bay are declining in the watershed, and that these trends are statistically significant. DDT concentrations in seven other fish species (for which too few data are available to conduct a robust statistical analysis) are consistent with the trends observed in red shiners and mussels. The likelihood of 11 independent data sets showing a declining trend if a downward trend did not in fact exist is 1 in 2^9—i.e., vanishingly small.

Trends in DDT concentrations are evident in data collected for approximately 20 years in Newport Bay and Watershed. In the case of the fresh water fish species red shiner, tissue DDT concentration data are available from 1983 through 2002 (n = 54);

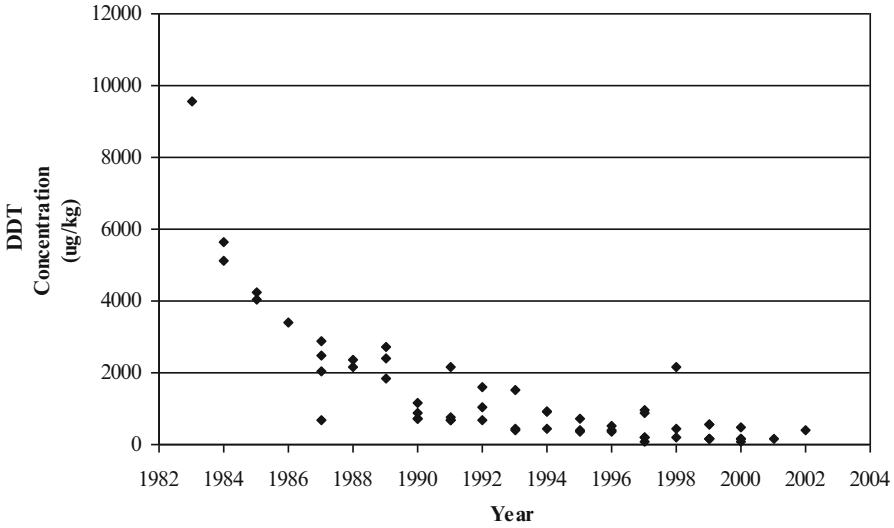

Fig. 4 DDT concentrations in red shiners from San Diego Creek and tributaries. Data from California Toxic Substances Monitoring Program (TSMP 1983–2002). Red shiner data are not available after 2002

the data are plotted in Fig. 4. Concentrations of DDT in red shiner are strongly indicative of concentrations within the Watershed, as this species has a short life span of approximately of 2 years (Baird and Girard 1853) and residents do not range outside of the fresh water streams flowing into Newport Bay.

Exponential regression was used to evaluate the strength of the declining trend in DDT concentration in red shiner tissue over time. A regression through the entire dataset indicated a highly significant exponential decline in DDT concentrations in red shiner tissue. The calculated rate of decline (without outliers) is −0.183 per year (equivalent to a DDT half-life in the watershed, as calculated from the surrogate endpoint of red shiner tissue, of 3.8 years), significantly shorter than the 20-year half-life typical for DDT decay in terrestrial soil. To confirm these trends, a regression analysis was performed for two 10-year sub-periods within the data set, 1983–1992 and 1993–2002, to evaluate whether rates of DDT loss in this species have changed over time. The rate of decline of DDT concentration in red shiners was lower for the later period (−0.135 per year) than for the earlier period (−0.245 per year), but both rates are highly significant.

Trends in DDT concentrations were also evaluated for seven additional fish species (California killifish, spotted sand bass, California halibut, diamond turbot, black perch, striped mullet, and yellow fin croaker) for which three or more DDT concentration data points were available during a time range of five or more years up until 2004. Although the data sets for any one of the additional fish species, taken alone, contained too few data points to infer long-term trends in Newport Bay, and in spite of possible sources of DDT for these fish outside of the Bay, the combined

datasets from these seven species are consistent with the strong trend evident in the red shiner data set. The combined evidence from all fish species lends far more weight to the conclusion that fish tissue DDT concentrations are declining in the watershed than could be concluded from data from any single species considered alone. As to the important point that some of these fish species may range outside the Bay to feed, and thus concentrations of DDT in the tissues of these fish species may reflect DDT obtained outside of Newport Bay, Allen et al. (2004) concluded that "monitoring studies are needed to determine if elevated DDT levels in the popular sport fishes noted above are due to contamination in the bay or to sources outside the bay." Also, in a follow-up study, Allen et al. (2008) found mean DDT levels in striped mullet from Newport Bay to be 499 ppb. As in the 2004 study (Allen et al. 2004), the authors caution that: "striped mullet (the species with the highest DDT levels) can leave the Bay and move up and down the coast. Its very high levels of DDT may be accumulated outside the Bay (perhaps in the Los Angeles/Long Beach Harbor area,"

Allen et al. (2008) also reported whole-body DDT levels in seven species of forage fish that are consumed by birds in Newport Bay. The species average DDT level was 143 ppb. The mean level was skewed by one species, the deep body anchovy, a nocturnal feeder that had an average whole-body DDT level of 495 ppb.

Like the red shiner, mussels are good bioindicators. Mussel tissue data from three locations in the Newport Bay Watershed—San Diego Creek, Upper Newport Bay, and Lower Newport Bay—were evaluated for trends in DDT concentrations over time. Like red shiner data, mussel tissue data collected since 1982 show statistically significant declines in DDT concentrations (Fig. 5). An exponential regression analysis of mussel data, including the entire period of record from 1982 to 1999,

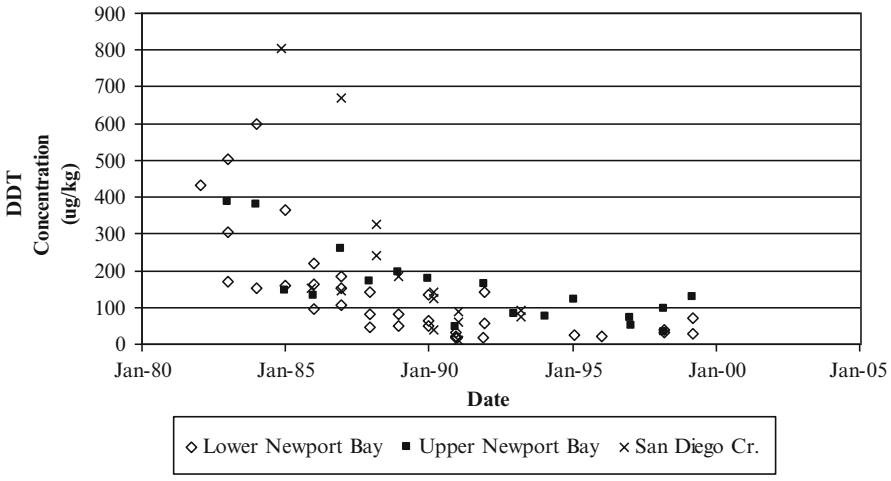

Fig. 5 DDT in mussels from Newport Bay and Watershed. Data from California Mussel Watch Program (1980–2000). The mussel watch program ended in 2000

showed a significant DDT concentration decline rate in mussels both when all three locations were considered together (−0.133 per year, equivalent to a half-life of 5.2 years), and for each individual location (San Diego Creek = −0.292 per year; Upper Newport Bay = −0.095 per year; Lower Newport Bay = −0.156 per year). Mussel data were also analyzed for the periods 1982–1990 and 1991–1999. The rate of decline of DDT concentrations in mussel tissue was statistically significant only for the earlier period (−0.236 per year). The rate of decline was lower for the later period, but period regressions for the later period have low statistical power due, in part, to small sample size. When the entire mussel tissue data set is considered, statistically significant rates of decline in DDT concentration are evident for each of the three locations, and these trends are consistent with trends observed in the fish tissue data sets.

However, as noted below, measured concentrations of DDT in red shiner and mussels indicate a half-life for DDT in biota of the watershed of 3.8 years and 5.2 years, respectively. These observed half-lives suggest that the fraction of DDT removed from the watershed as a whole since 1972 may be significantly higher than 70%, and declining concentrations of DDT in biota are likely from a combination of factors in addition to loss from soils.

3.1.5 DDT in Birds

Birds are exposed to DDT through their diet. Fish-eating birds typically attain the highest levels. DDT levels in fresh eggs correlate with the most sensitive toxic endpoint, eggshell thinning to the point of shell breakage, hatching failure and lowered productivity.

Limited data are available on DDT levels in bird tissue from Newport Bay and Watershed. In a 1984 internal memo, Harry Ohlendorf of the National Wildlife Service reported organochlorine levels in salvaged eggs from the endangered light-footed clapper rail; the eggs were collected during the period 1979–1981.[3] DDE levels ranged from 0.34 to 9.6 ppm. More recently, Sutula et al. (2005) reported total DDT levels of 0.45–1.07 ppm in nonviable eggs collected from light-footed clapper rail nests. The egg with the highest level had the thinnest shell. However, these more recent levels were less than those reported by Goodbred et al. (1996) for light-footed clapper rail eggs from the nearby Tijuana Slough, where eggshell thinning was not observed.

In 2004 and 2005, Gary Santolo at CH2M Hill collected eggs from six species of birds from Newport Bay and Watershed. DDE levels and shell thickness were measured in the eggs. Although the study has not as yet been published in the open literature, Santolo communicated the following general conclusions[4]: no correlation was found between shell thickness and DDE levels in American coot, black skimmer, Forster's tern, killdeer, or black-necked stilt eggs. However, there was a negative

[3] Richard Zembal, personal communication.

[4] Gary Santolo, personal communication.

correlation between DDE levels and shell thickness in an analysis of American avocet eggs, although the shell thinning was not within the range that would result in hatching failure.

The recent successful breeding of two osprey pair in Newport Bay implies that levels of DDT in the fish diet of this sensitive species are nontoxic. The breeding data are presented and discussed later in this review.

Many bird species resident to Newport Bay and Watershed have been studied in regard to the levels and effects of DDT at other locations. A detailed review of the effects of DDT on shell thinning and hatching success in the brown pelican, osprey, petrels, and sparrow hawk can be found in later chapters of this review, where effects from DDT in these species played a central role in establishing guidance levels for fish and water. The endangered species, the Belding's savannah sparrow and California gnatcatcher, are not known to be sensitive to the reproductive effects of DDT, most likely because these species feed in a food chain with a relatively low potential for bioaccumulation. The American coot (*Fulica americana*), eating mostly a vegetable diet, would also not be expected to be sensitive to the reproductive effects of DDT. The peregrine falcon is highly sensitive to the reproductive effects of DDT when consuming birds feeding in an aquatic environment. However, the lack of nesting sites (remote rock cliffs) proximate to Newport Bay and Watershed would preclude this food pathway. A possible exception is nesting on bridges and buildings. The following is a review of representative scientific studies of the levels in and effects of DDT on some additional bird species found in Newport Bay and Watershed.

DDT and Terns. There are many reports on DDT levels in tern eggs as well as associated measures of eggshell thickness, hatching success and productivity. Data from Forster's tern, common tern, Caspian tern and least tern are summarized below. The terns are closely related, providing a measure of susceptibility to DDT effects on reproduction for the genus and for the threatened tern species, the least tern.

King et al. (1991) measured DDE levels, eggshell thickness, and hatching success in Forster's tern (*Sterna forsteri*) eggs from two Texas bays. Seventy-one eggs were analyzed for DDE. One egg was taken per nest. Hatching success was monitored in the remaining eggs. DDE levels in eggs ranged from 0.1 to 9.0 ppm. The geometric mean concentrations of DDE in eggs from the two bays were 0.8 and 1.6 ppm. No correlation was found between the level of DDE in eggs and eggshell thickness or hatching success.

Ohlendorf et al. (1988) measured DDT levels in ten Forster's tern eggs from Bair Island in San Francisco Bay. The geometric mean concentration of DDT (all DDE) was 1.92 ppm with a range of 0.88–7.1 ppm. DDE levels were not correlated with eggshell thickness.

Vermeer and Reynolds (1970) reported DDE levels ranging from 2.0 to 25.2 ppm in ten egg composites from common terns (*Sterna hirundo*) in Central Canada in 1968–1969 at the height of the DDT era.

Switzer et al. (1971) reported a mean DDE level of 7.57 ppm in 68 eggs collected from a common tern colony in Alberta, Canada in 1969. DDE levels ranged from 0.64 to 104 ppm. Productivity of the colony was very low, but the authors found no correlation between DDE egg levels and eggshell thickness. A follow-up study in

1970 (Switzer et al. 1973) found higher productivity and lower levels of DDE. Mean DDE levels were 4.52 ppm with a range of 0.13–26.2 ppm. A weak negative correlation was found between eggshell thickness and DDE level.

Fox (1976) reported DDE levels in eggs from common terns nesting in Alberta, Canada. Thirty-nine intact eggs had a mean concentration of 3.42 ppm DDE. A pooled sample of five eggs with dented shells had a mean concentration of 6.67 ppm DDE. The average shell thickness of intact eggs was not different from common tern eggs collected prior to the DDT era; average shell thickness in the dented eggs was 12.5% thinner. One could conclude, based on these findings, that the threshold for egg shell thinning and hatching failure for common terns is greater than 3.42 ppm.

Nisbet and Reynolds (1984) also reported high levels of DDE in a small sample of common tern eggs that failed to hatch during the 1975 breeding season. DDE levels in the three highest eggs ranged from 1.8 to 4.6 ppm. However, in 1971–1972, when the highest levels of DDE were measured in their study, productivity was very high.

Ohlendorf et al. (1985) concluded from the work of Fox (1976) and Switzer et al. (1973) that: "In Common Terns, DDE contamination above 4 ppm was thought responsible for reduced eggshell thickness and quality and lowered hatching success." Since DDE levels range widely above and below the mean of 4 ppm and hatching failure only occurs in a fraction of the eggs, the threshold for hatching failure may be well above 4 ppm.

Weseloh et al. (1989) concluded that DDE was no longer an important factor in the population dynamics of common terns in the Great Lakes. Geometric mean DDE levels ranged from 0.95 to 2.46 ppm at four locations. Eggshell thickness did not correlate with DDE level.

Hoffman et al. (1993) measured geometric mean levels of 1.7–2.9 ppm DDE in eggs from four colonies of common terns in the Great Lakes. The DDE levels ranged from 0.60 to 5.0 ppm. Embryotoxicity observed in the eggs was attributed to PCBs and dioxins. DDE in the eggs was not considered to be sufficiently high to singly account for the observed embryotoxicity.

King et al. (1978) reported mean DDT levels in Caspian tern (*Sterna caspia*) eggs of 15.1 ppm, but no correlation between DDT level and eggshell thickness.

Struger and Weseloh (1985) reported mean DDE levels of 4.6–8.8 ppm in Caspian tern eggs collected in 1980 at various locations in Lake Michigan. Eggshells were at or above the thickness measured in eggs collected prior to the DDT era. The authors concluded that: "Organochlorine levels exhibited in Lake Michigan in 1980 do not appear to have had a detrimental effect on reproductive success in Caspian Terns."

Ohlendorf et al. (1985) reported a geometric mean level of 9.3 ppm DDE (ranging from 2.1 to 56 ppm) in Caspian tern eggs collected in San Diego Bay in 1981. More than one-third of the eggs were lost before hatching, mostly from failure to hatch. On average, eggshells were 7.8% thinner. The authors stated that: "We suspect that higher DDE concentrations in eggs from some nests were, at least in part, responsible for reduced hatching success." Since shell thinning and hatching failure are correlated with DDE levels in eggs, one would expect that the more than one-third hatching failure occurred in eggs with DDE levels above the geometric mean of 9.3 ppm.

Ohlendorf et al. (1988) reported geometric mean DDE levels of 6.93 ppm in 22 Caspian tern eggs from San Francisco Bay and 7.64 ppm in ten Caspian tern eggs from Elkhorn Slough in 1982. In comparing these levels with levels from other reports, the authors stated that: "Caspian Terns in the Great Lakes (Struger and Weseloh 1985) experienced good reproductive success when mean DDE concentrations in eggs were generally similar to—and PCBs generally higher than—concentrations we found in California."

Blus and Prouty (1979) concluded that geometric mean DDE levels in 44 least tern eggs (*Sterna antillarum browni*) from South Carolina in 1972–1975 were low (0.33–0.63 ppm with a range of 0.19–1.22 ppm) and posed no identifiable threat to the species. Eggshells were 2–7% thinner than shells of eggs collected prior to the DDT era.

Hothem and Zador (1995) reported an overall geometric mean of 0.936 ppm DDE in nonviable least tern eggs collected from San Diego Bay in the mid-1980s. Factors such as putrefaction and desiccation in nonviable eggs do not cause loss of DDT, but can produce changes in fresh weight, and therefore the concentration of DDT. Hothem and Powell (2000) sampled 14 least tern eggs from three locations in the San Diego area. The eggs were taken after the breeding season. Although not stated, these eggs also were most likely nonviable. Geometric mean concentrations of DDE in eggs from the three sites ranged from 0.23 to 0.56 ppm. Hothem and Powell (2000) concluded as follows: "Similar or higher mean concentrations in least terns (0.19–1.22 μg/g) from South Carolina (Blus and Prouty 1979) and in Forster's terns from Texas (1.6 μg/g) (King et al. 1991) were not thought to pose a threat to reproduction. Likewise, DDE should not pose a threat to either species in our study."

DDT and Cormorants. Eleven cormorant colonies were studied by Anderson et al. (1969) in the upper midwest and central Canadian provinces in 1965. DDE residue levels were as high as 45 ppm in cormorant eggs with an average of 10.4 ppm. Egg size, weight and thickness varied between the locations. Egg laying is a mechanism for excretion of DDT. Egg residues are more closely related to residues stored in lipid than recent dietary intake. Eggshell thickness was decreased 8.3%. Increases in shell thickness during rebreeding suggest that low DDT levels in local diets were more important than reductions in DDT from utilization of lipid stores during breeding. One population of cormorants, with a 25% decline in eggshell thickness, had recently decreased to nearly zero. The authors claim that the eggshell thinning-DDE regression is linear to zero concentration of DDE. A minimal effect level could not be established. Figure 6 illustrates the eggshell thinning dose-response in cormorants.

Faber and Hickey (1973) reported on a 1969–1970 survey of egg residues and eggshell thinning in fish-eating birds from the upper Great Lakes states and Louisiana. The authors suggest that significant decreases in shell thickness will be found in virtually all fish-eating birds in these parts of America. "We are uncertain about the biological significance of decreases in shell thickness below 10%. Certainly, widespread eggshell breakage does not occur with changes below this magnitude." The level of DDE residue necessary to cause eggshell thinning varies greatly among species. Double-crested cormorants developed eggshell thinning of approximately 12% at 17.5 ppm total organochlorine residues.

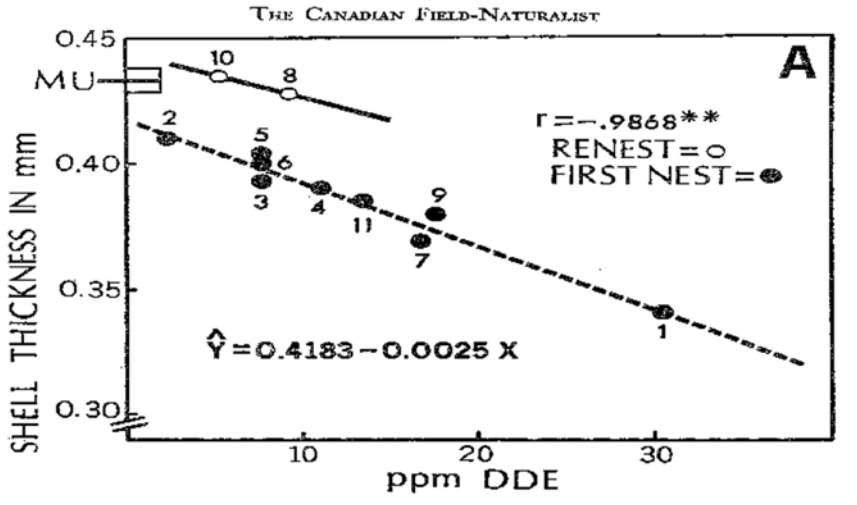

FIGURE 3. Relationships between DDE residues (A), estimated PCB residues (B), and shell thickness in Double-crested Cormorants, plotted on a colony-basis. Individual points are numbered in accordance with Figure 1. Open circles represent renest colonies (the original colony of the season was destroyed or disturbed away from the first-nest site, therefore, phenologically behind other colonies from the same general latitude and longitude) and closed circles represent first-nest colonies. "MU" in the upper figure represents the museum mean thickness (Table 5), bounded by 95% Confidence Limits. Figure 3A, $P<0.001$; Figure 3B, $P<0.01$. The line-of-fit for renests in A. was fitted by eye but clearly resembled the calculated regression based on individual pools. A line-of-fit for renests in B., though significant on an individual-pool basis, was not obvious on a colony basis.

Fig. 6 Eggshell thinning dose response in cormorants. Figure 3 in Anderson et al. (1969) reproduced with permission

Gress et al. (1973) reported on a survey of double-crested cormorant breeding colonies in the Channel Islands and the islands off of the west coast of Baja, California in 1969–1972. Breeding was almost nonexistent in colonies on the Channel Islands and South Los Coronados Island. Breeding appeared unaffected on San Martin Island farther south. No crushed eggs were found on San Martin. Eggshell thinning was 29% and 38% on Anacapa and Los Coronados, respectively. Gress stated that "The San Martin eggshells show no significant differences of any of the parameters from the museum specimens." DDE residues in eggs were 32 ppm, 24 ppm and 1.7 ppm on Anacapa, Los Coronados and San Martin, respectively. He noted that other studies on double-crested cormorants had not found reproductive impairment with DDE residues as high as 10.4 ppm. DDE was associated with 8.3% eggshell thinning. He concluded that "The comparatively low levels of DDE reported suggest that the degree of thinning, if present, would not be sufficiently great to affect reproductive success." Comparisons with studies of interior populations indicated that the relationship between DDE residues and eggshell thinning were the same. In addition, 80% of the variation in eggshell thickness could be explained by the regression on the natural log of DDE. The 1972 survey suggested that both the brown pelican and double-crested cormorant were beginning to recover.

The recovery was attributed to the fact that the DDT manufacturing plant in Los Angeles stopped discharging wastes to the Los Angeles outfall in April, 1970.

Morrison et al. (1978) reported on DDE residues and shell thickness in cormorant eggs collected in Texas in 1976–1977. The results were compared with an earlier study by King, in which cormorant eggs were collected in 1970. The results of the King study were provided by personal communication to the authors from K. A. King. DDE residues had declined dramatically in cormorant eggs from 1970 to 1976–1977 (Table 3).

Eggshell thickness was not significantly affected in either the 1970 or 1976–1977 studies, although the latter shells were thicker (Table 4).

The authors concluded that there was little difference in thickness between the pre-DDT era shells, the 1970 shells and the 1976–1977 shells. "Most authors agree that a 10–20% change in shell thickness is needed before reproductive failures are indicated." and "Cormorant eggshell thickness was apparently not affected by residues in the 1970s in Texas."

Pearce et al. (1979) reported DDE residues in cormorant eggs collected along eastern Canadian coastal waters from 1970 to 1976. Average residues by site ranged from 1.49 to 8.57 ppm. Individual eggs contained from 0.16 to 20 ppm DDE. The authors reported measuring shell thickness, but no data were present in the publication. The authors claimed that 10 ppm DDE in eggs produces 20% shell thinning. This conclusion was based on an extrapolation of the residue—shell thinning data. Again, no data or regression plots were present in the article.

Table 3 Marked reduction in DDE in cormorant eggs from Texas from 1970 to 1976–1977. Data from Table 1 in Morrison et al. (1978)

Residues in olivaceous cormorant eggs in Texas[a]							
	1970 (n = 5)			1976–1977 (n = 7)			
Residue	Mean	S. E.	(%)	Mean	S. E.	(%)	% Change
p,*p*′-DDE	6.22	2.08	100	0.400	0.036	100[b]	−93.6

[a]Values represent residues on a wet-wt basis
[b]$p < 0.05$

Table 4 Shell thickness in cormorant eggs collected in Texas. Table 2 in Morrison et al. (1978) reproduced with permission

TABLE 2

SHELL THICKNESS OF OLIVACEOUS CORMORANT EGGS IN TEXAS (MM)

Date	n (eggs)	$\bar{x}$	S.E.	% Change from Pre-1940	% Change from 1970
Pre-1940	75	0.328	0.004	—	—
1970	24	0.323	0.006	−1.5	—
1976–77	21	0.341	0.004	+4.0*	+5.5*

* $p < 0.05$, t-test.

Weseloh et al. (1983) reported on the status of double-crested cormorant colonies in Lake Huron. Six colonies were studied in 1972 and 1973. DDE residues in eggs averaged 14.5 ppm. Eggshell thickness was reduced an average of 23.9%. Egg breakage, hatching failure, and population declines were evident.

Fossi et al. (1984) reported high levels of DDE in cormorant eggs collected from the Danube Delta. DDE levels in 13 eggs averaged 9 ppm. Eggshell thickness was not measured. The authors noted that: "Despite the heavy contamination of the eggs, however, the population of the colonies of Common Cormorant seems to have stabilized…".

King and Krynitsky (1986) studied cormorants nesting in Galveston Bay from 1980 to 1982. DDE levels in eggs averaged 1.73 ppm in 1980 and 0.67 ppm in 1981. Mean shell thickness for the period 1980–1982 was similar to eggs collected prior to the DDT era. Eggs collected from Galveston Bay in 1970 (King et al. 1978) were 7% thinner; eggs collected in 1980 were 5% thinner; eggs collected in 1981 were 3% thinner; eggs collected in 1982 were 1% thicker. The 3% and 1% effects were not statistically significant. One eggshell from an egg collected in 1980 was 22% thinner than in pre DDT era eggs. Although not indicated by the authors, this egg may have contained the highest residue measured in the 1980 eggs (N = 13). That level was 31 ppm DDE. The authors noted that cormorant populations had remained stable in recent years.

Dirksen et al. (1995) reported a detailed study of organochlorines in cormorants in the Netherlands. Reproductive effects of DDE were confounded by high levels of PCBs in adult tissue and eggs. However, the authors concluded that 4 ppm DDE in cormorant eggs produced 5% shell thinning. They also noted that the threshold for population reproductive failure and population instability was associated with shell thinning of 20%. This level of thinning was associated with egg residues of 10 ppm.

In 1998, the US Department of the Interior published a National Irrigation Water Quality Program Information Report No. 3 titled: "Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment. DDT." The US Fish and Wildlife Service participated and presumably wrote the section on toxicity to avian species. According to the report, "Toxic effect levels for various types of birds are presented in Table 16." Beginning on page 70, Table 16 lists various avian species, the DDTs studied, the concentration in eggs, the effects observed, and the reference. For the double-crested cormorant, a concentration of 10 ppm of DDE in eggs was stated to cause 20% shell thinning. The reference for this data point is the Pearce et al. (1979) article discussed above. This study claims to have measured shell thinning and to have correlated the shell thinning with DDE residues. However, no shell thinning data and regression plots are to be found in the publication. Hence, this data point in the Department of Interior review is based only on a statement without data or analysis. Comparison of other data points in Table 16 with the referenced article revealed errors and misinterpretations.

For example, Table 16 lists 1 ppm DDE in Western grebe eggs as causing 1% shell thinning. The DDE concentration reported in the cited study was 1.4 ppm, not 1 ppm (Boellstorff et al. 1985). The 1% was reported by Boellstorff et al. (1985) to not be statistically significant. The authors concluded: "Thickness of grebe eggshells

collected at Tule Lake NWR in 1972 and 1981 and in northern California from 1952 to 1960 were not significantly different from each other and were not thinner than eggs collected before 1947 (Table 4)."

The very next line in Table 16 states that 5.4 ppm DDE caused 2.3% eggshell thinning and reduced productivity. The research article cited for this data point (Lindvall and Low 1980) reported a DDE residue of 6.6 ppm and a thinning of 3.1%. The authors did not conclude that productivity was reduced. To the contrary, the authors concluded: "The small amount of eggshell thinning seen in western grebe eggshells at Bear River MBR appeared to have little or no effect on reproduction, because no crushed, cracked, or broken eggs were seen during this study. Average brood sizes of 1.6 in 1973 and 1.8 in 1974 from Bear River compare well with the Rudd and Herman determination of a normally reproducing population (18)."

The Department of the Interior report also states in Table 16 that less than 1 ppm DDE produced 6.5% shell thinning in black-crowned night-herons. The reference for this data point (Findholt and Trost 1985) reported a linear regression of shell thickness and log DDE egg residue that had a zero residue intercept of 0.26 mm. Since pre-DDT era shells in this study were 0.275 mm, the linear regression is likely to be inaccurate, particularly at low residue levels. A similar phenomenon has been reported in brown pelican studies. The obvious fallacy in the Table 16 listing is made clear by the fact that eggs containing 1.01–4.0 ppm DDE had thicker shells than eggs with less than 1 ppm DDE.

Table 16 states that 0.52 ppm DDE in common goldeneye eggs causes 15.4% shell thinning and egg breakage. The 15.4% shell thinning is a comparison of 1981 Minnesota colonies with North Dakota and Manitoba eggs collected in 1896 and 1903. The authors (Zicus et al. 1988) conclusion on egg breakage is as follows: "The high rate of egg breakage observed for Common Goldeneyes may be related to eggshell thinning or may be characteristic of the species and perhaps a result of frequent nest parasitism."

Finally, Table 16 states that 12 ppm DDE in Leach's storm petrel eggs results in 12% eggshell thinning. The cited reference (Noble and Elliott 1990) reports only on raptors and makes no mention of Leach's storm petrel. In the few data points in Table 16 that were checked against the original publications, the Department of Interior report repeatedly made errors and misrepresentations of the literature findings on the effects of DDT on avian reproduction.

Custer et al. (1999) reported on cormorant colonies on Cat Island in Green Bay, Wisconsin. Eggs contained 3.9 ppm DDE and 13.6 ppm PCBs. DDE concentration correlated with decreased shell thickness and hatching failure (thinning data were not reported). However, the authors concluded that reproductive performance was generally good to excellent compared to other locations, including those considered to have low levels of persistent organochlorine contamination. "Number of young produced (2.0–2.3 to 12 days of age) was also similar or greater than the 0.7–2.5 young per nest reported in relatively uncontaminated colonies." "…DDE-contamination does not seem to be a significant risk factor to double-crested cormorant populations in this region." A low level of chick deformities was not attributed to DDE.

Cormorants are less sensitive to the reproductive effects of DDTs than ospreys and brown pelicans. Residues of DDE in eggs in excess of 10 ppm, resulting in eggshell thinning of 15% or more, appear to be necessary to produce significant hatching failure.

DDT and Black Skimmers. The black skimmer (*Rynchops niger*) feeds in estuaries, catching small fish by skimming the water surface with its lower mandible; maximum lifespan is 20 years (US GS 2010). King et al. (1978) reported 4% eggshell thinning in black skimmer eggs collected in 1970. Mean DDT levels in the eggs were 9.68 ppm. No correlation was found between DDT levels and eggshell thickness.

Blus and Stafford (1980) reported that DDE ranged from 0.81 to 12.1 ppm in eggs from black skimmer nests that apparently failed. Eggs from successful nests contained 0.43–3.40 ppm. Overall eggshell thinning was 5%. The authors concluded that DDE and other pollutants had little influence on overall productivity of black skimmers in South Carolina.

White et al. (1984) reported that 35% of black skimmer eggs collected in 1978–1981 along the Texas coast contained DDE levels of 10 ppm or more (levels of DDE ranged as high as 51 ppm), but no significant correlation was found between residue levels and fledgling success. The authors state that: "Some degree of eggshell thinning was detected in most colonies (9/10), ranging from 4% to 12%, but thinning was below that (15–20%) believed to cause population declines in other avian species (Anderson and Hickey 1972; Blus et al. 1972a; Longcore and Stendell 1977)."

King and Krynitsky (1986) assessed DDT residues, shell thinning and reproductive success in black skimmers along the Texas coast in 1980–1982. Geometric mean DDE levels in eggs were 1.62–3.25 ppm (range of 0.2–86 ppm). Eggshell thinning for the years 1980, 1981 and 1982 occurred to the extent of 2–6%. Some cracked or crushed and broken eggs were observed to be up to 36% thinner than shells of eggs collected prior to the DDT era. Overall, DDE level did not correlate with shell thickness.

Custer and Mitchell (1987) reported on a study of black skimmers along the Texas coast in 1984. Geometric mean DDE levels were 5.9 ppm (range of 2.3–17.9 ppm) in eggs from nests where the remaining eggs did not hatch and 1.9 ppm (range of 0–7.4 ppm) in eggs from nests where all of the remaining eggs hatched. DDE level did not correlate with shell thickness. The authors concluded that: "The breeding population of Black Skimmers in Texas does not seem to be declining nor does DDT contamination seem to be a major influence on skimmer numbers." In a follow-up publication, King et al. (1991) reported that: "we found no evidence that shell thinning of either tern (7%) or skimmer (5%) eggs adversely affected reproduction in 1984."

DDT and Black-necked Stilts. Setmire et al. (1993) collected 84 black-necked stilt (*Himantopus mexicanus*) eggs from the Salton Sea area in 1988–1990. The geometric mean concentration of DDE was 2.57 ppm with a range of 0.05–12.1 ppm. Eggshell thinning was estimated to be 7% at 12 ppm DDE from a plot of DDE egg residue against eggshell thickness from an Imperial Valley data set of 33 black-necked stilt

eggs. However the relationship between DDE level and eggshell thinning was not statistically significant.

Henny et al. (1985) reported that 40 black-necked stilt eggs from Carson Lake, Nevada contained from 0.31 to 15.6 ppm DDE. DDE levels did not correlate with eggshell thickness and shell thickness was similar to that of pre-DDT eggs. The authors concluded that: "No significant relationship was detected between DDE and eggshell thickness for stilts." They noted that Morrison and Kiff (1979) had reported only 1.9% eggshell thinning in stilts from Utah in 1959.

Henny et al. (2008) reported on DDE levels in black-necked stilts collected from the Salton Sea area from 1986 to 2004. Geometric mean levels ranged from 0.55 to 2.91 ppm by colony location, with individual values ranging from 0.05 to 23 ppm. The lowest levels were encountered in 2004, with a geometric mean concentration of DDE of 0.55 ppm and a range of 0.19–1.7 ppm. Eggshell thickness was no different than in stilt eggs collected in Utah prior to the DDT era. The authors concluded that: "The DDE concentrations in eggs documented in this and other studies seem not to produce eggshell thinning in stilts and the associated adverse effect on reproductive success."

DDT and the American Avocet. The American avocet (*Recurvirostra americana*) and black-necked stilts are closely related species. Vermeer and Reynolds (1970) reported 3.32 and 3.16 ppm DDE in ten egg composites of avocet eggs collected in central Canada in 1968–1969. Hunt (1969) reported that avocet and killdeer eggs from the Sacramento Valley averaged 13 ppm DDE.

Henny et al. (2008) reported DDT levels and egg shell thickness in three avocet eggs collected in 2004 from the Salton Sea area. The geometric mean DDE level was 1.14 ppm with a range of 0.83–2.1 ppm. Although shell thickness was measured, eggshell thinning was not assessed because no comparison measurements were available from eggs collected prior to the DDT era. According to Henny et al. (2008), Setmire et al. (1993) had concluded that the risk to American avocets for DDE in the Salton Sea area was not high in the 1988–1990 period. Black-necked stilt eggs collected in the same area as the American avocet eggs declined in DDE concentration from 2.91 to 0.55 ppm from 1993 to 2004. Based on this limited and indirect assessment, low ppm levels of DDE in American avocet eggs do not appear to be of reproductive concern among scientists studying the effects of DDT on avian wildlife.

DDT and Killdeer. No studies were found that related DDT levels in killdeer eggs to eggshell thinning or reproductive success.

Discussion and conclusions. There is a wide range of DDT levels seen in individual eggs collected from the same species and location. Authors have attempted to explain the wide variability in avian egg DDT levels. Important factors appear to be the age of the bird, level of contamination in feeding grounds, prey selection and nutritional status. Since avian wildlife accumulate DDT throughout their lives, older birds have higher body burdens and lay eggs with higher levels of DDT. Most species considered here do not reproduce until the third or fourth year of life. Therefore, dietary sources of DDT in the first 3–4 years of life will be the major

source of DDT in eggs during the first year of reproduction. DDT in eggs thereafter will increase as a result of dietary DDT accumulated during each subsequent year. Egg laying and starvation can partially offset these life-long increases.

For Newport Bay, a hen laying eggs having DDT levels at the bottom of the range is probably in its first year of reproduction and has, by feeding location and prey selection, not been exposed to significant levels of DDT. These hens are most likely feeding several miles out in the Pacific Ocean in an area far from DDT contamination.

A hen laying eggs having DDT levels at the top of the range is probably an older bird (some live up to 20 years) with a history of frequenting locations and consuming prey that are high in DDT. In Southern California, the highest DDT levels in fish are found off of the Palos Verdes Peninsula. For several miles around the Los Angeles County Sanitation Districts outfall, the ocean bottom and aquatic biota have been contaminated by DDT manufacture wastes. The next highest levels are found in rivers draining from Mexico into California. The Alamo and New Rivers that drain into the Salton Sea and the Tijuana River that drains into the Pacific Ocean are examples. These rivers drain agricultural lands in Mexico where DDT was used long after the U.S. ban in 1972. In addition, the continuing use of DDT to control malaria along the southwestern coast of Mexico is also a significant exposure location. The next highest levels occur in rivers, estuaries and bays that receive agricultural drainage in California. Newport Bay is such an example, although for the Newport Bay Watershed the downward trend in DDT releases has been accelerated in recent decades by the conversion of agricultural lands to urban uses, a change that reduces the erosion of soils where DDT was historically used. The least contaminated aquatic biota can be found in areas of the Pacific Ocean not associated with contaminated effluents and in estuaries that do not receive drainage from lands containing DDT residues from past agricultural and other applications. Most of the fish-eating avian species resident to Newport Bay and Watershed are migratory, so all of the above exposure scenarios are possible.

When interpreting studies on the effects of DDT on avian reproduction, one must be aware of the wide range of egg residue levels. Average egg residue levels, average shell thinning and average hatching failure do not reveal the full range of effects (i.e., from no effect to severe effect) that can occur in individual birds at the same location and in the same data set. Thresholds should be estimated from the full range of effects in individual birds and not from the average effect for a particular study. For example, the average egg residue of 4 ppm DDT may result in a 30% hatching failure. Examination of the individual data may reveal that the hatching failure only occurred at egg levels above 10 ppm, so the threshold for hatching failure is closer to 10 ppm than 4 ppm.

Since the U.S. ban of DDT in 1972, residues in the aquatic environment and in bird eggs have declined to levels that, in most locations are below thresholds for eggshell thinning and in almost all locations, below thresholds for hatching failure.

The CH2M Hill study found that DDT levels in eggs of six avian species were below thresholds for hatching failure, with perhaps minimal egg shell thinning in one species. The lack of a correlation of DDT levels with shell thickness in Forster's

tern suggests that the closely related and endangered least tern is not being affected by DDT. The IAP to the Organochlorine TMDL for Newport Bay and Watershed has recommended (IAP 2009) the least tern as a sensitive indicator for the potential toxicity of DDT to wildlife in Newport Bay.

3.1.6 DDT in Marine Mammals

In April 2006, and again in December 2013, a comprehensive review of the scientific literature was undertaken to assess what is currently known regarding the effects of DDT in marine mammals, either resident to, or capable of visiting, Newport Bay, California. The first step of the review was to determine the species that should be included. Although there are numerous marine mammal species found in the northwestern Pacific Ocean, relatively few species reside in, or visit, Newport Bay.

Those that may potentially reside in the area for significant periods include the California sea lion (*Zalophus californianus*) and harbor seal (*Phoca vitulina*). Those species that may enter Newport Bay for at least short periods—an unlikely but conservative approach—include the Pacific bottlenose dolphin (*Tursiops gilli*), rough-toothed dolphin (*Steno bredanensis*) and common dolphin (*Delphinus delphis*), and two filter-feeding baleen whale species—the minke whale (*Balaenoptera acutorostrata*) and the migratory gray whale (*Eschrichtius gibbosus*; Ingles 1965; Burt and Grossenheider 1976).

Therefore, electronic database searches were conducted via both the ISI Web of Science and BIOSIS Previews using the following topical keywords:

Seals and DDT
Sea Lions and DDT
Dolphins and DDT
Whales and DDT

Several hundred documents dating from the mid-1960s through 2013 were identified. However, most involved species not relevant to the Newport Bay region (i.e., not listed above). Notwithstanding, a significant number of reports were identified and are summarized below. Although no search can necessarily identify and locate all publications on a topic, those summarized below provide a reasonable summary of what is currently known regarding DDT in marine mammals that may either reside in or visit Newport Bay.

One important factor to consider in this review is the virtual lack of publications encountered that describe the toxic actions or endpoints of DDT in the subject marine mammals. There are two key reasons for this. First, logistically marine mammals are very difficult to directly utilize in the statistically-significant numbers needed for valid potency or other mechanistic investigations. Although sea otters may only weigh a few pounds, whales are excessively large and not practical to handle or house. Second, marine mammals have been protected by the United States Government for many years, which has significantly reduced access for any purpose,

including research. Therefore, nearly all the papers published to date involve the measurement of DDT residues in tissues obtained from either live or dead (stranded and often decaying) animals. Such information can at least give an approximate estimate of the residues encountered by the subject marine mammals—and their ability to accumulate them. The following is a brief summary of the published reports involving DDT in marine mammals of importance to Newport Bay.

The summary is a chronology by species of DDT studies in marine mammals. DDT concentrations in these studies are reported as total DDT, which typically represents the sum of anywhere from three (*p*,*p*′-DDT + *p*,*p*′-DDD + *p*,*p*′-DDE) to six (*o*,*p*′-DDT + *o*,*p*′-DDD + *o*,*p*′-DDE + *p*,*p*′-DDT + *p*,*p*′-DDD + *p*,*p*′-DDE) analytes. Unless otherwise indicated, all residue values reported below are based on wet sample weight—concentrations reported on a lipid weight basis can average four or more times higher than those reported on a wet weight basis. Also note that while many reported values are geometric means (delineated below), some are arithmetic means.

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

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

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

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

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

Routti et al. (2008) compared DDT levels in gray and ring seals in the more contaminated Baltic Sea compared to lower contaminated sites in Canada and Norway. They reported that changes in circulating vitamin D and thyroid hormone were associated with DDT and PCB levels in liver, suggesting that bone lesions observed in Baltic gray seals may be caused by DDT and/or PCBs. Another possible explanation for their findings is that animals stressed by factors unrelated to body burdens of DDT will result in weight loss with the mobilization of fat stores of DDT. The mobilization of DDT from blubber to liver could result in a negative correlation between DDT levels in the liver and the effects of the unknown factor, even though DDT may not be at a level that is having any toxic effect.

Bredhult et al. (2008) did not find an association between DDT in blubber and uterine leiomyomas in Baltic gray seals.

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

More recently, Kajiwara et al. (2001) reported DDT concentrations (based on lipid weight) in the livers of ten stranded harbor seals collected between 1991 and 1997; the geometric mean concentration was 12 ppm (range, 2.8–85 ppm).

Greig et al. (2011) reported DDT levels (lipid basis) of 320–1,500,000 ppb in blubber from 202 stranded and wild-caught harbor seals in the central California coast. The highest levels were in pups during the post-weaning fast, suggesting that fasting pups may be the most vulnerable early life stage to the toxic effects of DDT, due to mobilization from lipid stores.

Hall et al. (2009) reported a negative correlation between blubber concentrations of DDT and survival in first year gray seal pups sampled from the Isle of May in 2002. Geometric mean DDT levels (lipid basis) were 229 ppb. The authors were careful to note that their finding did not indicate causation. Causation would seem unlikely considering the relatively low concentrations of DDT in the pups.

Fillman et al. (2007) reported 20–2,480 ppb DDT (mean level of 660 ppb) in blubber from stranded juvenile South American fur seals. The authors point out that the poor nutritional status of the seals is likely to have mobilized DDT from blubber to other tissues, increasing their vulnerability to possible toxic effects.

Roos et al. (2012) reported a decline in DDT residues in seal blubber from 192 to 2.8 ppm (lipid weight basis) from 1973 to 2010. Increases in uterine health and pregnancies and reduced uterine cancer were associated during the same period

with declining residues of persistent organochlorines, including DDT, suggesting possible causation.

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

Wang et al. (2007) reported relatively low levels of DDT in harbor seals from the Gulf of Alaska. Blubber samples contained 78–325 ppb with a mean level of 159 ppb. The authors point out that levels in nursing females were much lower than those in male adults, due to lactation transfer of DDT from mother to newborns.

Sakai et al. (2006) reported on an assay for androstane receptor activity in Russian seals. The authors concluded that DDT was active in their assay and that the lowest observable effect level was comparable to a 10 ppm tissue level.

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

DDT and Sea Lions. There are several reports of DDT in sea lions (*Z. californianus*) residing along the California coast. In 1971, Le Boeuf and Bonnell published a seminal report of blubber concentrations in California sea lions collected in 1970 (n = 12), a full 2 years prior to the banning of the use of DDT in the U.S. In it, they reported high concentrations for both DDT (geometric mean, 17 ppm; range, 8.8–34 ppm) and DDE (geometric mean, 740 ppm; range, 370–1,500 ppm).

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

In 1995, Lieberg-Clark et al. followed up on the above 1971 report of Le Boeuf and Bonnell by measuring ΣDDT and ΣDDE concentrations in blubber from seven California sea lions sampled between 1988 and 1992. Their numbers clearly indicated a significant decline (greater than 99%) in residues over the 20-year time span for both DDT (geometric mean, 0.16 ppm; range, 0.07–0.35 ppm) and DDE (geometric mean, 5.0 ppm; range, 2.5–10 ppm). Therefore, they concluded the following:

1. The decline in the residue levels in California sea lions over this period was accompanied by a significant increase in their population during the same time period.

2. The extremely high ΣDDT concentrations reported in the 1970s may have been associated with reproductive problems in California Sea Lions.
3. The decline in ΣDDT residues in California sea lions was so dramatic because their breeding area in southern California was much less contaminated with DDT residues than in 1970.

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

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

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

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

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

More recently, two studies designed to correlate toxic actions with DDT in California sea lions have been published. Debier et al. (2005) investigated a possible relationship between DDT in the serum of 12 healthy California sea lions and circulating levels of vitamins A and E and the thyroid hormones thyroxine (T4) and triiodothyronine (T3). Although several negative correlations were reported for PCB, only vitamin A was significantly correlated with DDT, and only when concentrations were reported on a lipid weight basis.

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

Blasius and Goodmanlowe (2008) reported that DDT levels in blubber collected from marine mammals in the southern California bight were higher in resident harbor seals and sea lions than in the transient northern elephant seal. Adult female sea lions had lower residue levels than pups, yearlings and adult males. DDT levels in sea lion blubber declined approximately tenfold to a mean (lipid weight basis) of approximately 200 ppm from 1994 to 2006, but not in the transient northern elephant seal that was less impacted by the high levels of contamination attributed to production wastes released prior to 1970. The highest concentrations of DDT in blubber, as high as 13,271 ppm (lipid weight basis), were measured in stressed sea lions that had lost almost all of their blubber.

In contrast to the high blubber residues of DDT in the highly contaminated southern California bight, blubber from sea lions stranded along the Baja California coast in 2000 and 2001 had residues averaging 3.8 ppm on a lipid weight basis (Del Toro et al. 2006). Nino-Torres et al. (2009) reported a mean DDT level of 3.4 ppm (lipid weight basis) in blubber collected from sea lions in 2005 and 2006 in the Gulf of California.

Ramsdell (2010) reported a novel zebra fish model for the interaction of DDT and the diatom poison, domoic acid, in sea lions feeding in the highly contaminated Channel Islands of the southern California bight. Pretreatment of embryonic zebra

fish with levels of DDT comparable to those found in more highly contaminated sea lion fetuses, increased the neurological response to domoic acid. This model suggests that high residues of DDT, typically found in sea lions residing near the Channel Islands may increase susceptibility to domoic acid poisoning.

In summary, several studies have confirmed the presence of DDT in California sea lions. Hence, they accumulate DDT primarily via biomagnification by feeding on contaminated fish, as do seals. DDT concentrations have generally been reported in the parts-per-million range, but have been on the decline in recent years from discontinuation of its use. The highest DDT level in sea lions world-wide occurred in the Channel Island area of the southern California bight as a result of past dumping of DDT manufacture wastes there. Similar to harbor seals, toxic effects from DDT in California sea lions have yet to be conclusively demonstrated.

DDT and Dolphins. Dolphins and porpoises are not likely to spend much time (if any) in Newport Bay. However, to be conservative they have been included in this review. There are relatively few published reports of DDT in dolphins and porpoises that might be relevant to Newport Bay. In 1980, O'Shea et al. reported DDT in the blubber, brain and muscle tissues of 69 small cetaceans, including one Pacific bottlenose dolphin (*T. gilli*) that had an excessively high blubber DDT concentration of 2,695 ppm.

Smyth et al. (2000) reported concentration ranges of DDT in the blubber and liver of six common dolphins (*D. delphis*) accidentally caught in fishing nets off the coast of Ireland to range from 3,998 to 9,444 ppb and 2,293 to 4,528 ppb, respectively. In 2001, Borrell et al. reported the DDT concentrations measured in the blubber of common dolphins accidentally caught in fishing nets along both the Atlantic and Mediterranean coasts of Spain during a 12-year time span. In dolphins from the Atlantic, mean DDT concentrations (lipid weight basis) declined significantly between 1984 and 1996 (1984: 15.54±8.82 ppm; 1996: 7.95±4.49 ppm). In dolphins from the Mediterranean, mean DDT concentrations of animals sampled in 1992 through 1994 was 33.40 ± 38.64. Of note was the fact that males in both regions accumulated significantly higher concentrations than females. In a follow-up study, Borrell and Aguilar (2007) reported again on DDT levels in dolphins from Spain's Mediterranean coast. Levels continued to decrease in bottlenose dolphins, a reduction from 303 to 13 ppm lipid weight basis from 1978 to 2002. The ratio of *p*,*p*′-DDE to *p*,*p*′-DDT continued to increase, suggesting the continued breakdown of DDT with the absence of new releases into the environment.

Castrillon et al. (2010) reported a sixfold decrease (from approximately 400–70 ppm lipid weight basis) in blubber DDT in striped dolphins (*Stenella coeruleoalba*) dying from two Mediterranean epizootics, due to morbillivirus in 1990 and 2007. The second epizootic was not believed to have been enhanced by DDT or PCBs residues in the dolphins. Wafo et al. (2012) reported an average DDT level (lipid weight basis) of 16 ppm in blubber collected in 2007–2009 from striped dolphins stranded along the French Mediterranean coast. Shoham-Frider et al. (2009) reported DDT levels of 0.92–141 ppm in blubber from bottlenose dolphins stranded in 2004–2006 along the Israeli coast.

Das et al. (2006) pointed out that even though DDT levels in blubber correlated with thyroid fibrosis in harbor porpoises from northern Europe, this type of association was insufficient to establish a cause-effect relationship. DDT levels (lipid weight basis) were highest (viz., 1,481–2,292 ppb) in the more industrialized areas and were lowest at 1,122 ppb in the less industrialized Icelandic coast.

Siebert et al. (2011) found no correlation between stress hormones and DDT levels in blood collected in 1997–2002 from free-ranging and captive harbor porpoises (*Phocoena phocoena*) from the North and Baltic Seas. Geometric mean DDT levels in blood ranged from 0.2 to 8.2 ppm on a lipid weight basis. Weijs et al. (2010) reported a decline in DDT in tissues of harbor porpoises in the southern North Sea during the period 1990–2008.

Fair et al. (2010) reported levels of DDT (lipid weight basis) in blubber biopsies taken from bottlenose dolphins in 2003–2005 along the southeastern US coast. Adult males were highest at a geometric mean level of 29 ppm for Charleston, South Carolina. At the same location, adult females were lowest at 3.0 ppm with juvenile dolphins intermediate at 14.7 ppm. Litz et al. (2007) measured DDT in dolphin blubber biopsies taken in Biscayne Bay, Florida in 2002–2004. The geometric mean level in adult males and juveniles was 2.98 ppm, whereas the geometric mean level in adult females was lowest at 0.097 ppm.

Lailson-Brito et al. (2011) reported DDT levels (lipid weight basis) of 264–5,811 ppb in blubber of franciscana dolphins stranded along the Brazilian coast. In a follow-up study, Lailson-Brito et al. (2012) measured DDT levels in blubber from four species of dolphins. Much higher *p*,*p*′-DDT/*p*,*p*′-DDE ratios were measured in blubber from Fraser's dolphins that feed in deep open water, where slower breakdown of *p*,*p*′-DDT to *p*,*p*′-DDE would be expected. The blubber of Fraser's dolphins had relatively low residue levels of 0.99 ppm DDT. Higher levels of DDT at 5.0 ppm, 26.4 ppm and 2.4 ppm were measured in bottlenose, rough-toothed and long-beaked common dolphins, respectively, that inhabit estuarine and coastal waters that are more contaminated from DDT agricultural use.

Law et al. (2013) reported DDT levels in blubber from 43 female short-beaked common dolphins (*Delphinus delphis*) bycaught in fisheries during the period 1992–2006 off the southwest coast of the UK. Levels ranged from 0.2 to 16.1 ppm on a lipid weight basis. DDT levels declined during the period of investigation although the trend was not statistically significant.

Stockin et al. (2007) reported DDT levels in blubber from common dolphins in Hauraki Gulf, New Zealand from 1999 to 2005. Levels ranged from 17 to 337 ppb in females and 654–4,430 in males. In follow-up studies, Stockin et al. (2010) reported on levels of DDT in Hector's dolphin (*Cephalorhynchus hectori hectori*) and Maui's dolphin (*Cephalorhynchus hectori maui*) from 1997 to 2009 from all parts of New Zealand. DDT in blubber of females ranged from 94 to 8,210 ppb with a mean of 1,358 ppb, whereas DDT in male blubber ranged from 252 to 57,390 ppb with a mean of 12,400 ppb. DDT residues in individual dolphins reflected the proximity of their habitat to agricultural and industrial releases of DDT.

Wu et al. (2013) reported levels of DDT ranging from 0.845 to 179 ppm (with a mean of 64.2 ppm) in blubber from Indo-Pacific humpback dolphins (*Sousa chinensis*)

stranded in 2004–2009 in the Pearl River Delta in China. Seventy five percent of the DDT residue was *p*,*p*′-DDT, suggesting fresh releases of DDT. The authors note that DDT is still being used in China for control of malaria.

In summary, there are few reports of DDT concentrations in dolphins or porpoises important to the Newport Bay region. Those above are for animals sampled elsewhere in the world. Although they demonstrate the ability of several species of dolphins to accumulate DDT and its degradation products, the actual concentrations may not reflect what would occur in animals residing on the California coast. Similar to harbor seals and California sea lions, toxic effects from DDT in the subject dolphins have yet to be conclusively demonstrated via controlled studies.

DDT and Whales. Although whales (baleen or toothed) are not likely to spend time in Newport Bay, to be conservative, we summarize the pertinent publications involving DDT and the whale species most likely to, at least, briefly visit the area. Over the years, many studies have reported on the contaminants present in the blubber of baleen whales, including gray and minke whales. For instance, in gray whales (*E. gibbosus*) Wolman and Wilson (1970) measured DDT concentrations as high as 680 ppb in some 23 animals collected between 1968 and 1969, and Schafer et al. (1984) reported a concentration of 470 ppb in a single animal sampled in 1976. In 1994, Varanasi et al. reported the concentrations of DDE in the tissues and stomach contents from 22 gray whales stranded between 1988 and 1991 along the coast from Kodiak Island, Alaska, to San Francisco, California. Gray whales have the unique habit of filter feeding along benthic sediments. Therefore, they are potentially capable of ingesting sediment-sorbed organic contaminants. Mean concentrations, and the ranges, measured in blubber were: DDT, 68 ± 22 ppb (1–370 ppb); DDD, 76 ± 24 ppb (1–470 ppb); and DDE, 310 ± 96 ppb (9–2,100 ppb). In liver, residues were predictably reduced: DDT, 1 ± 0.4 ppb (0.4–3 ppb); DDD, 23 ± 5 ppb (0.6–52 ppb); and DDE, 100 ± 28 ppb (7–280 ppb). Most interestingly, they found no significant differences in the concentrations from whales collected in the more pristine Kodiak Island/Washington outer coastal areas versus those collected in the more impacted areas of Puget Sound, Washington, and San Francisco.

Tilbury et al. (2002) sampled gray whales from a subsistence harvest in the Arctic during the fall of 1994 and compared their DDT concentrations (per lipid weight) with those of stranded gray whales from the same general collection area. They discovered significant differences in the harvested versus stranded whale blubber concentrations of males (200 ± 38 ppb versus 39,000 ± 23,000 ppb), females (360 ± 66 ppb versus 2,800 ± 1,000 ppb) and juveniles (330 ± 53 ppb versus 11,000 ± 4,300 ppb), respectively. The consistently higher concentrations in stranded animals may indicate their possible cause of death. However, tissue degradation of dead and potentially decaying animals limits the usefulness of such a comparison.

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

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

The DDT concentrations were also determined for some 155 minke whales harvested in 1998 from the North Atlantic and European Arctic Oceans (Hobbs et al. 2003). Results ranged from 65.3 to 6,280 ppb (lipid weight basis), which encompass the concentrations measured in whales taken 6 years earlier by Klevaine and Skaare (1998).

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

Lailson-Brito et al. (2012) reported DDT levels (lipid weight basis) of 125.6 ppm in blubber from a female killer whale (*Orcinus orca*) and 17.9 ppm in a neonatal female false killer whale (*Pseudorca crassidens*) from the southern Brazilian coast. Considering that female marine mammals tend to have lower residues of DDT than males due to lactation, male killer whales would have even higher residues than existed in the females mentioned above. Transient killer whales, known to feed primarily on marine mammals (the authors of this study reported the presence of a dolphin remains in the stomach of the killer whale), are at the highest point in the marine food-chain and therefore would be expected to have the highest residues of DDT as reported in this study.

Elfes et al. (2010) collected blubber biopsies from adult male humpback whales (*Megaptera novaeangliae*) along the North American Pacific coast in 2003–2004 and in the Gulf of Maine in 2005–2006. Whales sampled in southern California had the highest levels (lipid weight basis) of DDT at 4,900 ppb. Residues decreased more than one order of magnitude as the sampling points moved north to the Bering Sea. DDT residues increased with age.

Hoguet et al. (2013) sampled blubber in Beluga whales (*Delphinapterus leucas*) from the Alaskan artic from 1989 to 2006. The median DDT level (lipid weight basis) in blubber was 1,890 ppb. Male residues were more than three times the residue level in females. The authors note that the wide fluctuation in seasonal blubber thickness may result in mobilization of DDT from lipid stores in blubber.

McKinney et al. (2006) reported liver DDT levels (lipid weight basis) in beluga whales from the Saint Lawrence Estuary and Hudson Bay. The average residue level of DDT was 4,536 ppb for whales from the Saint Lawrence Estuary and 284 ppb for whales from Hudson Bay.

Nino-Torres et al. (2010) reported in DDT levels in blubber biopsies from fin whales (*Balaenoptera physalus*) collected in 2004–2005 in the Gulf of California, Mexico. DDT levels ranged from 200 to 1,900 ppb with an average of 500 ppb in females and 850 ppb in males. These levels are among the lowest reported worldwide for fin whales and other marine mammals.

In summary, the few studies reporting DDT residues in gray and minke whales indicate that they are also capable of accumulating residues in their blubber and other tissues. However, since they feed fairly low on the marine food web (invertebrates), their residue levels tend to be relatively lower than those of fish-eating marine mammals (viz., seals, sea lions, and dolphins). Similar to the other marine mammals discussed above, toxic effects from DDT in gray and minke whales have yet to be conclusively demonstrated.

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

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

Such a restriction has limited the field to speculation of effects, based upon measured residues and, in some cases, weak correlations. However, since metabolic activity in blubber is relatively low, it is assumed that large concentrations would need to be attained before measurable effects would be observed. Thus, to date,

few if any toxic impacts have been clearly delineated for DDT in the marine mammals that are the focus of this report, and with tissue residues clearly on the decline, the likelihood that they might be identified in the future is declining.

3.2 TMDL Targets

The Regional Board's TMDL targets for DDT in the Newport Bay and Watershed (SARWQCB 2006) are summarized in Table 5. In addition, wildlife guidance for a fish diet by Environment Canada (2000) and by US EPA (1995) has been considered as potential TMDL targets for Newport Bay and Watershed. The following sections address the science underlying each of the proposed and considered targets.

3.2.1 Sediment TELs

The decision by US EPA Region IX (2002) and the Regional Board (SARWQCB 2006) to use threshold effect levels (TELs) as TMDL sediment targets raised concern among the regulated community, because TELs do not fully consider dose-response. TELs rely to a considerable extent on the occurrence of a chemical in toxic sediments

Table 5 Proposed sediment, fish, and water column TMDL targets for total DDT in the Newport Bay Watershed

Sediment targets (µg/kg dry wt)[a]	
San Diego Creek and tributaries	6.98
Upper and Lower Newport Bay	3.89
Fish tissue targets for protection of human health (µg/kg wet wt)[b]	
San Diego Creek and tributaries	100
Upper and Lower Newport Bay	100
Fish tissue targets for protection of wildlife (µg/kg wet wt)[c]	
San Diego Creek and tributaries	1,000
Upper and Lower Newport Bay	50
Water column targets (µg/L)[d]	
San Diego Creek and tributaries	
Acute Criterion (*CMC*)	1.1
Chronic Criterion (*CCC*)	0.001
Human Health Criterion	0.00059
Upper and Lower Newport Bay	
Acute Criterion (*CMC*)	0.13
Chronic Criterion (*CCC*)	0.001
Human Health Criterion	0.00059

[a]Buchman (1999)
[b]Pollock et al. (1991); Brodberg and Pollock (1999)
[c]National Academy of Sciences (NAS 1972)
[d]US EPA Region IX (2000)

rather than on direct causation. In addition, TELs for total DDT are many orders of magnitude below thresholds for DDT in spiked sediment bioassays. These scientific issues triggered a more detailed analysis of the freshwater and marine TELs for total DDT.

The TEL sediment targets for total DDT were those summarized by Buchman (1999) from the original publications by MacDonald et al. (1996) and by Smith et al. (1996). As shown in Table 5, the targets are 6.98 ppb for the fresh water drainage into Newport Bay and 3.89 ppb for the estuarine and salt water of the Bay.

Several key authors in the TMDL regulation for Newport Bay and Watershed have expressed reservations about TELs for total DDT. Buchman (1999), author of the National Oceanic and Atmospheric Administration (NOAA) table listing the sediment targets, wrote:

> 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 references cited by Buchman wrote:

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

Peter Kozelka and David Smith at US EPA Region IX, authors of the 2002 TMDL report for Newport Bay and Watershed, wrote:

> 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.

The Regional Board's organochlorine TMDL technical report (SARWQCB 2006), addressed sediment quality guidelines (SQGs), which include TELs as one kind of SQG:

> SQGs should be used with caution since individual SQGs are often unreliable indicators of toxicity and do not necessarily identify the correct cause of toxicity (Vidal and Bay 2005). In particular, use of empirically-derived marine SQGs for DDT and PCBs has been found to be relatively inaccurate in predicting toxicity (Long et al. 1995).

A detailed analysis of the TEL method and the data sets used to derive the TELs for total DDT has revealed much more of concern that should be considered and resolved before TELs are used as TMDL targets.

To gain a full understanding of the individual data points from which the marine and fresh water sediment TELs for total DDT were derived, each data point in the TELs reported by Buchman (1999) as cited by US EPA Region IX (2002) was reviewed. The data points and reference citations were purchased from the author of the TELs.[5] The data sets are slightly different from the ones used to derive the published TELs.[6] The derivation of the TELs is explained by MacDonald et al. (1996) as follows:

> "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."

[5] MacDonald DD, BEDS data bases for total DDT purchased in 2005.

[6] Personal communication from DD MacDonald to J. Byard (2005).

Table 6 The no-effects and effects data sets for total DDT in marine and fresh water sediments

Total DDT in marine sediments[a]		Total DDT in fresh water sediments[a]	
No-effects data set	Effects data set	No-effects data set	Effects data set
0.8	0.4	0.384	1.5
1.04	0.7	0.384	1.9
1.08	1.5	0.384	5
1.27	1.58	0.384	7
1.39	1.6	0.384	9
1.5	2.92	0.384	10
3.42	2.92	5	10
4.6	2.93	5	50
4.94	3.27	5	120
5	15.4	5	222
5	18.8	5.42	4,200 ± 125
5	22.3	6	4,800
5	24	19.6 ± 18.4	11,000 ± 650
5	24.2	50.7 ± 119	11,100 ± 190
5	27	200	16,100
5	27	1,300	16,100
5	27	1,800	19,600 ± 2,180
5	29.7	5,300	21,100
5.18	33.2	5,800	30,600
5.23	46.1	22,100	46,300
5.38	54.5		49,600
6.9	55.2		49,700 ± 3,030
8.38	69		71,300
11	125		88,400
11.6	210		198,000
14.1	350		
22.5	432		
50	505		
100	596		
1,018	665		
2,170	3,000		
35,300	13,420		
	14,190		
	16,500		
	18,260		

[a]All values are in ppb dry weight in ascending order

Shaded values represent the 50th percentile of the no-effects data sets and the 15th percentile of the effects data sets

Data from DD MacDonald (personal communication)

The no-effects data sets and the effects data sets for marine and fresh water sediments are listed in Table 6 above.

Inspection of the four data sets raises the important question of why there are no effects at thousands of ppb and effects at a few or even less than 1 ppb. As will become apparent, these discrepancies are not so much the result of species and ecosystem differences, but rather, errors and misinterpretations in the data sets.

Using the 50th percentile of the no-effects data set ignores the most important information. The highest half of the data points that were not associated with a toxic effect are counted only by number and not by concentration. For example, the 50th percentile of the no-effects data set for total DDT in marine sediments is 5 ppb (average of two shaded values in Table 6). However, in the same data set, levels from 5 to 35,300 ppb are also without effect, but do not weigh by concentration of total DDT into the TEL value. Based on this data set, the threshold for toxicity is likely to be three orders of magnitude higher than the 50th percentile value. The threshold for toxicity is not appropriately weighed by the TEL calculation. This point has also been made by MacDonald, the author of the TELs. When MacDonald assessed total DDT toxicity in marine sediments from Southern California (MacDonald 1994), he reported a lowest observed effect level for total DDT for the most sensitive life stage of the most sensitive organism to be 7,120 ppb.

The 15th percentile of the effects data set for marine sediments is 2.92 ppb total DDT (shaded data point in Table 6 above). This means that only six out of 37 data points influence the 15th percentile by concentration. Twenty two data points matter only by count. Hence, whatever dose-response data are in the data set, only the 15th percentile value weighs into the TEL. Review of the underlying studies for each data point in the effects data set for marine sediments revealed highly significant errors and inconsistencies. The analysis is summarized below.

The data point of 1.6 ppb (Lyman et al. 1987), one of the 6 data points that count in the 15th percentile, is a re-publication of the 1.58 ppb data point (JRB Associates 1984) shown in Table 6. Hence, a single data point, based on equilibrium partitioning, is given the weight of two data points (Table 6).

The lowest five data points all rely on outdated and inaccurate K_{ow} values. K_{ow} values were used to estimate K_{oc} values that were then employed to determine equilibrium partitioning between water and organic carbon in sediment. The old methodology of determining K_{ow} has been superseded by the slow-stir method (de Bruijn et al. 1989). There is general consensus among scientists measuring K_{ow} that the slow stir method gives the most accurate value, and deserves to supersede earlier methodologies. K_{ow} values based on the slow stir method are recommended by U.S. EPA (2002) and are listed for the DDTs in the SARWQCB staff TMDL report (SARWQB 2006). The five data points relying on outdated K_{ow} values display errors of more than one order of magnitude.

Dealing with mixtures of chemicals becomes very important when evaluating many of the TEL data points in the total DDT effects data sets, particularly the five data points ranging from 2.92 to 3.27 ppb in marine sediments from highly polluted areas in San Francisco Bay (Table 6). Based on spiked-sediment bioassays and equilibrium partition calculations, these levels are three orders of magnitude below

effect levels for total DDT. Yet, they are included in the effects data set and the lowest value is within the 15th percentile. Inspection of the underlying study (Chapman et al. 1987) revealed pollutant levels of 9,460 ppb PAHs (polyaromatic hydrocarbons), 129,000 ppb Pb, 710 ppb Hg, and 990 ppb Cu. Here, the problem is one of toxicological plausibility. The plausible explanation for benthic toxicity is the presence of these other pollutants and not the presence of low ppb levels of total DDT. The TEL method ignores obvious toxicological interpretation of data.

The effects data points of 22.3, 46.1, 54.5, 55.2, and 125 ppb are all LC_{50} values in pore water (Word et al. 1987), rather than LC_{50} values in sediments. These values should be multiplied by the partition coefficient (a factor of at least tens of thousands) to obtain estimates of sediment LC_{50s}.

The first of the two 27 ppb AET (apparent effects thresholds) values in the marine sediment effects data set is the highest threshold associated with *Rhepoxynius abronius* toxicity for total DDT in sediments from Northern California (Becker et al. 1989). The highest threshold represents the highest concentration found in sediments without toxicity. The comparable value for Southern California, where sediments have much higher levels of total DDT, due to contamination from a DDT manufacturing site, is greater than 9,300 ppb! The second 27 ppb value is the highest threshold associated with bivalve toxicity for total DDT in sediments from Northern California (Becker et al. 1989). 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 much higher residues of total DDT were not toxic in the selected bioassays.

The association between sediment residue and sediment toxicity at 68 ppb 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 (Anderson et al. 1988). The authors stated: "Most notably, DDT concentration did not correlate with short-term toxicity or macrofaunal patterns."

The value of 210 ppb (Lyman et al. 1987) is derived in the same way as the 1.58 ppb value by JRB Associates (1984). The only difference is the use of the National acute marine criterion of 130 pptr instead of the chronic marine criterion of 1 pptr in the water column. The sediment equilibrium concentration is derived from a log K_{ow} of 5.98. The slow-stir log K_{ow} reported by de Bruijn et al. (1989) is 6.914. The K_{ow} derived by the superior slow-stir method gives a sediment acute marine threshold nearly an order of magnitude higher.

Using a method similar to the one used to estimate the 505 ppb 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 screening levels are most likely in error. Based on bioassay results, the freshwater screening level appears to be too low.

The claim that DDT is toxic to benthic organisms at low ppb levels in marine sediments is not supported by findings reported in studies cited in the effects data base. Reburial and survival of amphipods was not significantly affected by sediments

from the Palos Verdes site (Anderson et al. 1988). The total DDT concentration in the Palos Verdes sediment sample was 5,966 ppb. Marine worms that live in sediment appeared to be in excellent condition, and displayed normal burrowing behavior after 288 h of exposure to sediments containing 16,500 ppb DDT (McLeese et al. 1982).

A similar detailed analysis was done for the no-effects and effects total DDT data points used to derive the fresh water sediment TEL. The DDT analytical data for the first five data points at 0.384 ppb (Table 6) is not consistent with the major degradate being DDE. The major degradate of DDT in this study (Marking et al. 1981) was DDD. For example, the Red Wing Commercial Harbor sediments 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.

The four no-effect data points at 5 ppb were all from nontoxic sediments, and the DDTs were not detected. The value of 5 ppb was chosen as one-half the detection limit of 10 ppb. Because the TEL is derived, in part, from the median of the no effects data set, these four data points with no detectable DDTs have much greater weight than do the last four data points containing thousands of parts per million DDTs, also with no effects.

The 200 ppb data point is one-half the detection limit of the unspiked control sediment used to determine the LC_{50} of DDT in the amphipod *Hyalella azteca*. The 1,300 and 1,800 ppb values are spiked sediments used to determine the LC_{50} of DDT in *Hyalella azteca* (Schuytema et al. 1989). These levels did not measurably affect the survival of this amphipod crustacean.

In the effects data set for total DDT in freshwater sediments, the study containing the 1.5 ppb value (Bolton et al. 1985) lists threshold contamination concentrations in Table 2.1 for sediments based on 4% organic carbon (OC) and an equilibrium existing between organic carbon and water. The threshold in water is the National chronic criterion. The OC is corrected to 1%. Only DDT is included. Total DDT from this study is not determined and is not used in the TEL data set. Notably, the threshold concentration for DDE (the predominant form of DDT in the environment) in this study is 28,000 ppb. The problem with these data is the apparent use of old K_{oc} values that give inaccurate estimates of the partition of DDTs between sediment organic carbon and water. The likely equilibrium partitioning threshold for DDT is higher than 1.5 ppb and lower for DDE and DDD at 7,000 ppb and 3,250 ppb at 1% OC, respectively.

The fresh water screening level concentration approach (SLCA) data point is 1.9 ppb. The salt water SLCA is 428 ppb and is based on sediments from the Southern California Bight. Neff et al. (1986) 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 was not used to derive the marine sediment TEL.

The fish tissue-based guidance of 5 ppb is derived from the equilibrium between water and sediment organic carbon, using a log K_{oc} of 5.92 (Hart et al. 1988).

The log K_{oc} of 5.92 is a geometric mean of values ranging from 5.26 to 6.58. The slow-stir log K_{ow} for DDT reported by de Bruijn et al. (1989) is 6.914. The K_{ow} derived by the superior slow-stir method gives a sediment acute marine threshold for DDT that is one order of magnitude higher.

The SLCA method that is used to derive the 7 ppb data point is the same as used by Neff et al. (1986) to derive the above mentioned value of 1.9 ppb. The method is described, but the actual data used to determine the 7 ppb value were not presented (Persaud et al. 1991).

The value of 9 ppb for DDT is for the parent compound and not total DDT. Actual data and calculations are not presented in the reference (Environment Canada 1992). 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 SLCA 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.

The value of 50 ppb is reported to be the 90% effect level for the parent DDT according to the SLCA method (Environment Canada 1992). 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.

The 120 ppb effect data point is supposed to be the 95% effect level for the parent DDT according to the SLCA method (Persaud et al. 1991). This 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 this severe effect level for DDT as determined by the SLCA method.

According to the Illinois Environmental Protection Agency (IEPA 1988), the DDT effect level of 222 ppb is the average of three stations that had organisms of the lowest taxa. These three stations were also polluted by several additional contaminants, not just DDT. For example, Station GBL-08 sediments contained 270,000 ppb lead and 3,900 ppb mercury.

The value of 4,200 ppb is the calculated LC_{50} from the dose-response data from spiked sediment determination of the DDT LC_{50} for *Hyalella azteca* (Schuytema et al. 1989).

Discussion and conclusions. The TELs for total DDT are calculated from a variety of data types. 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 partition coefficients; 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 partition coefficients are included and should be removed or replaced with more accurate constants based on the slow-stir methodology (de Bruijn et al. 1989). Effects associated with relatively low concentrations of DDT are included, even though DDT concentrations that were orders of magnitude higher in sediments were 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 OC, 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 K_{oc} values that underestimate sediment thresholds.

Differences in freshwater and marine ecosystems can account for differences in toxic effect, but differences of several orders of magnitude are more likely explained by the presence of toxic levels of pollutants other than DDT in the sediments, rather than to inherent differences in the biological communities. How else can an effect level of DDTs at low parts per billion in one water body, and a no-effect-level of several thousand parts per billion in another water body be explained? The likely explanation is that other pollutants are present at toxic levels in the former and not in the latter.

The problem with using TELs as TMDL sediment targets is that risks are not accurately assessed, resulting in the assignment of resources disproportionate to risk and thereby not minimizing overall risk to humans and wildlife.

As an alternative to TELs, a sediment target for DDT can be derived that is based on the equilibrium between sediment and water as well as on bioaccumulation in fish and sensitive avian species. If one assumes a proportion of 80% DDE, 10% DDD and 10% DDT as an example of the residues typically found in sediments, the weighted log K_{oc} would be 6.77, using the slow stir K_{oc} values selected by the EPA in their 2002 DDT TMDL for Newport Bay and Watershed. Using this weighted K_{oc} value and the National criterion for the water column that is based on bioaccumulative avian toxicity, the resulting sediment target is 59 ppb.

3.2.2 NAS Fish Guidance to Protect Wildlife

The national debate over the impact of DDT on wildlife culminated in the U.S. cancellation of DDT in 1972. In the same year, the National Academy of Sciences (NAS) made recommendations for DDT residue levels in fish for the protection of wildlife (NAS 1972). One panel made a recommendation of 1,000 ppb in fresh water fish and another panel made a recommendation of 50 ppb in marine fish. The two panels cited many of the same scientific studies, in which eggshell thinning and reproductive failure were measured in sensitive avian species. Why then are the recommendations so different and which guidance is appropriate?

The recommendation of 1,000 ppb in fresh water fish appears to be based on laboratory studies in ducks and chickens, which are less sensitive species. The dose levels in these studies were intentionally high to assure inducing eggshell thinning and reproductive failure. The studies did not establish a chronic threshold for these effects. The panel admitted that their recommendation may not protect all species. The recommendation of 1,000 ppb in fresh water fish to protect wildlife appears not to be protective of reproduction in sensitive avian species.

The panel for marine fish guidance had one member, Robert Risebrough, who was an active investigator of the effects of DDT on eggshell thinning and reproduction in birds. At the time of this recommendation, Robert Risebrough had just published an article with Helen Hays on DDT in terns and fish scraps on Great Gull Island, 6 miles off the Connecticut coast in Long Island Sound. The 1970 fish data in this study (Table 7) appear to have become the basis for the 50 ppb recommendation. The data are reproduced below from the original publication by Hays and Risebrough (1972).

Table 7 DDT in fish from Great Gull Island. Table 2 in Hays and Risebrough (1972) reproduced with permission

HAYS AND RISEBROUGH
TABLE 2
DDT AND PCB RESIDUES IN FISH BROUGHT BY TERNS TO THE GREAT GULL ISLAND COLONY

Species	N	Mean weight (g)	ppm, fresh weight p,p′-DDE	p,p′-DDD	p,p′-DDT	PCB	ppm, lipid[1] DDT	PCB	DDT/PCB
Alosa aestivalis									
Blueback herring	5	12.4	0.22	0.18	0.011	0.64	6.4	10	0.64
Brevoortia tyrannus									
Atlantic menhaden	7	0.5	0.10	0.037	0.012	0.27	—	—	0.57
Clupea harengus									
Atlantic herring	2	3.3	0.022	0.027	0.00	0.38	—	—	0.13
Etrumeus teres									
Atlantic round herring	10	8.0	0.21	0.11	0.008	1.2	8.3	30	0.28
Anchoa mitchelli									
Bay anchovy	17	2.6	0.15	0.060	0.011	1.1	14	69	0.20
Menidia menidia									
Atlantic silverside	10	6.7	0.28	0.25	0.024	3.2	9.1	52	0.17
Morone americanus									
White perch	2	6.2	0.013	0.007	0.004	0.88	4.8	176	0.027
Scomber scombrus									
Atlantic mackerel	19	4.3	0.034	0.022	0.007	1.2	4.2	79	0.053

[1] Concentration of DDT is the sum of the concentrations of p,p′-DDE, p,p′-DDD and p,p′-DDT.

The recommendation of the panel is reproduced below from the NAS (1972) summary.

DDT Compounds

DDT compounds have become widespread and locally abundant pollutants in coastal and marine environments of North America. The most abundant of these is DDE [2,2-bis(p-chlorophenyl) dicholoroethylene], a derivative of the insecticidal DDT compound, p,p'-DDT. DDE is more stable than other DDT derivatives, and very little information exists on its degradation in ecosystems. All available data suggest that it is degraded slowly. No degradation pathway has so far been shown to exist in the sea, except deposition in sediments.

Experimental studies have shown that DDE induces shell thinning of eggs of birds of several families, including Mallard Ducks (*Anas platyrhynchos*) (Heath et al. 1969),[48] American Kestrels (*Falco sparverius*) (Wiemeyer and Porter 1970),[77] Japanese Quail (*Coturnix*) (Stickel and Rhodes 1970)[66] and Ring Doves (*Streptopelia risoria*) (Peakall 1970).[57]

Studies of eggshell thinning in wild populations have reported an inverse relationship between shell thickness and concentrations of DDE in the eggs of Herring Gulls (*Larus argentatus*) (Hickey and Anderson 1968).[36] Double-crested Cormorants (*Phalacrocorax auritus*) (Anderson et al. 1969),[31] Great Blue Herons (*Ardea herodias*) (Vermeer and Reynolds 1970),[70] White Pelicans (*Pelecanus erythrorhynchos*) (Anderson et al. 1969),[31] Brown Pelicans (*Pelecanus occidentalis*) (Blus et al. 1972;[26] Risebrough *in press* 1972),[62] and Peregrines (*Falco peregrinus*) (Cade et al. 1970).[27]

Because of its position in the food webs, the Peregrine accumulates higher residues than fish-eating birds in the same ecosystem (Risebrough et al. 1968).[64] It was the first North American species to show shell thinning (Hickey and Anderson 1968).[36] It is therefore considered to be the species most sensitive to environmental residues of DDE.

The most severe cases of shell thinning documented to date have occurred in the marine ecosystem of southern California (Risebrough et al. 1970)[64] where DDT residues in fish have been in the order of 1–10 mg/kg of the whole fish (Risebrough *in press* 1972).[62] In Connecticut and Long Island, shell thinning of eggs of the Osprey (*Pandion haliaetus*) is sufficiently severe to adversely affect reproductive success; over North America, shell thinning of Osprey eggs also shows a significant negative relationship with DDE (Spitzer and Risebrough, *unpublished results*).[78] DDT residues in collections of eight species of fish from this area in 1970 ranged from 0.1 to 0.5 mg/kg of the wet weight (Hays and Risebrough 1972).[67] Evidently this level of contamination is higher than one which would permit the successful reproduction of several of the fish-eating and raptorial birds.

Recommendation

It is recommended that DDT concentrations in any sample consisting of a homogenate of 25 or more fish of any species that is consumed by fish-eating birds and mammals, within the same size range as the fish consumed by any bird or mammal, be no greater than 50 μg/kg of the wet weight. DDT residues are defined as the sum of the concentrations of p,p'-DDT, p,p'-DDD, p,p'-DDE and their ortho-para isomers.

The DDT measured in the terns and in scraps of fish cast from their nests on Great Gull Island was not reported to have any effect on the terns. However, as detailed below, the breeding failure of ospreys along the Connecticut coast and on nearby Gardiner Island were clearly established in other studies. The implied assumption in the panel's recommendation is that the ospreys would be eating the same fish with the same level of residues found on Great Gull Island, and therefore that level was clearly toxic. What the summary didn't say was that the ospreys tend to feed along the coast and up the estuaries, resulting in a DDT exposure quite different from that of the terns. For example, osprey feeding patterns at a location further north were described by Greene et al. (1983). One could conclude from this and other studies that ospreys often catch fish from fresh or brackish water and, therefore, may not have been the best avian species for assessing the reproductive effect of DDT residues in marine fish. Fish from the nearby Connecticut River had much higher residues of DDT than the fish cast from tern nests on Great Gull Island.

Henderson et al. (1971) reported 0.85–3.27 ppm total DDT in fish collected from the Connecticut River in 1968 and 1969. In addition, Ames and Mersereau (1964)

reported total DDT levels of 2.5–9.2 ppm in scraps of fish cast from osprey nests on Great Island near the mouth of the Connecticut River. Moreover, ospreys feeding in the Connecticut River estuary in 1967 were poisoned by dieldrin (Wiemeyer et al. 1975). These facts were known in 1972, but were not mentioned in the recommendation. The recommendation of 50 ppb does not appear to have taken into account all of the information available in 1972.

The above information sets the stage for considering the adoption of the NAS fresh water and marine fish recommendations for use today, more than 40 years later. Much has been learned about DDT and its effects on wildlife since 1972. The feeling among investigators in 1972 was concern, frustration, and even outrage at what was happening to avian species at the top of food chains. Within only a few years, however, recovery was well underway, and by 1980 was nearly complete in many species. The study of the recovery of the sensitive avian species indicates that toxicity thresholds do exist for DDT residues in fish diets. The results of such studies provide a way of observing dose-response over time as residues slowly declined. However, the relationship between fish and egg residues became less certain as levels in the United States declined below probable but unknown levels on wintering grounds in Latin America, where DDT use continued, even until today. The following chronology will focus on some of the key studies documenting the decline and recovery of the osprey, since this species is key to the NAS panel's recommendation for marine fish. Some of the earlier studies have been reviewed by Ware (1975).

Ames and Mersereau (1964) and Ames (1966) reported on the status of the osprey along the Atlantic coast. Most populations were experiencing dramatic declines associated with poor hatching and fledgling rates. Eggs and fish remnants from nests on Great Island at the mouth of the Connecticut River were assayed for DDT and metabolites in 1962. Eggs contained an average of 8.1 μg/ml (about 9 ppm fresh weight) total DDT and fish remnants cast from the osprey nests contained 2.5–9.2 ppm total DDT. A crude biomagnification factor would be 9/5.7 = 1.6.

In 1963, Ames (1966) again studied osprey eggs from Great Island, and did a comparison with eggs from Maryland, where Ospreys were experiencing greater reproductive success. A few eggs from other locations along the Atlantic coast were also analyzed for DDT. The results are reproduced below in Table 8.

Table 8 DDT in osprey eggs from Northeastern United States. Table 2 in Ames (1966) reproduced with permission

Table 2. *DDT and its metabolites in the eggs of Ospreys from the north-eastern United States*

Locality	Year	No. of eggs	Average volume (ml)	DDE		DDD		DDT		Total residues	
				μg	μg/ml	μg	μg/ml	μg	μg/ml	μg	μg/ml
Maine	1963	3	72	120	1·7	7	0·1	5	0·06	130	1·8
Rhode Island	1963	1	68	500	7·4	100	1·5	ND	ND	600	8·8
Connecticut	1962	6	68	450	6·7	100	1·5	Trace	Trace	550	8·1
Connecticut	1963	15	68	320	4·7	20	0·3	10	0·1	350	5·1
New Jersey	1963	2	Not measured	350	5·1	40	0·6	10	0·1	400	5·9
Maryland	1963	25	70	160	2·3	40	0·6	5	0·07	205	3·0

ND = None detected.

Table 9 DDT in fish from Connecticut and Maryland. Table 3 in Ames (1966) reproduced with permission

Table 3. *DDT residues in fish samples from Connecticut and Maryland*

Species	No. of individuals	Total wet weight (g)	DDE µg	DDE ppm	DDD µg	DDD ppm	DDT µg	DDT ppm	Total residues µg	Total residues ppm
CONNECTICUT										
Black-backed Flounder	6	376	160	0·4	30	0·1	300	0·8	490	1·3
Windowpane Flounder	2	70	50	0·7	10	0·1	140	2·0	200	2·9
Alewife	4	60	20	0·3	10	0·2	100	1·7	130	2·2
Shad	1	70	80	1·1	40	0·6	100	1·4	220	3·1
Cunner	1	19	Trace		Trace		60	3·1	60	3·1
Eel	1	40	80	2·0	40	1·0	100	2·5	220	5·5
MARYLAND										
Eel	4	572	60	0·1	110	0·2	60	0·1	230	0·3
Yellow Perch	3	256	20	0·1	10	0·04	30	0·1	60	0·2
White Perch	2	93	Trace		Trace		Trace		5	0·05
Striped Killifish	1	22	Trace		Trace		Trace		5	0·1
Menhaden	2	125	Trace		Trace		Trace		5	0·05
Toadfish	1	140	20	0·1	10	0·1	10	0·1	40	0·3

The Connecticut eggs contained an average of 5.1 µg/mL total DDT compared to 3.0 µg/mL in the Maryland eggs. Ames (1966) also collected fish from osprey nests in the Maryland and Connecticut studies (Table 9).

The Connecticut fish residues ranged from 1.3 to 5.5 ppm total DDT, whereas the Maryland fish residues ranged from 0.05 to 0.3 ppm total DDT. The differences shown in DDT levels in fish diet, and in eggs and reproductive success between the two colonies, is the first report of this kind. The results provide the first indications of the relationship between levels of DDT in the fish diet, in the egg, and hatching success. A crude biomagnification factor for Connecticut osprey in 1963, based on a weighted average fish residue of 2.1 ppm, is 5.7/2.1 = 2.7. For the Maryland data, again using a weighted average fish residue, a crude estimate of the biomagnification factor from fish to egg is 3.3/0.23 = 14. The increase in biomagnification factor with declining fish residues could result from the slow equilibration between dietary residues and adipose residues in the osprey and/or dietary sources higher in DDT than the fish that were measured. Because of the second possibility, greater weight should be given to fish data that were based on scraps from osprey nests. Even these data are subject to limitations, however, because what is measured is what the osprey didn't eat and often the remnants are dehydrated, resulting in higher residues than would exist for fresh weight. Other investigators also documented the decline in osprey populations.

Peterson (1969) reported on declining populations of ospreys in the United States and Europe. The declines were mostly the result of hatching failure and were attributed to pesticides. Henny and Ogden (1970) reported on the breeding success and status of osprey populations in seven states (Table 10).

Reese (1977) reported on productivity for osprey all across the United States for the period 1966–1974 (Table 11).

Table 10 Status of U.S. osprey populations. Table 1 in Henny and Ogden (1970) reproduced with permission

STATUS OF U.S. OSPREY POPULATIONS • *Henny and Ogden*

Table 1. The estimated present status of osprey populations in portions of seven states. The complete nesting populations of each state were not sampled, thus the total number of active nests presented in this table does not represent the size of the breeding populations and may not represent the status of the complete population in each state.

State	No. Active Nests (All Years Summed)	Year of Study	No. Fledged Per Active Nest	Percent Nests Successful	Estimated (Minimal) Annual Rate Decline (Percent)	Source of Nesting Study
Florida	83	1968–69	1.22	70	stable	This paper
Minnesota	161	1966–68	1.03	65	2–3	Dunstan 1968
Maryland[b]	136	1964–65	1.03	54	2–3	Reese 1965
Wisconsin	128[a]	1952–59	0.98	53	3–4	Berger and Mueller 1969
Wisconsin	67	1960–65	0.39	30	12–13	Berger and Mueller 1969
Michigan	162	1965–67	0.39	23	12–13	Postupalsky 1969
Maine	8	1964	0.38	25[c]	12–13	Kury 1966
Connecticut	157	1960–63	0.29	23[d]	13–14	Ames and Mersereau 1964
Connecticut	30	1964–65	0.27	27[d]	13–14	Peterson 1969

[a] No data for 1957.
[b] Reese (Personal communication 1968) stated the first year of the study (1963) was preliminary and not as reliable as the following years. It was omitted.
[c] Kury (Personal communication 1969).
[d] Maximum percent of nests successful, assuming one young fledged per successful nest.

Table 11 Reproductive success in U.S. osprey populations. Table 8 in Reese (1977) reproduced with permission

JAN G. REESE
TABLE 8

RECENT NEST SUCCESS IN U.S. OSPREY POPULATIONS[1]

Location	Years	Nests	Nests Successful	Young Produced	Brood Size	Fledglings per nest	Reference
S. Massachusetts	1970–74	73	42	82	1.9	1.12	Fernandez (pers. comm.)
Chesapeake Bay:							
Eastern Bay	1966–74	323	128	229	1.8	0.71	Reese (1975)
This study	1970–74	684	386	741	1.9	1.08	
Choptank River	1968–74	188	106	190	1.8	1.01	Reese (1972, MS)
Smith Island	1968–71	71	55	98	1.8	1.38	Rhodes (1972)
Potomac River	1970–71	237	81	135	1.7	0.57	Wiemeyer (1971, 1977)
Virginia	1970–71	416	203	333	1.6	0.80	Kennedy (1971)
Michigan	1969–74	463	205	405	2.0	0.88	Postupalsky (1977 and pers. comm.)
Wisconsin	1966–69	237	111	193	1.7	0.81	Sindelar (1971)
Minnesota (Chippewa Nat. For.)	1968–72	249	120	216	1.8	0.87	Mathisen (1973)
Wyoming (Yellowstone Nat. Park)	1972–74	107	44	68	1.5	0.64	Swenson (1975)
Montana (Flathead Lake)	1967–70	80	42	77	1.8	0.96	Koplin (pers. comm.)
N. Idaho-E. Washington	1972–73	342	233	481	2.1	1.41	Melquist (1974)
Oregon (Deschutes Nat. For.)	1971	52	31	60	1.9	1.15	Lind (1971)
N. California	1969–71	136	71	139	2.0	1.02	Garber (1972)

[1] Data for all except this study were collected by two or infrequent nest visits and may not allow for mortality between final visit and fledging. Unpublished data are subject to revision.

Studies in other species soon identified eggshell thinning as the primary lesion causing hatching failure. DDE was shown to cause eggshell thinning in numerous declining species, including the osprey. Anderson and Hickey (1972) reported 21% shell thinning in osprey eggs collected in Connecticut, New Jersey and Maryland in 1957.

Johnson et al. (1975) reported 17% shell thinning in osprey eggs taken in Idaho in 1972 and 1973. Total DDT in eggs averaged 10.3 ppm. Hatching success was impaired. No fish residue measures were made. The general lack of use of DDT in the nesting grounds led the authors to suggest that exposure to DDT had occurred primarily during migration or at wintering grounds in Central America.

By 1973, fish residues, egg residues, eggshell thinning and hatching success appeared to be the critical determinants of the effect produced by DDE on osprey reproduction. All four parameters are highly correlated in declining species with exposures sufficient to cause eggshell thinning in excess of 10%. Mechanistic studies suggested that DDE acts directly on the transport, formation and/or deposition of calcium carbonate in the shell gland (e.g., see Risebrough et al. 1969).

Wiemeyer et al. (1975) evaluated known factors impacting reproduction in East Coast ospreys. The study period was 1968–1969. An egg exchange between nests in Maryland and Connecticut revealed that Connecticut eggs had lower hatching success than Maryland eggs, whether they remained in Connecticut or were moved to nests in Maryland. In contrast, Maryland eggs had higher hatching success than Connecticut eggs, whether they remained in Maryland or were moved to nests in Connecticut. The problem appeared to be the egg and not the parents or the setting. This finding is consistent with the direct effect that DDE has on the shell gland to produce thinner shelled eggs that were more susceptible to breakage, and therefore lower hatching success. DDE levels were higher in the fish diet of the osprey in some breeding areas than others, explaining the differential productivity along the East Coast.

Fish collected in Connecticut waters contained an average total DDT residue of 2.0 ppm. Fish collected in Maryland averaged 0.2 ppm total DDT. Fish scraps from osprey nests in Connecticut averaged 1.0 ppm, whereas one eel scrap from a nest in Maryland had 0.1 ppm total DDT. Fish scraps were judged to be very slightly dehydrated. Henderson et al. (1971) reported total DDT residues for 1969 in fish of 0.68 ppm for the Susquehanna River and 0.60 for the Potomac River. Both rivers flow into the Chesapeake Bay. Sampling locations on both rivers were in Maryland.

Total DDT in Connecticut osprey eggs collected in 1968–1969 was 10.3 ppm. This residue level compares with 10.9 ppm in 1964. Egg residues of total DDT from Maryland averaged 3.1 ppm. Eggshell thinning averaged 15% in Connecticut eggs and 12% in Maryland eggs. Only two eggs hatched out of 25 eggs studied in Connecticut. Fifteen eggs hatched out of 38 eggs studied in Maryland. Dieldrin may have contributed to hatching failure in Connecticut. Lethal concentrations of dieldrin were measured in a dead adult osprey found near the Connecticut River in 1967. Crude estimates of biomagnification from fish to egg were 10.9/(2.0 or 1.0) = 5.4–10.9 for Connecticut and 3.1/(0.68–0.1) = 4.6–31 for Maryland.

In a 1972 study done on an offshore island along the Gulf coast of Florida, Szaro (1978) reported an average of 0.11 ppm total DDT in fish (lipid basis converted to fresh weight assuming 5% lipid), an average of only 0.43 ppm total DDT in eggs, a 9%

thinning of eggshells and 0.73 young per female. The lower than normal reproductive success was not attributed to the eggshell thinning, which was described as near normal. The fish were scraps taken from the same nests as the eggs. The fish muscle was analyzed. A crude biomagnification factor can be calculated as 0.43/0.11 = 3.8. Whole fish would undoubtedly give a lower biomagnification factor. The population of ospreys in Florida is not migratory, remaining in Florida year-round.

Wiemeyer et al. (1978) reported on studies on osprey reproduction in New Jersey in the years 1970–1974. Until 1974, these breeding populations had high egg residue levels and poor productivity (Table 12). Fish residue data were not reported.

Eggshell thinning is summarized in Table 13.

Table 12 Average DDE residues in eggs and population status of osprey in New Jersey, 1970–1974. Data are from Table 2 in Wiemeyer et al. (1978)

Population	Year	p,p′-DDE (ppm wet weight)	Population trend and reproductive success
Potomic River, Maryland	1968–1969	2.4	Stable population; reproduction slightly depressed
Lake Coeur d'Alene, Idaho	1972–1973	8.5	Stable or increasing population; reproduction normal
Connecticut	1968–1969	8.9	Declining population; reproduction greatly depressed
Barnegat Bay Area, New Jersey	1974	16.0	Declining population; reproduction greatly depressed
Avalon-Stone Harbor, New Jersey	1970 + 1972	14.0	Declining population; reproduction greatly depressed

Table 13 Changes in shell thickness of New Jersey osprey eggs. Table 3 in Wiemeyer et al. (1978) reproduced with permission

TABLE 3

Changes in shell thickness of New Jersey osprey eggs.

Area	Year	Sample Size[a/]	Average Shell Thickness ± 95% CL[b/]	% Change from pre-1947
Eastern U. S.[c/]	pre-1947	365 (-)	0.505 ± 0.004	--
Barnegat Bay Area	1971	2 (2)	0.485 ± 0.064 (0.48 - 0.49)	-4
Barnegat Bay Area[d/]	1974	7 (4)	0.408 ± 0.073 (0.34 - 0.44)	-19
Avalon-Stone Harbor Area[d/]	1970 + 72	8 (8)	0.443 ± 0.024 (0.40 - 0.49)	-12

a/ Number of eggs measured; number of clutches represented in parentheses.
b/ Means for current samples are on a clutch basis, while that for pre-1947 is on an egg basis. Complete clutches are usually represented in museum collections (pre-1947), whereas most recent samples are single eggs from clutches. Extremes of clutch means in parentheses. CL = confidence limits.
c/ From ANDERSON and HICKEY (1972).
d/ The eggs represented here are different in part from those that were analyzed for pollutants, as reported in Table 1; see text.

The first report of a significant recovery of ospreys was by Spitzer et al. in 1978, 6 years after the ban of DDT. Eggs collected from osprey populations in Connecticut and eastern Long Island from 1967 to 1970 had 15–20% thinning, approximating the critical level associated with hatching failure in other species. DDE levels in osprey eggs from this area declined fivefold between 1969 and 1976 and threefold between 1973 and 1976. "The productivity of these ospreys has since increased from about 0.5 fledged young per pair in 1969–1973 to 1.2 fledged young in 1976–1977 (Fig. 1), approaching the range observed in 1938–1942." The results are reproduced in Fig. 7.

Productivity improved when DDE residues in eggs fell below 12 ppm (60 ppm dry weight), a finding that is consistent with those of Henny et al. (1977) for other areas. The authors acknowledged that dieldrin probably affected survival and reproduction of ospreys in the Connecticut River estuary. No fish residue data were reported.

MacCarter and MacCarter (1979) reported improving reproduction in osprey at Flathead Lake in Montana even with high egg residues of DDTs, reproduced in Table 14.

From 1967 to 1977, the number of breeding adults gradually increased even though productivity was marginal as might be expected with the high levels of DDTs. Eggshell thinning and fish residues were not reported. The productivity data are reproduced in Table 15.

A report by Spitzer et al. in 1983 gave further indication of the recovery of osprey breeding along the northeastern coast as shown in Fig. 8.

The authors noted that DDE in osprey eggs had not been measured since 1976. Presumably DDE residues were declining as reproduction improved. They also made note of a brood-size reduction of 50% or more due to food limitations on Gardiner Island, the same island mentioned as impacted by DDT in the NAS recommendation for marine fish.

Spitzer and Poole (1980) and Poole (1989) revisited the issue of the struggling population of ospreys on Gardiner Island. The population was decimated by DDT in the 1950s and 1960s. Local citizens took up the cause to save the osprey. They sued Suffolk County to stop spraying DDT for mosquito control and achieved a ban on eastern Long Island. This local citizens group later became the Environmental Defense Fund. One of their members, Dennis Puleston, was an author of the 1978 report (Spitzer et al.) on the recovery of osprey populations on eastern Long Island. Recovery of the osprey on Gardiner Island was well underway in the 1970s when reproduction failed again due to a limited food supply. Apparently male osprey had to travel long distances to reliable supplies of fish in the marshes of the south fork of Long Island. According to the authors, when this colony thrived it was dependent on menhaden in nearby Gardiner's Bay. Excessive commercial fishing removed this food source, leading to a marginal food supply.

Reporting on a national survey of osprey breeding in 1983, Henny stated: "Ospreys at locations with poor production have all showed improvement following the DDT ban in 1972."

Wiemeyer et al. (1988) reported DDT effects on osprey eggs and reproduction from several data sets generated in the 1960s and 1970s. Some declines in residue

Fig. 7 Beginning of recovery of osprey productivity in Connecticut–Long Island. Figure 1 in Spitzer et al. (1978) reproduced with permission

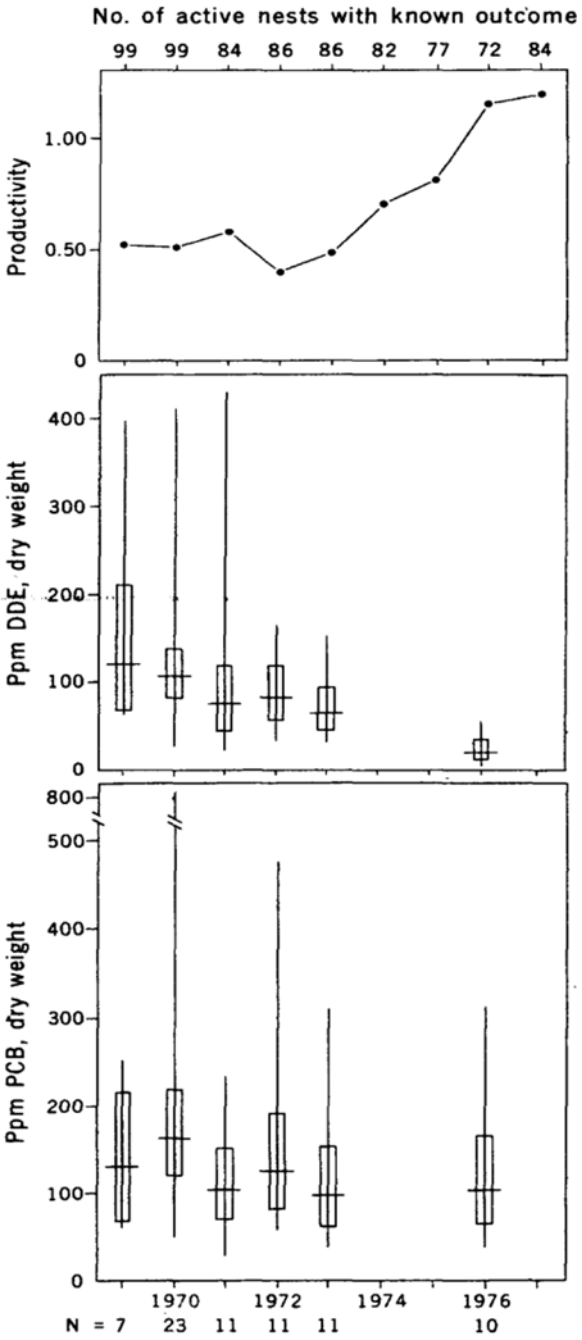

Fig. 1. Active nests of ospreys in Connecticut–Long Island with known outcome, 1969 to 1977; productivity, defined as young fledged per active nest; DDE and PCB residues, parts per million dry weight, with the sample sizes. Horizontal bars are geometric means; rectangles are the 95 percent confidence intervals of the means; vertical lines are the sample ranges.

Table 14 DDT residues in addled osprey eggs from Flathead Lake, Montana. Data are from Table 2 in MacCarter and MacCarter (1979)

Year	Nest number	Egg number	DDE[a]	DDD	DDT	Total DDTs
1968	BI-1	1	5.1	1.2		6.3
	BI-3	1	7.9	1.3		9.2
	DB-1	1	11.4	0.85		12.2
	DB-1	2	10.4	4.4		14.8
1969	BI-1	1	13.5	2.6		16.4
	BI-2	1	6.5	2.0		8.5
	BI-2	2	10.1			10.1
	DB-1	1	5.2			5.2
	DB-1	2	9.5			9.5
	BI-5	1	22.6			22.6
1970	BI-1	1	16.0		1.5	17.4
	BI-2	1	13.5		2.2	15.7
	BI-5	1	5.3		0.4	5.7
	BI-5	2	3.8			3.8
	N-D-1	1	5.9	0.6	1.7	8.2
1976	BI-3	1	3.1	0.12		3.22
	BI-5	1	37.0	3.3	0.35	40.65
	CB-8	1	35.0	5.6		40.60
1977	BI-3	1	2.9	0.14		3.04
	BI-5	1	16.0	1.2		17.20
	CB-8	1	8.7	1.1		9.8
	CB-8	2	11.0	1.2	0.20	12.40

[a]All residues are ppm wet weight

Table 15 Nesting productivity in osprey eggs from Flathead Lake, Montana. Table 3 in MacCarter and MacCarter (1979) reproduced with permission

TABLE 3. Nesting productivity of Ospreys at Flathead Lake, Montana.

Year	No. nesting pairs (A)	No. young (B)	No. young fledged (C)	No. nestlings per pair (B/A)	No. fledglings per pair (C/A)
1967	16	18	17	1.12	1.06
1968	20	14	14	0.70	0.70
1969	20	20	15	1.00	0.75
1970	24	33	31	1.38	1.29
1974	28	36	34	1.31	1.21
1975	30	41	38	1.37	1.27
1976	36	43	40	1.19	1.11
1977	38	38	36	1.00	0.95
Total					
Average	212	243	225	1.15	1.07

THE MURRELET

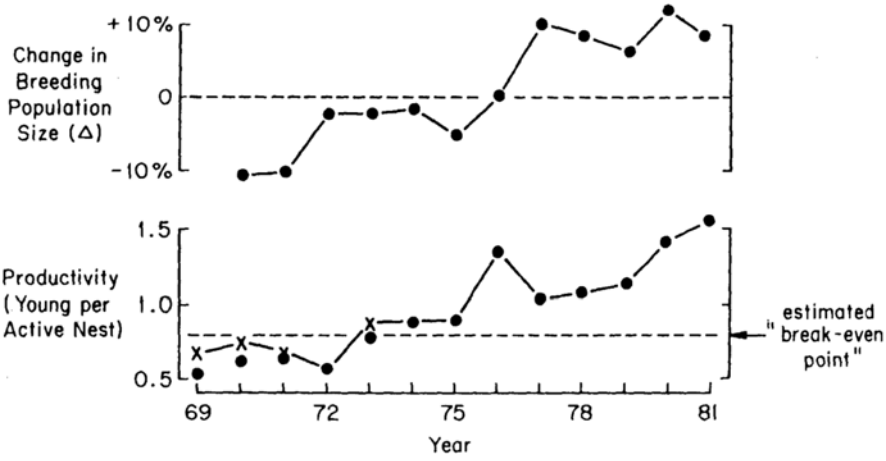

FIGURE 2. A comparison of Osprey reproductive rate and change in population size, N.Y. City to Boston, 1969-1981. Points denoted by "X" on the lower graph are productivity values which include young introduced from Maryland by Spitzer (1978).

Fig. 8 Productivity and population size of osprey in the region between New York City and Boston. Figure 2 in Spitzer et al. (1983) reproduced with permission

levels and shell thinning were noted. Analysis of the DDE egg residue—shell thinning relationship revealed 10% thinning at 2.0 ppm, 15% at 4.2 ppm and 20% at 8.7 ppm. Reproductive failure was attributed to DDE causing thinning of eggshells. Ospreys were considered to be as sensitive as other sensitive species.

In his book on ospreys, Poole (1989) published Figure 9.7 relating DDE residues in osprey eggs with eggshell thinning as shown in Fig. 9.

Poole's data illustrate the wide variability in eggshell thinning at each residue level, explaining why populations increase even at levels of DDE that result in some shells breaking and failing to produce viable young. Reproductive failure and mortality due to high residues of dieldrin and PCBs, particularly in the 1960s and early 1970s, may account for some of this variability. Poole also reported on the DDE egg residue—production dose-response as shown in Fig. 10. Poole set the reproductive effect threshold at 4.3 ppm DDE. This number compares with the 15% shell-thinning value suggested by Wiemeyer et al. (1988) at 4.2 ppm DDE.

Schmitt et al. (1990) published the results of a national fresh water fish residue survey for 1984. Total DDT residues in fish from the Connecticut River averaged 0.22 ppm. For all sites sampled nationwide, the trend of the geometric average total DDT residue was 0.39 ppm in 1976–1977, 0.36 ppm in 1978–1979, 0.32 ppm in 1980–1981 and 0.28 ppm in 1984. Schmitt et al. (1981) had earlier published a nationwide level of 1.08 ppm in fish collected between 1970 and 1974. Bilger et al. (1999) discussed EPA analysis of multi-species composite analyses done in 1987. The mean DDE concentration was 0.295 ppm in a nationwide sampling. The USGS

Figure 9.7. Osprey eggshell thickness in relation to DDE residues in the egg. Data shown are mean values (vertical lines show ranges) based on analyses of 112 eggs, most collected in the northeastern United States during the 1960s and 1970s. Data from Spitzer *et al.* (1978) and A. Poole & J. Farrington (unpublished).

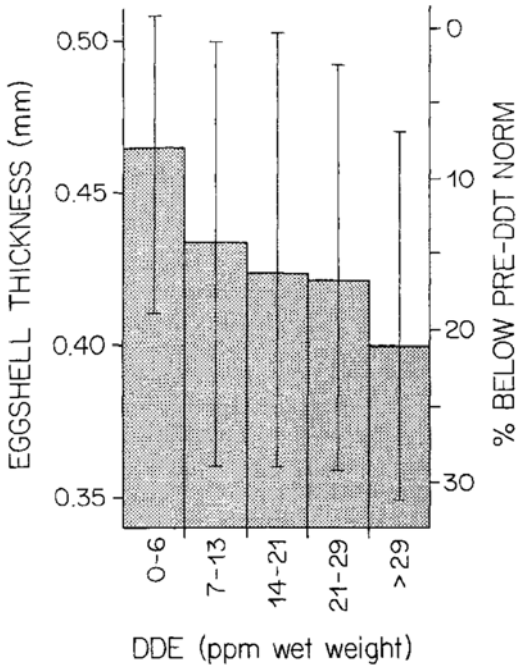

Fig. 9 Osprey eggshell thickness in relation to egg residue of DDE. Figure 9.7 in Poole (1989) reproduced with the permission from the author and from Cambridge University Press

Figure 9.8. Mean brood size at Osprey nests in Sweden (1971–1973) and in New England (USA) (1969–1984), in relation to DDE levels in unhatched eggs from those nests. Swedish data, from Odsjö (1982), show young hatched per nest ($N = 108$ nests); US data, from Spitzer *et al.* (1978) and Poole & Farrington (unpublished), show young fledged per nest ($N = 101$ nests).

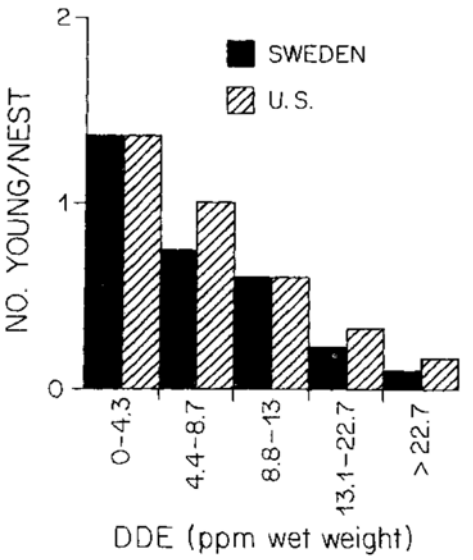

Fig. 10 Osprey productivity in relation to egg residue of DDE. Figure 9.8 in Poole (1989) reproduced with the permission from the author and from Cambridge University Press

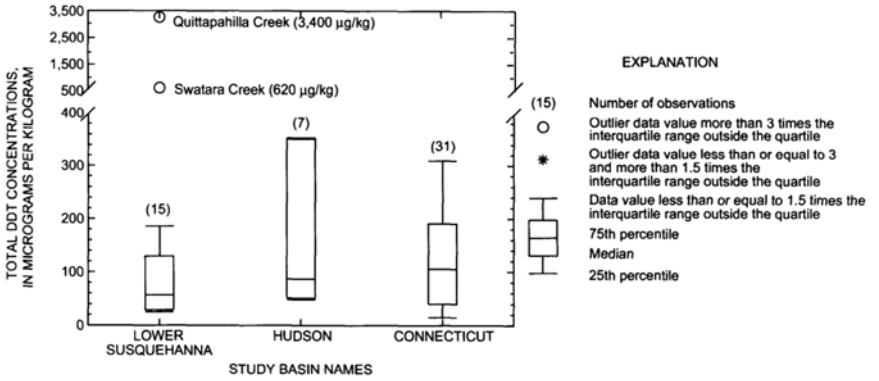

Figure 2. Concentrations of total DDT in white sucker whole fish tissue for the Lower Susquehanna, Hudson, and Connecticut River Basins.

Fig. 11 Total DDT in white sucker tissue from Northeastern United States. Figure 2 in Bilger et al. (1999) courtesy of the U.S. Geological Survey

multi-species fish sampling of the lower Susquehanna River basin by Bilger et al. (1999) in 1992 found median residues of 0.250 ppm of total DDT. Variability between sites was very high as shown in results for white suckers collected from the Susquehanna, Hudson and Connecticut River Basins (Fig. 11).

The overall trend for DDT in fish residues in the 1970s and 1980s was a steady decline, although hot spots were clearly evident. If these hotspots are sources of food for ospreys and are missed in fish surveys, then the residue exposures may be greatly underestimated, resulting in an overestimate of biomagnification from fish to osprey egg.

Steidl et al. (1991a, b) published two papers on osprey reproduction in three regions of southern New Jersey. The three locations were the more polluted Delaware Bay, the less polluted Atlantic Coast and an intermediate location along the Maurice River that flows into the lower Delaware Bay. Eggs were collected in 1985–1989. The authors noted that average fish residue of total DDT in the Delaware River was 0.88 ppm in 1984. Total DDT residues in eggs were low with the highest levels in Delaware Bay as shown in Table 16. Eggshell thickness was negatively correlated to DDE levels in the eggs as can be judged from Tables 16 and 17.

Apparently, eggs with shells thinned near to or at 15% had a greater probability of breaking, contributing to the lower productivity observed in Delaware Bay compared to the other two locations as shown in Table 18.

Analysis of DDT residues in prey fish revealed the following results as shown in Table 19.

One should keep in mind that these fish samples were not scraps from the osprey nests but fish caught locally in the breeding grounds. Since ospreys often feed up the rivers from their breeding grounds, more contaminated fish may well have been consumed. Viscera were removed from whole fish prior to analysis. Viscera would contain liver, some adipose tissue and other organs that would be expected to have

Table 16 Geometric mean DDE and DDD residues (ppm wet wt) in osprey eggs from Delaware Bay. Data from Table 1 in Steidl et al. (1991b)

Region and egg type	n	DDE (geometric mean)	DDE (range)	DDD (geometric mean)	DDD (range)
Delaware Bay					
Random	7	3.2	1.7–5.2	0.4	0.3–0.7
Addled	4	2.9	1.6–4.7	0.4	0.3–0.6
All	11	3.1		0.4	
Atlantic Coast					
Random	8	1.2	0.5–2.8	0.2	0.1–0.6
Addled	4	1.6	1.4–1.8	0.2	0.2–0.3
All	12	1.4		0.2	
Maurice River					
All	2	1.9	1.6–2.3	0.2	0.2–0.2

Table 17 Eggshell thickness of random (1989) and addled (1985–1988) osprey eggs, and eggshell fragments (1987–1988), from three regions of New Jersey. Data from Table 3 in Steidl et al. (1991b)

Region and shell type	n	Eggshell thickness (mm) Mean	Eggshell thickness (mm) SE	% Below pre-1947 thickness[a] Mean	% Below pre-1947 thickness[a] SE
Delaware Bay					
Random	7	0.444	0.020	12.0	3.9
Addled	8	0.466	0.014	7.8	2.7
Fragment	2	0.430	0.005	14.9	1.0
All types	17	0.453	0.011	10.4	2.1
Atlantic Coast					
Random	8	0.485	0.020	4.0	3.9
Addled	22	0.488	0.011	3.3	2.2
Fragment	19	0.472	0.011	6.5	2.1
All types	49	0.482	0.007	4.7	1.4
Maurice River					
Random	2	0.490	0.045	3.0	8.9
Fragment	2	0.465	0.005	7.9	1.0
All types	4	0.478	0.020	5.5	3.9

[a]Compared to data from Anderson and Hickey (1972)

relatively high concentrations of DDT. Finally, these fish were caught in 1989 and the eggs were collected from 1985 to 1989. Some decline in fish residues from 1985 to 1989 would be expected, based on data from other locations. Given all of the above, crude bioconcentration factors can be calculated as 5.7/0.54 = 5.7 for the Delaware Bay, 1.4/0.09 = 15.6 for the Atlantic coast and 1.9/0.095 = 20 for the Maurice River.

As the level of DDT in fish decreased, the bioconcentration factor appears to have increased. This pattern became even more evident as fish residues continued to

Table 18 Osprey productivity in three regions of New Jersey. Table 2 in Steidl et al. (1991b) reproduced with permission

Table 2. Reproductive parameters of ospreys nesting in 3 regions of New Jersey, 1987–88.

Region	*n*	% eggs hatched	$\bar{x}$ young fledged/ pair	% nest success[a]
Delaware Bay[b]	24	50.0[c]	1.08	50.0
Atlantic Coast[b]	38	68.5	1.61	78.9
Maurice River	6	62.5	1.33	66.7

[a] Nests fledging ≥1 young.
[b] Data from Steidl et al. (1991).
[c] $n = 12$ nests.

Table 19 DDE and DDD (ppm fresh wt) in osprey prey fish collected from three regions of New Jersey. Data from Table 5 in Steidl et al. (1991b)

Region and species	n[a]	DDE	DDD	% Moisture
Atlantic Coast				
Menhaden	5	0.05	0.04	62.8
Delaware Bay				
Menhaden[b]	5	0.17	0.12	66.2
White perch[c]	5	0.68	0.27	71.8
Channel catfish[d]	2	0.25	0.14	71.4
Maurice River				
White perch	6	0.05	0.03	72.2
Channel catfish	2	0.08	0.03	76.0

[a]Number of fish in composite sample
[b]Composite contained (ppm) 0.02 *p*,*p*′-DDT, 0.03 *o*,*p*′-DDE, 0.08 *o*,*p*′-DDD
[c]Composite contained (ppm) 0.11 *o*,*p*′-DDE, 0.27 *o*,*p*′-DDD
[d]Composite contained (ppm) 0.03 *o*,*p*′-DDE, 0.05 *o*,*p*′-DDD

decrease. One must keep in mind that as DDT residues continued to decrease in the United States, following the ban in 1972, exposure to DDT in wintering grounds in Latin America accounted for an increasing proportion of egg residues. DDT was used in Latin America after 1972 and is still in use in some locations today. These wintering ground exposures became more important as residues in fish in the U. S. continued to decrease. The multi-year half-life of DDT ensures that the highest exposures will be reflected in adipose concentrations that are passed directly into the yolk of the egg.

Other factors contributing to reproductive effects in ospreys in southern New Jersey include the presence of 4.1–26 ppm PCBs in the osprey eggs from Delaware Bay (Steidl et al. 1991b). The authors noted that the Delaware Bay is routinely dredged to maintain a shipping channel to ports on the Delaware River. They suggested that dredging exposed biota to old sediments containing higher residues of DDT and PCBs, resulting in a slower decline of residues and the persistence of effects no longer seen at other locations. Another factor is the travel time required to catch fish due to the lack of clarity of the water in the nesting areas that are in the more polluted parts of the Bay. Long travel times did not limit the food supply but did increase the

Table 20 The importance of wintering ground exposures to DDT in peregrine falcons in the late 1970s. Table 1 in Henny et al. (1982a) reproduced with permission

TABLE 1. DDE (geometric means, ppm wet weight) in blood plasma of Peregrine Falcons captured during migration at Assateague Island, Maryland/Virginia and Padre Island, Texas.

	Maryland/Virginia[a]			Texas			Maryland/Virginia			Texas		
Year	Mean	(95% C.I.)	n	Mean	(95% C.I.)	n	Mean	(95% C.I.)	n	Mean	(95% C.I.)	n
	HY♀♀						HY♂♂					
1976-77	0.11	(0.07–0.19)	15	0.05	(0.03–0.08)	15	0.08	(0.05–0.14)	9	0.16	—	2
1978	0.04	(0.02–0.08)	25	0.03	(0.02–0.07)	20	0.03	(0.01–0.10)	8	0.06	(0.03–0.09)	16
1979	0.07	(0.05–0.10)	36	0.05	(0.04–0.07)	74	0.08	(0.06–0.11)	26	0.05	(0.04–0.08)	22
Totals	0.06	(0.05–0.09)	76	0.05	(0.04–0.06)	109	0.07	(0.05–0.09)	43	0.06	(0.04–0.08)	40
	SY♀♀						ASY♀♀					
Fall												
1976-78	0.82	(0.44–1.53)	11	0.28	(0.01–6.75)	4		—		0.60	(0.27–1.33)	6
1979	0.64	(0.38–1.07)	6	0.27	(0.02–3.91)	3	0.71	(0.14–3.67)	5[b]	0.33	(0.14–0.77)	12
Totals	0.75	(0.50–1.13)	17	0.28	(0.07–1.16)	7	0.71	(0.14–3.67)	5	0.40	(0.22–0.72)	18
Spring												
1978-79		—		1.43	(0.52–3.87)	8		—		0.88	(0.60–1.29)	21
1980		—		0.42	(0.24–0.73)	19		—		0.62	(0.48–0.79)	63
Totals		—		0.60	(0.36–1.00)	27		—		0.67	(0.55–0.83)	84

[a]Excludes 3 HY♀♀ that were released along East Coast by Cornell University biologists.
[b]Includes one sample from 1978.

time the nests were unattended, leading to potentially greater predation by great horned owls.

Considering the importance of the unknown exposure of ospreys to DDT in wintering grounds, digression to an article by Henny et al. (1982a) is enlightening. Henny et al. reported the measurement of DDT in the blood of peregrine falcons captured during migration north in the spring and south in the fall (Table 20). The peregrine falcon migration is similar to that of the osprey.

The table requires explanation. HY falcons are those migrating in the year they hatched. SY falcons are second year falcons and ASY means falcons migrating after their second year. Focusing on the Texas data for female falcons, one can see that just fledged falcons on their way south have quite low levels of DDE. SY falcons returning north in the spring of the next year have more than ten times as much DDE in their plasma. Plasma levels are lower in SY falcons migrating south from northern breeding areas. Apparently, body burdens gained in the south during the winter are decreasing in the north during summer as a result of both egg laying and ever decreasing exposures in the northern breeding areas. The same pattern would be expected in osprey.

This exposure paradigm is even more important for the osprey since fledglings do not return to northern breeding grounds until their third year. Southern exposures to DDT could explain the ever increasing bioconcentration factors calculated from just northern exposures. As DDT levels decreased in the United States to levels below those in Latin America, the importance of the unknown southern exposure eventually becomes essential to understanding the relationship between DDE residues in the fish diet and levels in eggs associated with thinning and hatching failure. With the understanding gained from this digression, let us resume reviewing the chronology of studies of the effects of DDE on osprey reproduction.

Audet et al. (1992) measured DDT residues in osprey eggs from three locations on the East Coast and compared them with residue levels in the early 1970s. The study was prompted by the finding of an isolated area in Chesapeake Bay with declining nestling survival. Median DDE levels in 1986 were 2.3 ppm in an area of declining fledgling survival (Martin Refuge), 0.65 ppm in coastal Virginia and 0.56 ppm in southern coastal Massachusetts. Relatively high ratios of DDT to DDE in the eggs from Massachusetts prompted the authors to suggest recent exposure to DDT from an unknown source (winter breeding grounds?). Eggs taken in 1972–1973 from the same area of Massachusetts had DDE residues of 4.2 ppm. The authors concluded that the 0.65 and 0.56 ppm levels of DDE "were well below reported values associated with biologically significant effects on eggshell thickness and reproductive success." In 1973, the Martin refuge had a median DDE level in eggs of 3.4 ppm with 17% eggshell thinning, but nonetheless, 1.5 young per active nest. Productivity of 1.5 young per active nest was considered by these authors to be excellent. No reason was given or suggested for the declining fledgling survival at the Martin Refuge in 1986. Fledgling survival data were not reported.

Falkenberg et al. (1994) provided data on a nonmigratory population of osprey and their prey from the south coast of Australia. Six eggs collected in 1987 had an average total DDT residue of 0.22 ppm. Total DDT residues in three species of prey fish averaged 0.3 ppm, giving a very low biomagnification factor of 0.73. Shells of osprey eggs collected in 1987–1988 were no thinner than shells of eggs collected prior to the DDT era. The biomagnification of DDT into osprey eggs was so low in this study as to put into question the representativeness of the fish samples as a significant part of the diet eaten by osprey that produced the eggs collected in the study. The determination of a biomagnification factor is theoretically more certain in a nonmigrating population. Most likely, the biomagnification factor is small, based on studies in the 1960s and early 1970s in the U. S., probably less than ten.

In 1997, Ewins published an article about the behavior and history of osprey in North America. Figure 1 from Ewins (1997) illustrates the recovery of ospreys in Wisconsin and the Georgian Bay area of the Great Lakes Region (Fig. 12).

Woodford et al. (1998) reported geometric mean DDE residues of 0.20–0.52 ppm in osprey eggs collected in 1992–1993 from two breeding areas in central and northern Wisconsin.

Ewins et al. (1999) reported on eggs collected between 1980 and 1989 from two osprey breeding areas in central Michigan. The known age of each female osprey producing the eggs permitted a study of DDT residues in eggs produced by females from 3 to 15 years of age. No age-related changes were found. The egg residues were independent of the age of the female, and DDE averaged 1.2 ppm. Eggshell thickness increased from 1980 to 1989. Eggs collected from 1980 to 1984 were 5% thinner and eggs collected from 1985 to 1989 were 3% thinner than eggs collected prior to the DDT era. Eggs collected from the same areas in 1972–1973 had geometric mean concentrations of 5.1 ppm DDE and 10% average shell thinning. The decrease in DDE residues was associated with improved reproduction and population increases. Apparently, female osprey in Michigan reached a steady-state DDE residue level in their tissues in the first 2–3 years of life (most of that time is spent

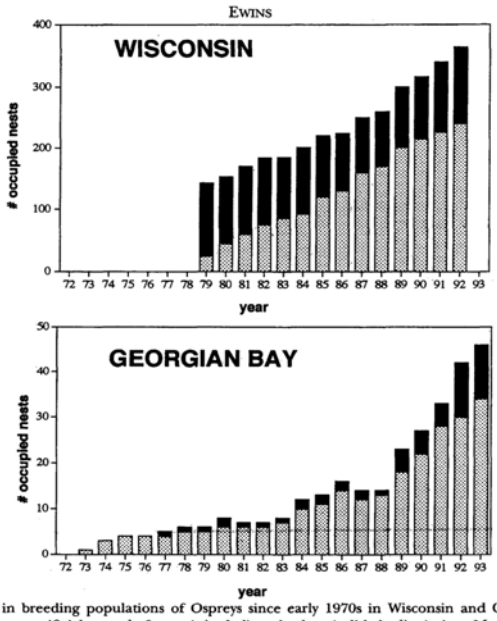

Figure 1. Changes in breeding populations of Ospreys since early 1970s in Wisconsin and Georgian Bay (Lakes Huron and Ontario), at artificial nest-platforms (stippled) and other (solid shading) sites. Most "other" sites were in trees. Wisconsin data are from Gieck et al. (1992).

Fig. 12 Recovery of ospreys in Wisconsin and the Georgian Bay area of the Great Lakes. Figure 1 in Ewins (1997) reproduced with permission

on the wintering grounds in Latin America). Part of this ongoing steady-state is the elimination of accumulating adipose residues by laying eggs. The DDE residues in eggs from midwestern breeding grounds and some east coast locations appear by the mid 1990s to be below levels associated with any significant effects on shell thickness or hatching success.

Elliott et al. (2000) reported on DDT residues in osprey eggs collected from the Columbia and Fraser River areas in the northwest. DDE residues were high and variable. Geometric means ranged from 1.0 to 13.8 ppm by area and year from 1991 to 1997. No trends by area or year were evident. Individual eggs ranged from 0.1 to 23.7 ppm DDE. DDE/DDT ratios were also highly variable. Some of the locations were in forested wilderness areas where little DDT had been used. Fish sampled in 1994 from these remote areas contained less than 0.005 ppm total DDT. The authors suggested that DDT was coming from an outside source, possibly from wintering grounds in southern Mexico. Another factor is the very high rate of DDT applications to apple orchards during the DDT use era (Blus et al. 1987). Twenty three percent of the osprey eggs had DDE residues greater than 4.2 ppm, the level associated with eggshell thinning that is significant to hatching success.

Clark et al. (2001) published a follow-up study of the Steidl et al. (1991b) Delaware Bay study summarized above. Comparisons between 1989 and 1998 at three locations in southern New Jersey were made in residue levels in eggs and fish,

eggshell thinning, and productivity. DDE residues in osprey eggs had declined to 1.4 ppm with an associated eggshell thinning of 7% in the more contaminated Delaware Bay area. The authors concluded that "PCBs and DDE in osprey eggs were below levels considered to be toxic to egg development." Fish were collected in the same manner as in 1989. Total DDT residues in fish for the Delaware Bay averaged 0.23 ppm. Biomagnification factors from fish to eggs ranged from 9 to 11. Osprey productivity increased to 1.1 young per nest in the period from 1994 to 1998. Availability of nest structures and owl predation were thought to be limiting the population of ospreys in the Delaware Bay area.

In 2003, Martin et al. reported on ospreys in Great Lakes Canada. The study was conducted in 1991–1995. DDE levels averaged 1.3–2.9 ppm in five study areas. A few eggs exceeded the 4.2 ppm (15% eggshell thinning) threshold, suggesting that reproduction in a few individual ospreys was affected. The authors concluded, however, "…ospreys now appear to be relatively unaffected by current low levels of chlorinated hydrocarbon contaminants."

Henny et al. (2003) reported on a detailed 1993 study of bioaccumulation of DDE from fish to osprey eggs in Oregon. The number of breeding pairs along the Willamette River increased from 13, in 1976, to 78, in 1993 and 234 in 2001. Overall productivity was 1.67 young per active nest. The geometric mean of DDE egg residues was 2.3 ppm. Two of the ten eggs analyzed had levels of DDE that would be expected, based on other studies, to have reduced hatching success as a result of cracked shells.

The median level of DDE in the major food fish for ospreys, the largescale sucker, was found to be only 0.022 ppm. This very low fish residue resulted in a bioaccumulation factor for fish to osprey eggs of 87, prompting the authors to suggest that ospreys received significant exposures during winter migration to southern Mexico and Central America. This idea was reinforced by lower than expected bioaccumulation of PCBs and unexpectedly high levels of DDT in some eggs. However, much higher levels of DDE in largescale suckers from the Willamette River have been reported. A single composite collected between 1996 and 1998 contained 0.835 ppm (US EPA Region X 2006). A bioaccumulation factor from this fish residue value would be 2.3/0.835 = 2.8.

In a chapter in Raptors Worldwide, Henny et al. (2004) described a study of the effects of DDE residues on osprey eggshells and reproduction at nest sites along the Columbia River in northwestern United States. The number of ospreys had been increasing with each survey through 1998. Mean productivity was 1.64 young per active nest (Table 21). Eggs were collected in 1997 and 1998.

Dividing the nests into three classes by DDE egg residue level indicates a dose-response for thinning of eggshells and impairment of reproduction. Even at these high levels, with measurable impacts, the osprey population continues to grow. The geometric mean residue of DDE in eggs from nests along the Columbia River was 4.9 ppm, a value higher than residues reported by the same authors for eggs collected in 1993 along the adjoining Willamette River. These residues are the highest reported nationwide for osprey eggs during the late 1980s and 1990s. Henny et al. suggested the possibility of exposure to DDT on the wintering grounds in

Table 21 Productivity, eggshell thinning and DDE egg residues in osprey along the Columbia River. Table 5 in Henny et al. (2004) reproduced with permission

Table 5. Number of young Ospreys produced per nest (with one egg collected) in relation to DDE concentrations in the sample egg collected, and eggshell thickness.

	Number of Nests with DDE (μg kg^{-1})		
Number of Young	< 4200	4200-8000	> 8000
0	1	3	3
1	6	3	2
2	10	6	3
3	1	0	0
Active Nests	18	12	8
Successful Nests	17	9	5
Adv. Young	29	15	8
Young/Successful Nest	1.71	1.67	1.60
Young/Active Nest	1.61	1.25	1.00
Geo. Mean DDE (μg kg^{-1})	2131	5473	10510
Mean Shell Thickness (mm)	0.488	0.441	0.419
Shell Thinning[a]	-3.4%	-12.7%	-17.0%

Note: One nest sampled did not have complete information for productivity (it was excluded), and 10 nests were included from the Willamette River in 1993 (Henny and Kaiser 1996).

[a] Compared to 0.505 mm for pre-DDT era eggshells from eastern U.S.A. (Anderson and Hickey 1972).

southern Mexico and Central America. Another explanation could be the high application rates of DDT to apple orchards, creating pockets of high residues in soil and biota, including fish (Blus et al. 1987).

Fish residues were stated to be elevated, although levels were not reported. Previous investigations from 1991 to 1993 were cited by the authors to have found an average of 0.089 ppm DDE in largescale suckers, an important food fish for the ospreys. Schmitt et al. (1990) had reported 1.0 ppm total DDT in largescale suckers from the Columbia River in 1984. The US EPA Region X (2006) reported average total DDT residues of 0.450 ppm in largescale suckers collected in 1996–1998 from the Columbia River Basin. Figure 2-4b from the report (Fig. 13) illustrates the high variability in the fish residues at different locations, explaining to some degree the high variability in DDE levels in osprey eggs. A crude estimate of the biomagnification of DDE from fish to egg would be 4.9/(0.450–0.089) = 11–55.

Martell et al. (2001) used satellite telemetry to track the migration of osprey from northern breeding areas to southern wintering areas. East coast osprey winter primarily in Brazil, west coast osprey winter primarily in southern Mexico and midwestern osprey winter in both locations or in between (Fig. 14).

Mora (1997) reviewed available information on reports of DDT contamination of migratory birds in Mexico. Contamination generally was found to be similar to that in southwestern United States through the 1980s.

Rattner et al. (2004) reported on contaminant exposure and reproductive success of ospreys in the Chesapeake Bay area. From a population estimated at 1,450 nesting pairs in 1973, the Chesapeake Bay osprey population more than doubled to an estimated 3,473 pairs by 1995–1996. However, reproduction rates have not fully recovered in the more polluted waters of the Bay. Geometric means of DDE levels

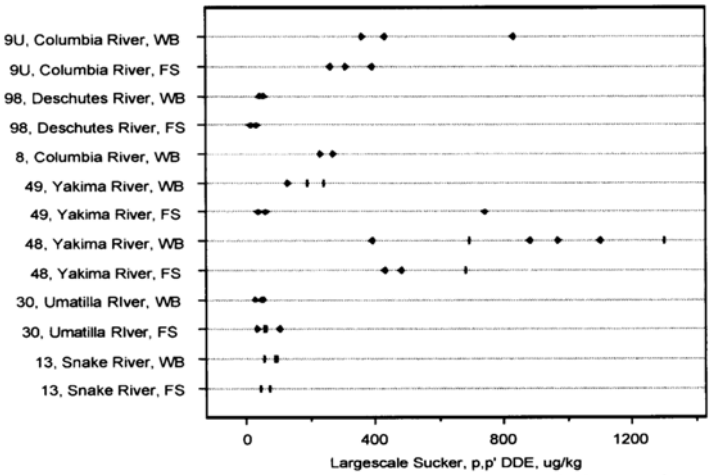

Fig. 13 DDE residues in largescale suckers from the Columbia River Basin. Figure 2-4b reproduced from US EPA Region X (2006)

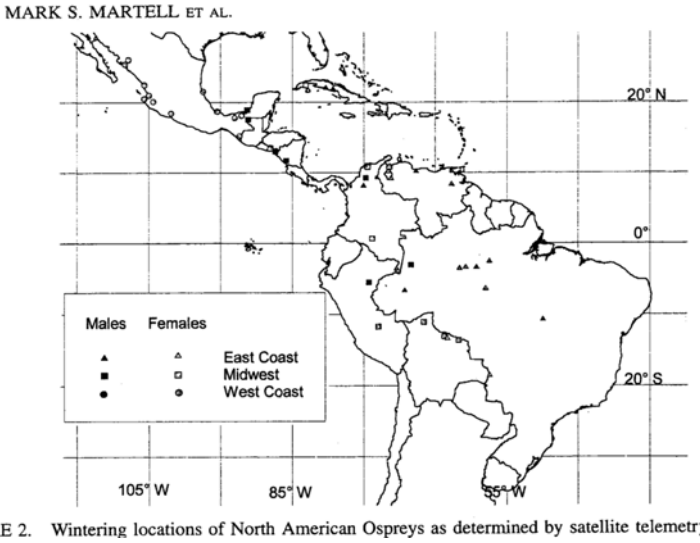

Fig. 14 Wintering locations of North American Ospreys. Figure 2 in Martell et al. (2001) reproduced with permission from author and from The Condor, published by the Cooper Ornithological Society

in eggs collected in 2000 from different parts of the Bay ranged from 0.4 to 1.2 ppm. Eggshell thinning ranged from 0 to 9%. PCBs were as high as 19 ppm in eggs from nests in the more polluted areas. A limited sample of fish scraps from nests in some of the less polluted areas contained less than 0.05 ppm DDTs, corresponding to

0.4–0.8 ppm total DDT in osprey eggs from those areas. A crude estimate of the bioaccumulation of total DDT from fish to eggs would be 0.4–0.8/<0.05 =>8 to >16. Marginal productivity in the more polluted areas was not linked to egg concentrations of DDE. DDE levels in osprey eggs from the Chesapeake Bay have decreased tenfold from the DDT use era. "…concentrations of p,p′-DDE…in sample eggs did not cause direct and biologically significant toxic effects on osprey reproduction in Chesapeake Bay regions of concern."

A third study of the Delaware Bay was conducted in 2002 by Toschik et al. (2005). Geometric mean DDE levels in eggs from four parts of the Bay were 0.4–1.8 ppm. Eggshells from the northern part of the Bay were 10% thinner. A few eggs from failed nests contained more than 4 ppm DDE. The authors stated that "All nestlings appeared in good health; no external lesions or other abnormalities were found." and "Additionally, no evidence of chromosomal damage in nestlings was found." Based on only a few eggs, DDE in eggs from the Prime Hook National Refuge were 0.6 ppm in 2002 compared to 5 ppm in 1974. Marginal reproduction rates in the more polluted areas were the result of lost eggs. Lost eggs can be the result of damaged or cracked eggs tossed out by the parents, eggs lost from precarious nests (e.g., on floating buoys), human interference, or predation. Some of these factors are more prevalent in the more polluted areas because they are also the more urbanized and industrialized areas. The authors concluded that "…the latitudinal trends seen in egg contaminant exposure are unlikely to result from contaminant exposure on the wintering grounds." This idea is somewhat contradicted by the wide range of DDE levels in eggs from each area (overall range of 0.17–4.61 ppm). No fish residues were reported.

The final osprey breeding study to be reviewed is one currently underway in Newport Bay. Newport Bay has historically seen osprey stopping briefly during migration. Ospreys prefer coniferous snags (e.g., dead fir trees) for nesting to minimize predation as well as to have a clear path for flying to and from the nest. The snag must also be close to fishing grounds so that a nesting pair has ample access to food for themselves and their young. Despite excellent fishing grounds, Newport Bay has historically not had this type of tree for nesting ospreys. In 1993, a wooden platform was affixed to the top of a 50 ft pole on Shellmaker Island in an attempt to attract an osprey pair to nest. Nothing happened for more than a decade. Sticks were added in an attempt to start the nest building activity. A pair built a nest in 2005. Mating resulted in two nonviable eggs. The next year, mating resulted in the successful production of two fledglings. Breeding has continued successfully each year by this pair. Tagging of the fledglings began in 2008. A female osprey, fledged at Shellmaker Island in 2008, returned to the area in 2010 with a mate and built a nest on a new platform at the San Joaquin Wildlife Sanctuary (SJWS) in the lower drainage into Newport Bay. This pair produced a single fledgling in 2010. In 2011 the same pair was in the process of raising two chicks, when suddenly both died of unknown cause. Hemorrhaging was noted in their lungs. The adults appeared unharmed and returned in 2012 to successfully fledge two chicks. The reproductive data for the two osprey pairs are listed in Table 22 below.

Table 22 Reproduction of osprey pairs at Shellmaker Island (SI) and San Joaquin Wildlife Sanctuary (SJWS)[a]

Year	Site	Eggs	Chicks	Fledglings	Productivity	Comments
2005	SI	2	0	0	0	First mating
2006	SI	≥3	3	2	2	Egg count not available
2007	SI	≥3	3	2	2	Egg count not available
2008	SI	≥3	3	3	3	Egg count not available
2009	SI	≥4	4	4	4	Egg count not available
2010	SI	3	3	3	3	
2010	SJWS	1	1	1	1	Female SI 2008; first mating
2011	SI	≥3	3	3	3	Egg count not available
2011	SJWS	≥2	2	0	0	Chicks died of unknown cause; hemorrhaging noted in lungs
2012	SI	≥3	3	3	3	Egg count not available
2012	SJWS	≥2	2	2	2	Egg count not available

[a]Data from Thomas (2010), Reed (2010), Reicher (2010), Kerr (2006) as well as personal communications from: Carla Navarro Woods, Reserve Manager, California Department of Fish and Game, who provided breeding data on the osprey pair at Shellmaker Island; Scott Thomas and of the Audubon Society, who provided breeding data for both osprey pairs; Nancy Kenyon of the Audubon Society, who provided breeding data on the osprey pair at the San Joaquin Wildlife Sanctuary

Productivity can be expressed as fledglings per breeding attempt. With 23 fledglings and 11 breeding attempts, the productivity is 23/11 = 2.09 fledglings/breeding attempt. This high level of productivity of ospreys in Newport Bay suggests that DDT levels in eggs are below the threshold associated with hatching failure. Because ospreys are particularly sensitive to the most toxic effect of DDT, namely, on reproduction, they serve as an ongoing sentinel for the potential of DDT to affect all wildlife.

Discussion and conclusions. Considering the information on the effects of DDT on osprey reproduction that has been reviewed and summarized, can a threshold for the action of DDE on reproduction in osprey be determined? A lot is known. However, one is also aware of unknown exposures and the high variability of residues and response. A wide range of endpoints and approaches can be taken.

For a given breeding area, a field study, in which no significant eggshell thinning was found defines the known threshold for any biological effect of DDE residues in those eggs. The threshold for that finding is approximately several hundred ppb DDE. A threshold for increased shell breakage and reduced hatchability is approximately 3–4 ppm DDE. Recovery and stabilization of DDE-poisoned populations of ospreys has been associated with DDE egg residue levels as high as 5–8 ppm.

Although postulated, toxicity has not been shown to occur for DDE residue levels in eggs that cause shell thinning up to 10%. Thinking in evolutionary terms, normal eggshell thickness evolved to prevent breakage during incubation, as well as to provide gaseous exchange and an appropriate degree of hydration. There is a considerable range in normal eggshell thickness. Neither hatching success, nor health of the fledgling appears to be compromised by minimal shell thinning. There is some

uncertainty here, but the recovery, stability and health of populations still experiencing marginal shell thinning, suggests no detrimental effect. In addition to choosing a threshold for toxicity, one must also determine an appropriate biomagnification factor from fish to egg.

Osprey are opportunistic feeders, catching the most nutritious and easiest to catch species at any given location and time. Typical prey species vary with season, latitude and whether the location is coastal or inland. One should expect, therefore, some variation in biomagnification from fish to eggs. The variation in literature biomagnification values, however, appears more related to a lack of representative sampling of fish from breeding grounds and a lack of data on residues in fish from wintering grounds.

The flounder, menhaden and largescale sucker appear to be the most important food species for osprey studied in North America. The largescale sucker is a fresh water species. Only the menhaden is among the species relied upon by the NAS panel in setting the marine fish recommendation to protect wildlife. For the 22 determinations of biomagnification from fish to egg determined from data in the reports above, there is considerable uncertainty. Therefore, the best estimate from these data for what constitutes an appropriate biomagnifications factor might be the median value of 10 (0.73–87, n = 22). Values based on fish scraps cast from the nest range from 1.6 to 31 (n = 5) with a median of 10.9. For reasons explained previously, a value of 10 is most likely to be high. For example, the two values from nonmigratory populations were 0.73 and 3.8.

A recommendation for DDT residues in marine fish should not consider DDD, because DDD has not been shown to cause shell thinning and is not converted to DDE. DDE causes eggshell thinning and DDT can be converted to DDE. Thus, DDT and DDE are the important terminal residues.

If the recommendation is to protect the osprey as a sensitive representative for other fish-eating species, then one needs to select a threshold level in eggs and divide by an appropriate biomagnification factor. If one were to use a threshold that is half of the approximate lower end of the hatchability effect threshold and divide that value by a biomagnification factor of 10, the recommendation would be 150 ppb in fish. This level is three times what the NAS panel recommended, but is based on additional information that they appeared to overlook or wasn't known until after 1972. The value of 150 ppb also served as the basis for the current National criterion for the water column discussed below.

3.2.3 US EPA Water Column Guidance to Protect Wildlife

In 1980, the US EPA published criteria for the protection of wildlife from DDT in the water column. The criterion was adopted as the California Toxics Rule (CTR) standard in 2002. The wildlife criterion of 1 pptr was based on the bioaccumulation of DDT from water into fish. A fish target residue was chosen to be 150 ppb from a study by Anderson et al. (1975) in a population of brown pelicans recovering from the reproductive effects of DDT. Accurate analysis of pptr levels of DDT in water is

difficult and produces uncertain results, limiting the utility of the criterion. Measuring levels of DDT in fish is much easier and more certain. Therefore, a criterion in fish is more useful than a criterion in water. This brings us to the question of whether the 150 ppb DDT residue level in fish, which is the basis for the National criterion and CTR standard in water, will protect wildlife, considering what is known today. To remain consistent with the criterion, the fish residue should protect the brown pelican, one of the most sensitive species to the reproductive effects of DDT. This question is addressed in the following review of studies of DDT effects on reproduction in brown pelicans.

A summary of the 1980 US EPA criterion is reproduced below:

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

The particular pelicans studied by Anderson et al. (1975) were reported to be feeding on northern anchovies. The northern anchovy diet of the recovering population of brown pelicans became the basis of the EPA chronic criterion to protect wildlife. The fish residue of 150 ppb is based on a study in which the brown pelican population was still recovering from the reproductive effects of DDT. The level of reproduction was judged to be inadequate to sustain the population. However, the authors referred to a slow reduction of DDE residues in brown pelican eggs compared to the fish diet. The fish residue had declined 27-fold during a period in which the egg residues had declined only ninefold. Moreover, DDT and DDE were detected in fish in 1974, but only the more stable DDE was detected in brown pelican eggs that year. Therefore, DDE in brown pelicans and in their eggs appears to have not

reached a steady-state with the more rapidly declining residues in the aquatic environment. If we assume that in time the DDE residue in the eggs would also decline 27-fold, the final egg residue would have a geometric mean of 1.7 ppm. Would this level be a no-effect level for reproductive effects in the brown pelican?

To answer this question, let us review in chronological order some of the key studies of the effects of DDT on various populations of brown pelicans during and after the DDT era. The recovery of brown pelicans following the ban of DDT in 1972 provides a measure of dose-response and thresholds for the reproductive effects of DDT. Some of the earlier studies have been reviewed by Ware (1975).

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

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

The DDT congener *p*,*p*′-DDE was the major cause of eggshell thinning in raptorial and fish-eating birds (Risebrough et al. 1969). The peregrine falcon, bald eagle and osprey were in decline due to DDE eggshell thinning. There was no evidence of thinning in eggshells of species that prey mostly on mammals, such as the Red-tailed hawk, golden eagle and great horned owl. Brown pelicans had declined 50% in the past 4 years at Point Reyes. Brown pelican and double-crested cormorant reproduction on the Channel Islands and Islas Coronados near San Diego were decimated in 1969. Western gull eggs on Anacapa Island in 1969 were normal. Some eggshell thinning is evident in ashy petrel and murre from the Farallon Islands. A "No effect" level has not been established for eggshell thinning. The relationship between DDE residues and eggshell thinning is linear with an absence of a "no effect" range of concentrations. DDE plus DDD in eggs from white pelicans, at levels ranging from less than 0.5 to 6 ppm, were associated with significant eggshell thinning.

"The complex series of behavioral events that lead up to mating, nest building, and egg laying were evidently not adversely affected." The likely mechanism of action is inhibition of calcium transport and mineralization in the shell gland. In the brown pelican, eggshell thickness is reduced about 15% at 75 ppm DDE on a lipid basis (3.3 ppm fresh weight). At higher residue levels the slope of the residue-thinning curve decreases to zero thickness at 3,000 ppm DDE (132 ppm fresh weight).

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

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

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

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

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

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

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

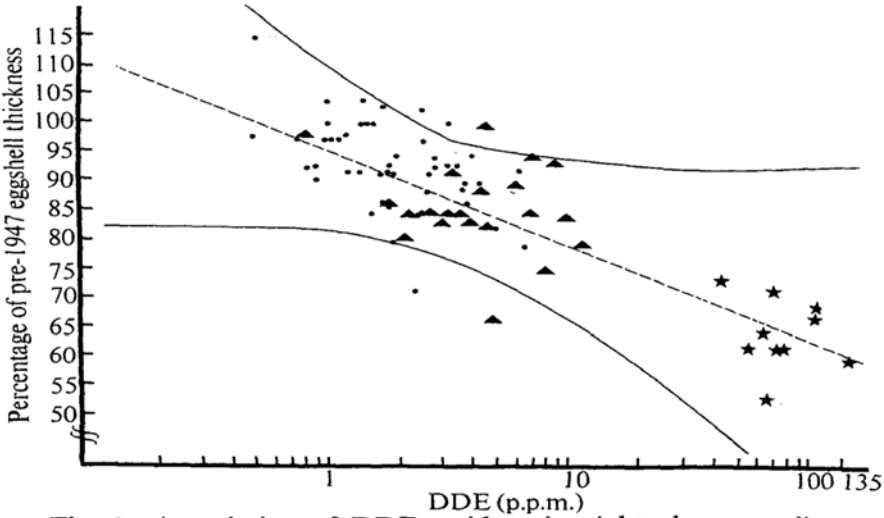

Fig. 1 Association of DDE residues in eighty brown pelican eggs from Florida (●), South Carolina (▲) and California (★) with the % of pre-1947 eggshell thickness. Solid lines represent 95% confidence limits. $\hat{Y}=95.787-15.689 \log_{10} X$; $r=-0.80$ ($P<0.01$).

Fig. 15 Relationship of DDE in eggs and eggshell thinning in brown pelicans. Figure 1 in Blus et al. (1972a) reproduced with permission from the author and from Nature

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

Schreiber and Risebrough (1972) published a review of the status of the brown pelican in the United States and Baja, Mexico. They also reported on Schreiber's work on brown pelicans in Florida. Hatching success in Florida decreased sharply with increasing frequency of inspection by wildlife biologists. The lipid content of Florida eggs was 5.0%. The authors claimed that very low concentrations of DDE were associated with significant thinning and that the relationship is linear from zero concentrations of DDE. Thinning of eggshells greater than 20% usually causes them to break during incubation. Total DDT residues in eggs collected in 1969 and 1970 in Florida were 1.2–2.9 ppm. The 9% reduction in eggshell thickness in Florida had not yet had an observable effect on population stability. There was no evidence that 9% shell thinning has an effect on gas exchange or water retention.

Keith and Gruchy (1972) published a comprehensive review of the past 5 years of reports on the effects of DDE on avian wildlife. They noted a wide species variation in eggshell thinning response to DDE residues (Fig. 16).

Jehl (1973) reported on the status of brown pelicans on islands off the west coast of Baja, California. Breeding was severely impacted at most of the locations, with empty nests and broken shells. Observations were complicated by destruction of

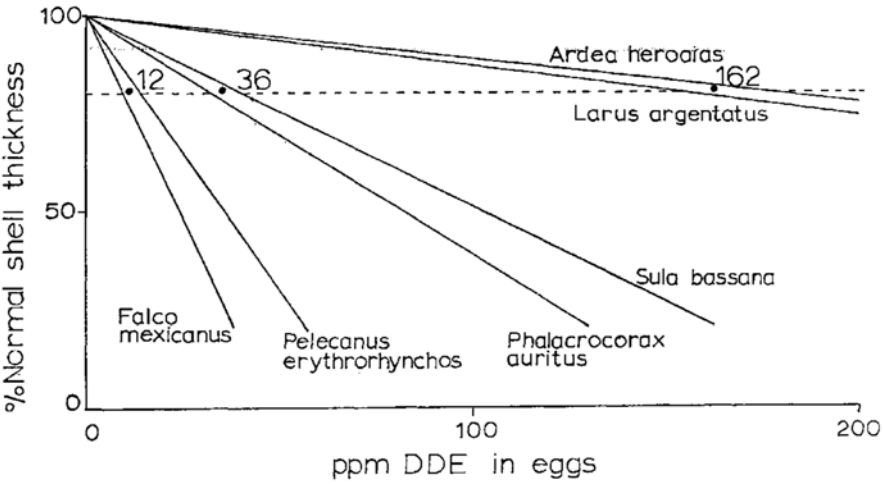

FIGURE 7. Variation between species in reproductive response to DDE. DDE values are of whole eggs on a wet-weight basis. Twenty-percent reduction in shell thickness (population damage threshold) is shown by a dotted line, and the calculated DDE values at that thickness are shown as 12, 36, and 162 ppm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

Fig. 16 Species variation in eggshell thinning in response to DDE egg residues. Figure 7 in Keith and Gruchy (1972) reproduced with permission

pelican eggs by gulls whenever nests were unattended. The source of DDE was attributed to the Los Angeles outfall. The dose-response for DDE in eggs and shell thinning is presented in Fig. 17 (DDE concentration in ppm lipid).

Blus et al. (1974a) reported on studies of brown pelican eggs collected in 1969 and 1970 from California, Florida and South Carolina. Eggshells were thinner than pre-DDT era eggshells. DDE residues were highest in California eggs and lowest in Florida eggs. Shell thinning was highly correlated with levels of DDE in the eggs. The calculated no-effect level was 500 ppb DDE. Thinning was 4% at 1 ppm and 15% at 5 ppm. The observed logarithmic relationship was also reported by others for the double-crested cormorant and the prairie falcon. Dieldrin may have contributed to reproductive failure of brown pelicans. Serious population declines have occurred in California and South Carolina as a result of DDE eggshell thinning. "The 17% eggshell thinning observed in South Carolina was associated with subnormal reproductive success." In areas with the greatest eggshell thinning, "Usually, the entire clutch exhibited the extreme thinning, and all the eggs were broken in some nests." Florida eggs from different breeding areas averaged 0.69–2.48 ppm DDE, with an average of 8% shell thinning. "…the bulk of the residues in all areas of Florida are low enough that one would not expect these residues to induce widespread, long-term, adverse effects on the populations there." The log-linear relationship between DDE residues in eggs and shell thinning is illustrated in Fig. 18 below.

A more systematic study was done in 1971 and 1972 by Blus et al. (1974b) in a breeding colony of brown pelicans in South Carolina. One freshly laid egg was

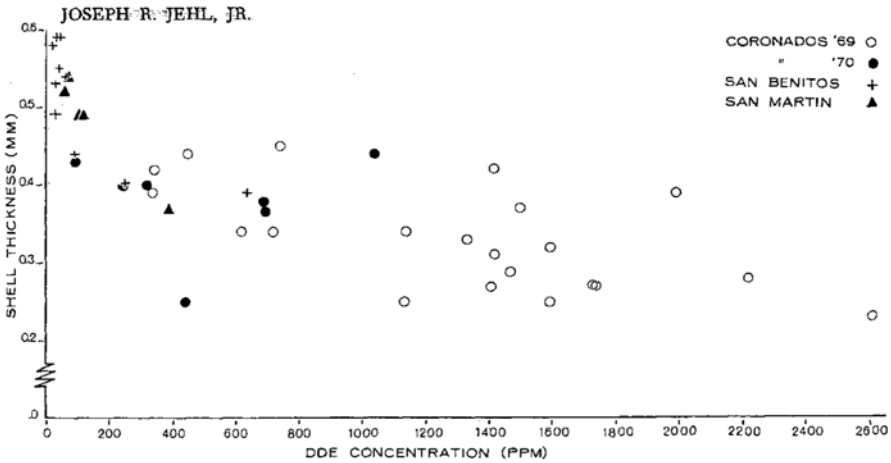

FIGURE 3. Relationship between DDE concentration and shell thickness in Brown Pelican eggs from northwestern Baja California.

Fig. 17 DDE dose-response in eggshell thinning in brown pelicans on the islands off the west coast of Baja California. Figure 3 in Jehl (1973) reproduced with the permission from the author and from The Condor, published by The Ornithological Society

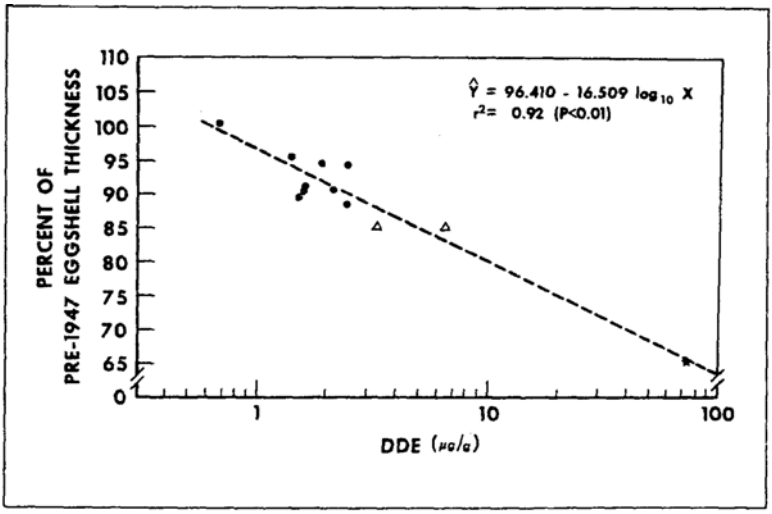

FIGURE 2.—*Association of DDE residues in brown pelican eggs from nine collections in Florida [•], two colonies in South Carolina [△], and one colony in California [★] with percent of pre-1947 eggshell thickness.*

Fig. 18 Log-linear relationship between DDE residues in eggs and eggshell thinning in brown pelicans from Florida, South Carolina and California. Figure 2 reproduced from Blus et al. (1974a)

Table 23 Early recovery of brown pelican reproduction off Baja and California coasts. Table 1 in Anderson et al. (1975) reproduced with permission from the author and from AAAS

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

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

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

taken from each of 93 marked nests. In this way, residue level and shell thinning could be related directly to nest success. The effects of DDE on eggshell thinning and reproductive success were confounded by dieldrin. Reproductive success was normal in those nests in which a sample egg contained less than 2.5 ppm DDE.

Anderson et al. (1975) published the first report of the recovery of the brown pelican following the ban of DDT in 1972. The major source of DDT for the study populations was the wastes of the DDT manufacturer being released into the ocean by way of the Los Angeles County Sanitation District's waste water outfall at White's Point. Releases were greatly reduced after April, 1970. Recovery of brown pelican reproduction on offshore islands to the north and south improved quickly during the period 1971–1974 (Table 23).

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

Direct observation confirmed that the northern anchovy was the major food item for this breeding colony of brown pelicans. The authors (Anderson et al. 1975) stated: "During banding at Anacapa from 1972 to 1974, we examined stomach contents regurgitated by young pelicans; the material consisted almost exclusively of anchovies. Our observations of feeding adults before and during the breeding season also indicated a heavy reliance on anchovies."

Residues of DDE in northern anchovies decreased 27-fold from 1969 to 1974 while during the same period, DDE in brown pelican eggs decreased ninefold (Table 24). The slower decline in egg compared to fish residues suggests that at a steady-state, the 150 ppb total DDT measured in northern anchovies in 1974 would result in an egg residue that is below the threshold for a reproductive effect.

Anderson et al. (1977) continued to study brown pelicans on Anacapa Island in 1975. The only breeding colonies in California observed by these investigators were on Anacapa Island and nearby scorpion rock. Only four eggs were collected and three of these were putrified. Lipid content of eggs was assumed to be 5%. DDE egg residue analysis, shell thickness and productivity appeared to have leveled off in 1975, following the recovery from 1969 to 1974. PCB egg residues were 5–10 ppm during this period. The data are summarized in Fig. 19. PCBs may have affected reproduction,

Table 24 Decreases in DDE in northern anchovies and brown pelican eggs off the Southern California and Northwestern Baja California coasts during 1969–1974. Table 2 in Anderson et al. (1975) reproduced with permission from the author and from AAAS

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

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

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

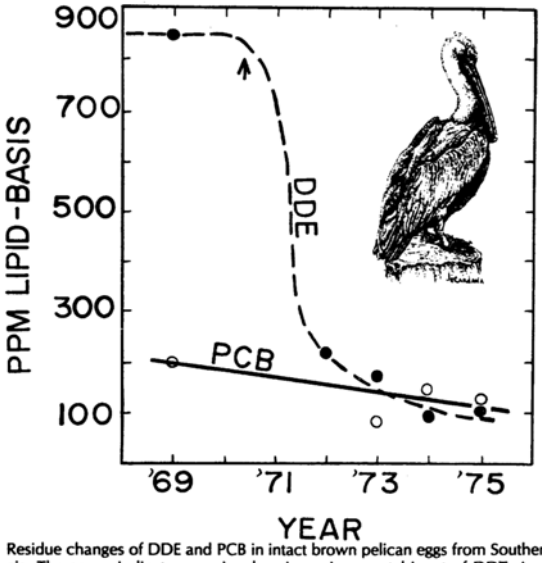

FIGURE 1. Residue changes of DDE and PCB in intact brown pelican eggs from Southern California. The arrow indicates a major drop in environmental input of DDT. According to published data, major input of DDT ceased in 1970 (Anderson et al. 1975) and by 1971 it had decreased to about 0.5% of previous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

Fig. 19 DDE levels in brown pelican eggs from Southern California. Figure 1 in Anderson et al. (1977) courtesy of the author and California Fish and Game

Table 25 DDT and metabolites in Atlantic menhaden regurgitated by brown pelicans in South Carolina in 1973. Data from Table 14 in Blus et al. 1977

Residues (μg/g fresh wet wt)			
DDE		TDE(DDD)	DDT
0.04		0.04	0.04
0.06		0.04	0.05
0.07		0.02	0.03
0.06		0.03	0.03
0.05		0.03	0.02
0.08		0.03	0.02
0.15		0.07	0.06
GM[a]	0.067	0.035	0.033
CL[b]	0.045–0.099	0.024–0.050	0.022–0.049
Range	0.04–0.15	0.02–0.07	0.02–0.06

[a]GM = geometric mean
[b]CL = 95% confidence limits

preventing the full recovery of the colony. Limited observations in 1976 suggested that an inadequate food supply also contributed to low productivity.

In 1977, Blus et al. published a follow-up report on the brown pelican breeding colonies in South Carolina. Shells of eggs collected from 1969 to 1973 averaged 14–17% thinner than shells of eggs collected prior to the DDT era. Crushed shells were thinner than shells from eggs that hatched. Shells of freshly laid eggs were thinner than shells of hatched eggs. Residues of DDE in eggs decreased from 5.45 ppm in 1969 to 2.09 ppm in 1973. Reproductive success of 1.66 per nest in 1973 was considered excellent. Atlantic menhaden, a major food item of the brown pelican, contained a residue of 0.135 ppm total DDT as shown in the author's Table 14 that is reproduced in Table 25 below. The menhaden were recovered from regurgitated stomach contents in 1973. The biomagnification value for total DDT from fish to egg was 18. The total DDT residue in menhaden in the late 1960s was 0.295 ppm. "The migratory habits of the Atlantic menhaden (15, 17) and the brown pelican confound the significance of biomagnification noted in this study."

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

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

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

Mendenhall and Prouty (1978) studied recovering populations of brown pelicans in South Carolina. A steady decline in DDE residues in eggs had a high negative correlation with increasing eggshell thickness (Fig. 20). Eggshell thickness in 1978 was only 6% below the pre-1947 mean thickness. Fledgling rates continued to increase and reached a population sustaining level in 1976 (Table 26). The authors noted that in 1977 all eggs sampled were below 2.5 ppm DDE. DDE levels above 2.5 ppm had been associated with consistent nest failure.

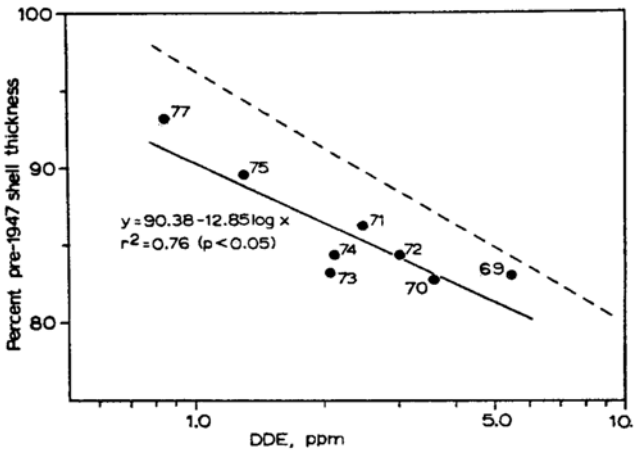

Fig. 1. (——) Change in eggshell thickness for South Carolina brown pelicans as related to DDE residues, 1969-1977. Each point shows mean shell thinning in relation to pre-1947 data (y) and mean wet-weight DDE residue (x) for one year. Sources of data as in Table 2. (----) Regression for 12 colonies in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

Fig. 20 Negative correlation between egg DDE residue and shell thickness for South Carolina brown pelicans. Figure 1 in Mendenhall and Prouty (1978) reproduced with permission

Table 26 Recovery of reproductive success in brown pelicans from South Carolina from 1969 to 1978. Table 3 in Mendenhall and Prouty (1978) reproduced with permission

TABLE 3.

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

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

* Approximate figure; see text.

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

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

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

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

An overall decline in organochlorine residues in brown pelican eggs is illustrated in data from Blus et al. (1979b), which is plotted in Fig. 21 below.

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

Blus (1984) reported a comparison of regression and sample egg methods for predicting the reproductive effects threshold for DDE. Brown pelican eggs from California, Florida, Louisiana, and South Carolina were analyzed for DDE residue, eggshell thinning, and these were then compared to reproductive success. Eggshell

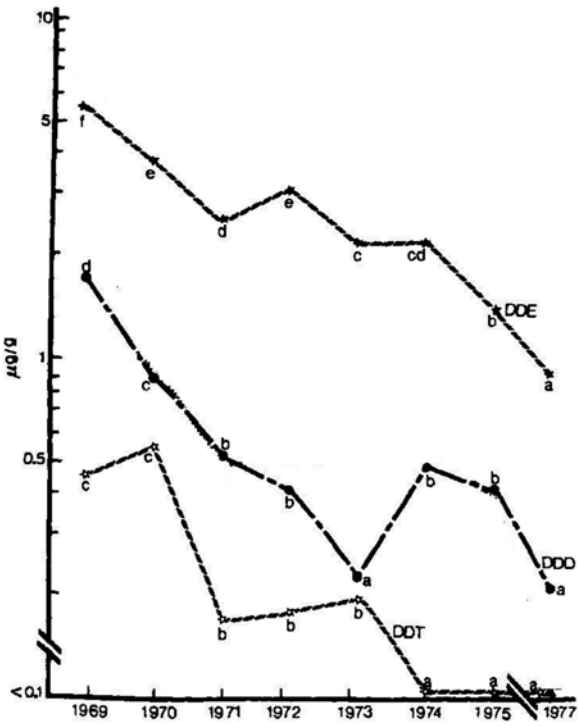

Fig. 21 Decline in DDT and metabolites in eggs of brown pelicans collected in South Carolina from 1969 to 1977. Data from Figure 1 in Blus et al. (1979b)

thinning of 18% or greater had been reported to be associated with reproductive failure and population declines. An DDE egg residue of 5 ppm was associated with 18% shell thinning by regression analysis (Fig. 22). Using the sample egg method, reproductive effects occurred at 3 ppm, with a threshold between 2.5 and 3 ppm DDE (Fig. 23). The critical level of 3 ppm is associated with eggshell thinning of 16% from the regression analysis of 813 eggs plotted in Blus's Figure 1.

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

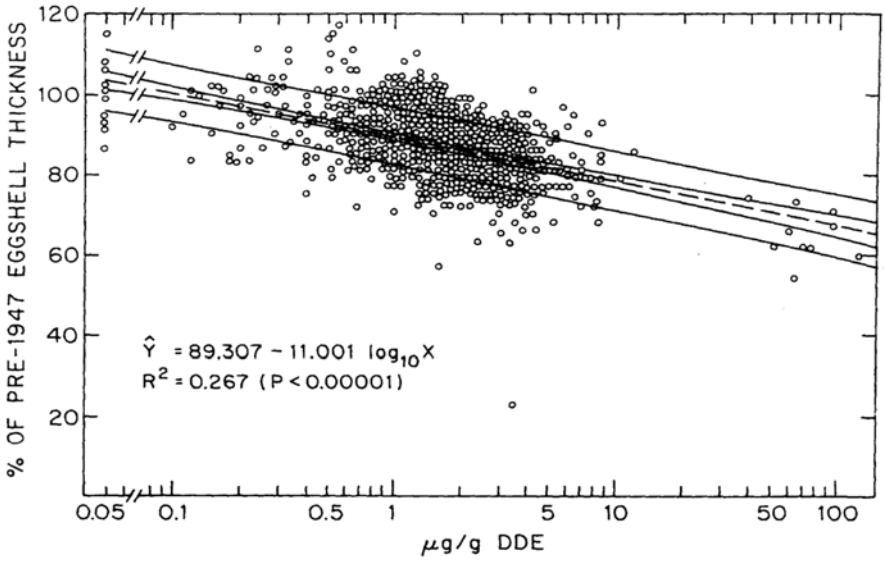

FIG. 1. Regression analysis showing the relationship of DDE residues in 813 eggs of Brown Pelicans to eggshell thickness; South Carolina, Florida, Louisiana, and California, 1969–1976. The dashed line is the regression line, the two pairs of solid lines delineate the 95% confidence limits for the population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

Fig. 22 Regression of DDE residue and shell thickness in brown pelican eggs collected from 1969 to 1976. Figure 1 in Blus (1984) reproduced with permission from the author and from The Wilson Journal of Ornithology

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

In 1995, Franklin Gress published his doctoral thesis on 22 years of studies of brown pelicans on Anacapa Island. DDE residues in eggs declined slowly during the late 1970s and 1980s to approximately 2 ppm in 1992. Eggshells increased in thickness during this period. Thinning was about 5% in 1992. Gress concluded: "… at present we have no evidence that brown pelican reproduction in the SCB is measurably impaired by DDE-related eggshell changes…" The only breeding colonies in the Southern California Bight (SCB) are on West Anacapa Island, Santa Barbara Island and Islas Los Coronados.

Discussion and conclusions. To summarize the key findings in the above chronology, brown pelican reproduction was reduced by the direct action of the DDT metabolite, DDE, during and after the DDT use era. DDE was magnified up the aquatic food

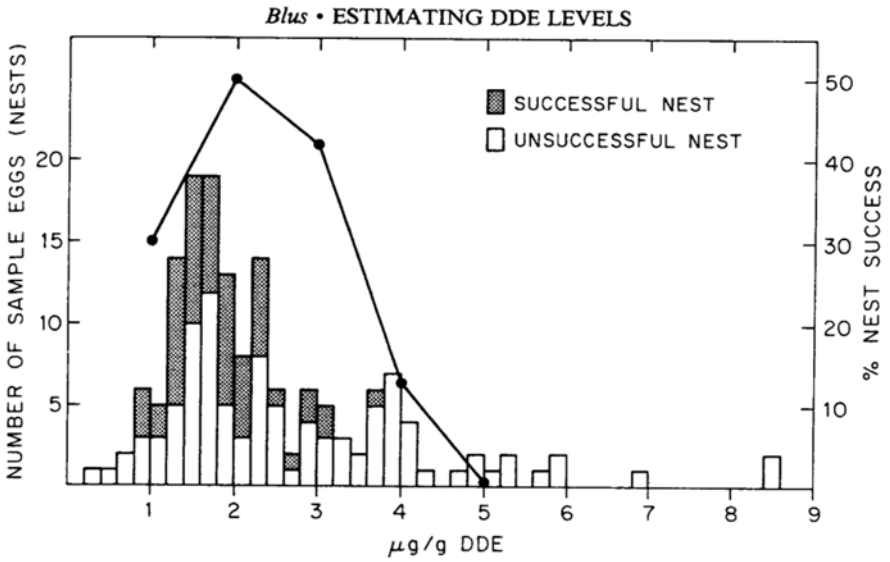

FIG. 2. Relationship of DDE residues in 156 sample eggs of Brown Pelicans to nest success. Bars represent success related to 0.2 μg/g intervals; dots on the line represent mean nest success by μg/g intervals.

Fig. 23 Threshold for DDE reproductive effects in brown pelicans. Figure 2 in Blus (1984) reproduced with permission from the author and from The Wilson Journal of Ornithology

chain to the fish diet of the brown pelican and was deposited in the lipid of the eggs. DDE residues exceeding 2.5 ppm in eggs were associated with eggshell thinning in excess of 15%, resulting in decreased hatching success. DDE egg residues below 2.5 ppm, although capable of producing measureable thinning of eggshells, were not associated with reduced hatching success or any other effect on reproduction. DDE residues in all populations of brown pelicans in the United States are currently below the threshold for reduced hatching success.

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

residues in eggs collected on Anacapa Island. Breeding colonies further south, off Baja California, have much lower egg residues.

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

Reports of DDE residues in the northern anchovy diet of brown pelicans were not found in published literature after 1975. Therefore, a confirmation of the biomagnification from fish diet to eggs of approximately 11, estimated from the Anderson et al. (1975) 1969 data from intact eggs, is not available.

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

3.2.4 Canadian Fish Guidance to Protect Wildlife

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

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

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

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

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

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

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

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

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

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

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

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

These assumptions and the procedures for deriving the TRG for DDT in fish were from a protocol document 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.

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 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. 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. 24).

The same data are summarized in Appendix 16 of the Assessment document (Environment Canada 2000) reproduced in Table 27 below. 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

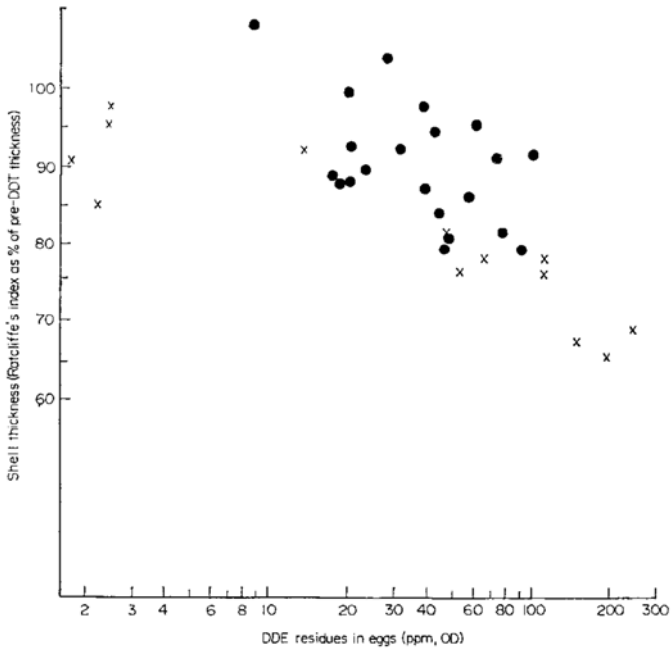

FIG. 3. Relationship between mean clutch shell-thickness and DDE residue of kestrel eggs collected in Ithaca, New York during 1970 (●) and same relationship experimentally induced with dietary DDE (×).

Fig. 24 Influence of laboratory and field dietary exposures to DDE on shell thickness in kestrel eggs collected in Ithaca, New York. Figure 3 in Lincer (1975) reproduced with permission from the author and from John Wiley & Sons

Table 27 Effect of dietary DDT on eggshell thickness in birds. Appendix 16 reproduced from Environment Canada (2000)

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

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 is more appropriately based on 0.5 mg/kg-day and not on the 0.13 mg/kg-day value used by Environment Canada.

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 sparrow hawks 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 sparrow hawk, which is calculated from a dietary level of 3 ppm. If one accepts the tenfold uncertainty factor for variability in species susceptibility, the one remaining variable to consider is the rate of dietary intake.

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 petrel's 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. (1982b) 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 0.75–6.81 ppm. The eggs were collected in 1972 and 1976 off the east coast of Canada. The authors reported measuring shell thickness, but no data were published. The authors claimed that 12 ppm DDE in eggs produced 20% shell thinning. This conclusion was based on an extrapolation of the residue shell-thinning data. Again, no data or regression plots were present 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. 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 among the listed species.

Using the Environment Canada method, the reference concentration can be 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 × 0.167/0.2 = 250 ppb.

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 shell thinning instead of hatching failure as the toxic endpoint and using data from an insensitive species for estimating the maximum food intake rate.

The Canadian guidance is 11 times more protective than the 150 ppb fish residue level that is the basis for the National criterion as well as a level protective of osprey derived in this review. Not every community can afford a level of protection as high as that provided by the Canadian guidance. Lower levels of protection have been shown to provide healthy wildlife populations at far less expenditure of limited resources. Continuing declines in fish residues of the DDTs will continue to increase the degree of protection for wildlife.

3.2.5 US EPA Region IX BTAG Fish Guidance to Protect Wildlife

The Navy/US EPA Region 9 Biological Technical Assistance Group (BTAG) developed toxicity reference values (TRVs) for ecological risk assessments. BTAG TRVs have been published by the California Department of Toxic Substances Control (2008). For the DDTs, the low TRV is 0.09 mg/kg-day. This value is borrowed from the US EPA Great Lakes Criteria (US EPA 1995). The US EPA derived the TRV from the Anderson et al. (1975) finding of a LOEL at 150 ppb DDTs in northern anchovy. The TRV is three times more protective than the US EPA national water criterion that also relies on the Anderson et al. (1975) study. The difference in the two US EPA criteria is the use of an uncertainty factor of 3 in the Great Lakes criterion to estimate a NOEL from a LOEL. This value for uncertainty was used despite the authors acknowledging that DDTs in brown pelicans and northern anchovies

were likely not at a steady state in the 1975 data from Anderson et al. The Great Lakes study estimated a sevenfold lower egg residue than found by Anderson et al. (1975), if brown pelicans took 2 years to reach steady state with the DDTs in northern anchovy. Two years is a reasonable estimate based on the very slow excretion of the DDTs and the 10–15 year lifespan of the brown pelican, compared to the 1–2 year lifespan of northern anchovy. Consideration of these facts supports the 150 ppb level in fish as a NOEL, when a steady-state is reached. The use of the BTAG fish guidance provides an extra threefold level of protection beyond that provided by the National criterion.

3.2.6 California EPA Sport Fish Guidance for DDT to Protect Human Health

The SARWQCB (2006) has concurred with U.S. EPA Region IX (2002) to use 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) and a later update (Brodberg and Pollock 1999). The following is a review of the sport fish guidance developed by OEHHA.

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 Sanitation District's wastewater outfall at White's Point. 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 OEHHA report stated that:

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

and that:

> 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 stated in the report as follows:

> 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 US 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), 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 advisory for Newport Bay.

More recently, OEHHA has revised the fish advisory for DDT (Klasing and Brodberg 2008). The recommendation is an tissue advisory level of 520 ppb for DDT. This guidance weighs the cancer and noncancer risks of DDT against the benefits of eating fish.

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 use with genotoxic carcinogens. The weight of evidence indicates that DDTs are not genotoxic. This point is made for DDE in a chapter on carcinogenesis (Pitot and Dragan 1996) in the most widely used text in toxicology. The authors indicated that DDE is not mutagenic and acts as a promoter. This conclusion is further explained in a publication from the Pitot laboratory (Holsapple et al. 2006) as quoted below.

> ***Mode of action and human relevance of phenobarbital-like rodent liver carcinogens***. Phenobarbital is the prototype of several rodent hapatocarcinogens (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 carcinogenic 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 (US FDA 2007).

The OEHHA guidance dealing with the risk of human cancer from ingestion of sport 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 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, the 520 ppb target is met and even the older guidance of 100 ppb is met. There is no impairment of sport fishing in Newport Bay.

4 Chlordane

4.1 Levels in the Environment

The following sections present available chlordane data for Newport Bay and Watershed. Downward trends in chlordane concentrations—particularly in fish tissue and mussel tissue—are evident in data collected for almost 20 years.

4.1.1 Agricultural Soils

The half-life of chlordane in soil is estimated at 350 days (or approximately 1 year), but can range from 37 to 3,500 days (approximately 10 years) (Hornsby et al. 1996). Chlordane is persistent in soils and volatilization is believed to be the major removal mechanism (US DHHS 1994). Chlordane data for agricultural soil are available for the Newport Bay watershed for the years 1989, 1990, 1995, 2000, 2002, 2004 and 2006 (Table 28).

Samples were taken from different locations and different years with the purpose of assessing site conditions for planning and development and not to establish concentration trends over time in the Watershed. The vast majority (approximately 95%) of chlordane soil samples returned concentrations below detection limits. Detectable concentrations ranged between 47 and 240 ppb.

Table 28 Chlordane concentrations in agricultural soils in the Newport Bay Watershed

Year	0-12 inch Sample Depth			12-24 inch Sample Depth			>24 inch Sample Depth			Detection Limits (ppm)
	Range of Detected Chlordane (ppm)	Total Samples	Total Non-detect Samples	Range of Detected Chlordane (ppm)	Total Samples	Total Non-detect Samples	Range of Detected Chlordane (ppm)	Total Samples	Total Non-detect Samples	
1989	0.240 - 0.240	3	2	--	1	1	0.120 - 0.190	5	3	0.08 or 0.12
1990	0.170 - 0.170	2	1	0.210 - 0.210	1	0	0.190 - 0.190	3	2	0.08 or 0.12
1995	0.047 - 0.055	24	22							0.03
2000	--	28	28							0.05 or 0.50
2002	0.050 - 0.130	174	161				--	27	27	--
2004	--	230	230				--	45	45	0.1 - 10.0
2006	0.077 - 0.160	6	1				--	6	6	0.05

Sources: Unpublished technical reports provided by The Irvine Company (1985–2006)
Data were not available for shaded areas

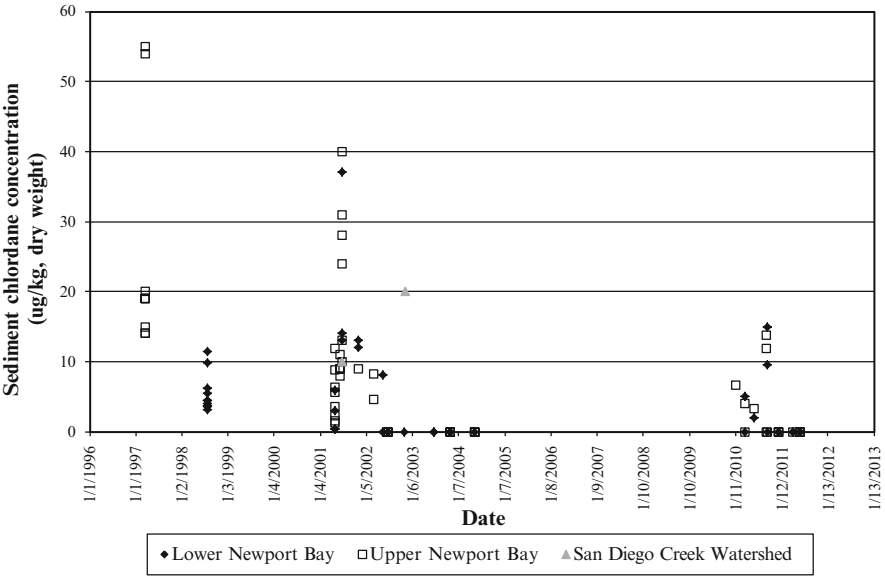

Fig. 25 Chlordane concentrations above method detection limits in sediments from Newport Bay and Watershed (1997–2011). *Sources*: Masters and Inman (2000); SCCWRP BIGHT 98 Survey (1998); Bay and Greenstein (2003); Bay et al. (2004); unpublished technical reports provided by The Irvine Company (2000–2004); SARWQCB (2006); Orange County Watersheds (2010–2011)

4.1.2 Sediments

Chlordane levels in Bay and Creek sediments are available for lower Newport Bay, upper Newport Bay, and San Diego Creek for the period 1997 through 2011 (Fig. 25). As with DDT, it is difficult to infer 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 among 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 resulted 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 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, correlated with storm events and wetter-than-average rainfall years. 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 notable that since 2002 Bay and Creek sediment samples have exhibited chlordane concentrations below 20 ppb.

4.1.3 Water Column

Data from 1998 to 2009 reveal a range of chlordane concentrations in water from Newport Bay and Watershed (Bay and Greenstein 2003; Bay et al. 2004, Orange County Watersheds).[7] Ten of 91 samples were above detection limits with a range from 2.5 to 186.5 pptr (ng/L). Detection limits ranged from 1 to 130 pptr. Detection limits were not reported for 8/10 of the samples above the detection limit, including the highest five values (22.5–130 pptr). The accuracy of chlordane analysis in water at the pptr level is questionable due to chromatographic interference caused by high background levels of organic matter and the binding of chlordane to microscopic (filterable) macromolecular organic matter.

4.1.4 Fish and Mussels

Red shiner tissue concentrations may be taken as an indicator of chlordane concentrations in the Watershed, as red shiners are local, short-lived species. For this species, chlordane tissue concentrations dating from 1983 show a substantial decline over time. First-order decay constants were derived using historical red shiner tissue data for the entire time period (1983–2000) and for two subsets of the data set (1983–1991 and 1992–2000). The equations of these decay curves are indicated in Figs. 26 and 27. Figure 27 also shows projected chlordane concentrations through 2010. Data are not available to confirm these concentrations.

When all red shiner data are considered together, a statistically strong (R^2 value of 0.774) downward trend in chlordane concentration is evident. Exponential decay curves fit to the two subsets of data revealed consistent downward trends during

[7] Personal communication from Amanda Carr at Orange County Watersheds.

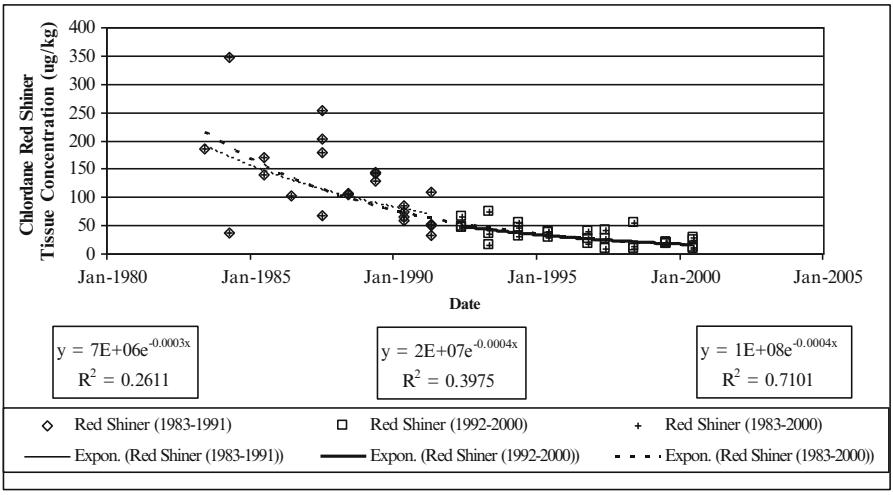

Fig. 26 Chlordane concentrations in red shiner, San Diego Creek and Peters Canyon Wash (1983–2000). Data from California Toxic Substances Monitoring Program (1983–2000). Red shiner data from 2002 to the present are not available

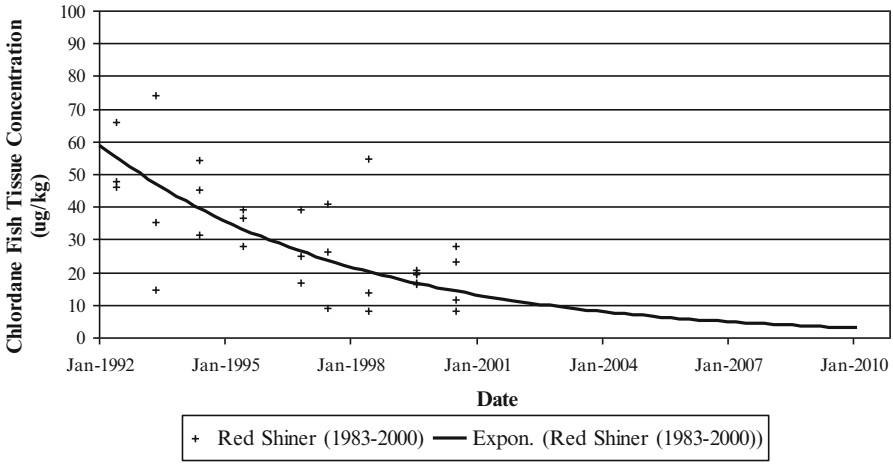

Fig. 27 Chlordane concentrations in red shiner in San Diego Creek and Peters Canyon Wash projected through 2010. Data from California Toxic Substances Monitoring Program (1983–2000). Red shiner data from 2002 to the present are not available

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 data set (1983–2000) is equivalent to a half-life of 4.1 years.

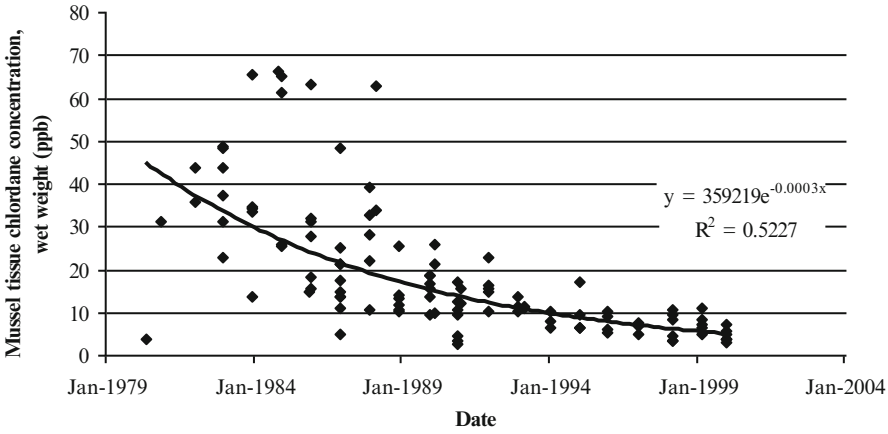

Fig. 28 Chlordane concentrations in mussels from Newport Bay and Watershed. Data from California Mussel Watch Program, 1980–2000. The Mussel Watch Program was ended in 2000

Like red shiner data, mussel tissue data dating to 1980 from Newport Bay show decreasing chlordane concentrations (Fig. 28). 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 1980–1989 and 1990–2000. Although the split analysis indicated that neither the earlier ($R^2 = 0.1176$), nor the later period ($R^2 = 0.2968$) demonstrate as statistically strong a decline as the complete period, the entire mussel data set (1980–2000) reflects a statistically significant decline in chlordane tissue concentrations that is equivalent to a half-life for chlordane of 6.2 years (decay rate of –0.00031 per day or –0.11 per year).

4.2 Benthic Triad Analysis of Impairment

The requirement for a TMDL for chlordane in Newport Bay is based on a triad assessment of sediment chemistry, sediment toxicity to benthic organisms and degradation of benthic communities. In this section, we examine the science underlying the triad to determine 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.

The sediment triad has three components. The first component is the concentration of chlordane in sediments from Newport Bay. The SARWQCB staff report contains data on chlordane levels in sediments from several reports (Appendix A-2 to SARWQCB 2006). Levels of chlordane in sediments from Newport Bay ranged from <1 to 55 ppb in the time period 1994–2011 (see Fig. 25 above). The overall average appears to be less than 10 ppb. As noted above, chlordane levels are declining in the Watershed.

Table 29 Correlation of sediment concentrations of metals and organics with amphipod toxicity. Table 30 reproduced from the Bay Protection and Toxic Cleanup Program report (CSWRCB 1998) Table 30 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

4.2.1 Correlations of Sediment Residues and Benthic Toxicity

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 29, which is reproduced above from the Bay Protection and Toxic Cleanup Program report (CSWRCB 1998).

The correlation coefficient was –0.38, and was significant at the 0.05 level. Higher correlations were observed in the same samples with percent fines, total organic carbon, and eight metals. The chlordane residue level was not correlated with inhibition of purple sea urchin fertilization or larval development in sediment pore water samples from Newport Bay (SWRCB 1998). The chlordane residue level was correlated with inhibition of purple sea urchin larval development in data collected in the entirety of the area regulated by the SARWQCB (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, the toxicity observed 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 reported to be correlated with chlordane concentrations in sediments from Newport Bay (Table 29 in CSWRCB 1998).

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 levels could well be explained by the presence of 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 the existence in the sediment and water of unmeasured organic compounds, possibly organophosphorus or pyrethroid insecticides. The authors of the Bay Protection and Toxic Cleanup Program report (CSWRCB 1998) also noted the presence of uncharacterized organic compounds that could have contributed to sediment toxicity.

4.2.2 Effects Range Median (ERM)

Since the SARWQCB staff report (2006) compared chlordane levels to the ERM (Long and Morgan 1990), the data underlying the ERM for chlordane becomes important to any conclusions reached by the triad analysis of impairment. The ERM is calculated from 12 data points reproduced below (Table 30) from Long and Morgan (1990).

The first two data points (0.3 and 0.6 ppb chlordane) were 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 K_{oc} values (Pavlou et al. 1987). These two data points are in error for two reasons. The first reason is that the K_{oc}

Table 30 Twelve data points used to calculate the ERM for chlordane. Table 33 reproduced 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*
<5,800.0	SSB LC50 for *N. virens*

values used are outdated, because they are not based on the superior slow-stir technique (de Bruijn et al. 1989). Second, the high variability in the outdated K_{oc} values is not seen in those derived by the slow-stir method, precluding the necessity of using the lower 95th and 99th percentile of the K_{oc} values. Multiplying the K_{oc} value for chlordane published by US EPA Region IX (2002) and the SARWQCB staff (SARWQCB 2006) by the chronic marine CTR standard gives a single data point of 65 ppb chlordane in sediment containing 1% OC.

The last two data points in Table 30 are based on spiked sediment bioassays (McLeese and Metcalfe 1980; McLeese et al. 1982). These two bioassays are used to assess toxicity primarily from the water column rather than from sediment. The first study was done with sand shrimp (*Crangon septemspinosa*) and involved adding an unreported amount of chlordane to a beaker, drying off the solvent, adding water and coarse sand (0.28% OC; 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 between the two studies is that in the second study the organism was a polychaete worm (*Nereis virens*) and the sediment was sandy silt that contained 2% OC. 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. The test organism would be added only after equilibrium was achieved.

Sediment LC_{50}s for these two data points can be estimated using equilibrium partitioning. If one applies the K_{oc} published in the U.S. EPA Region IX (2002) and SARWQCB staff reports (2006) and the water only LC_{50}s reported by McLeese et al. (1982), the estimated sediment LC_{50}s are 9,000 ppb for sand shrimp and 7,100,000 ppb for the polychaete worm.

The remaining 8 data points in Table 33 from Long and Morgan (1990) are based on the presence of chlordane (along with potentially hundreds of other chemicals) in toxic sediments. None of these eight data points provide dose-response information.

The flaws in the ERM data set preclude the use of the ERM value as an indication of the threshold for benthic toxicity due to chlordane in sediments. The threshold appears to be orders of magnitude greater than the ERM. This conclusion is further supported by other bioassay data.

4.2.3 Equilibrium Partition Estimates of Toxicity Thresholds

Let us look further at what is known about toxicity thresholds for chlordane to amphipods and other benthic organisms to gain an understanding of whether the levels of chlordane in sediments in Newport Bay are high enough to cause toxicity to these organisms. Cardwell et al. (1977) studied the chronic toxicity of chlordane in the amphipod, *Hyallela azteca* (Table 31).

Table 31 Growth and survival of *Hyallela azteca* exposed in the water column to chlordane. Table 23 reproduced from Cardwell et al. (1977)
Table 23 Relative survival and growth of Hyallela azteca exposed to technical chlordane

	Measured concentration of technical chlordane, µg/L					
Parameter	Control	1.4	2.6	5.3	11.5	20.5
Replicate I						
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[a]	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

In a 65 day study of mortality and weight gain, the 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 of 42,172 ppb at 1% OC (2.6 µg/L × 1,622,000 L/kg × 0.01 µg OC/µg sediment = 42,172 µg/kg).

The US Fish and Wildlife Service (Eisler 1990) reviewed the aquatic toxicity of chlordane. Eisler reported an LC_{50} of 40 ppb 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 243 ppb chlordane in sediment.

The triad analysis, although representing one kind of weight-of-evidence analysis, is incomplete and flawed as it was used to assess impairment of aquatic biota by chlordane in Newport Bay. Relying on the mere presence of chlordane along with hundreds of other chemicals in toxic sediments constitutes an incomplete weight-of-evidence analysis. The chlordane ERM is not a reliable measure of toxicity thresholds and should not be used in a weight-of-evidence analysis to assess impairment of aquatic biota. Most importantly, one should consider the results of dose-response bioassays. Valid spiked sediment bioassays could not be found for chlordane. Therefore, the triad analysis should have relied on spiked water bioassays and equilibrium partitioning to estimate toxicity thresholds for chlordane in sediments. The available aquatic toxicity bioassay data do not support an effect of chlordane on benthic organisms at the level of approximately 10 ppb (<1–55 ppb) as measured in sediments from Newport Bay. The lowest effect level exceeded 1,000 ppb and the NOEL in the most sensitive species and life stage was 243 ppb.

5 Toxaphene

5.1 Levels in the Environment

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

5.1.1 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. The majority of toxaphene soil samples returned concentrations below detection limits (Table 32). For example, for 2004 data, all 275 soil samples yielded concentrations below the analytical detection limit of 0.1 ppm. Although no statistically clear trends in soil toxaphene concentrations can be demonstrated from a data set in which roughly 90% of samples have toxaphene concentrations below detection limits, and in which samples were not taken at the same locations over time, it appears 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 below.

5.1.2 Sediments

Toxaphene was detected in only one of 42 sediment samples collected from 2005 to 2008.[8] The one sample had a toxaphene residue of 31.7 ppb. The detection limit for all 42 samples was 10 ppb. The most recent analysis of sediments in 2011 (Orange County Watersheds 2013) found no toxaphene above high detection limits that ranged from 70 to 1,900 ppb.

Table 32 Toxaphene concentrations in agricultural soils in the Newport Bay Watershed

Year	0-12 inch Sample Depth			12-24 inch Sample Depth			>24 inch Sample Depth			Detection Limits (ppm)
	Range of Detected Toxaphene (ppm)	Total Samples	Total Non-detect Samples	Range of Detected Toxaphene (ppm)	Total Samples	Total Non-detect Samples	Range of Detected Toxaphene (ppm)	Total Samples	Total Non-detect Samples	
1989	0.500 - 0.940	3	0	0.540 - 0.550	3	1	--	3	3	0.25
1990	0.190 - 0.220	2	0	--	1	1	--	3	3	0.16
1995	--	19	19							0.06
2000	--	28	28							0.20 & 2.00
2002	0.340 - 2.300	174	125				0.210 - 0.300	27	24	--
2004	--	230	230				--	45	45	0.10 - 10.0
2006	--	6	6				--	6	6	0.10

Sources: Unpublished technical reports provided by The Irvine Company (1989–2006)
Data were not available for shaded areas

[8] Personal communication from Amanda Carr at Orange County Watersheds.

5.1.3 Water Column

Ninety water samples collected from 2002 to 2009 have been analyzed for toxaphene (Bay and Greenstein 2003; Orange County Watersheds (see footnote 8)). Only one sample contained detectable toxaphene. The detected level was 5.5 pptr, which was less than the stated detection limit of 10 pptr.

5.1.4 Fish and Mussels

In the case of red shiner, toxaphene tissue concentration data dating from 1983 show a substantial decline (see Figs. 29 and 30). Red shiner may be taken as an indicator of toxaphene concentrations in receiving waters within the watershed, as red shiners are a 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 Fig. 29.

When all red shiner data are considered together, a statistically strong (R^2 value of 0.671) downward trend in toxaphene concentration is evident. The statistical analysis that characterizes these trends was confirmed by splitting the data set for red shiners into two separate sets consisting of the first 10 years of data (1983–1992) and the second 10 years of data (1993–2001). 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

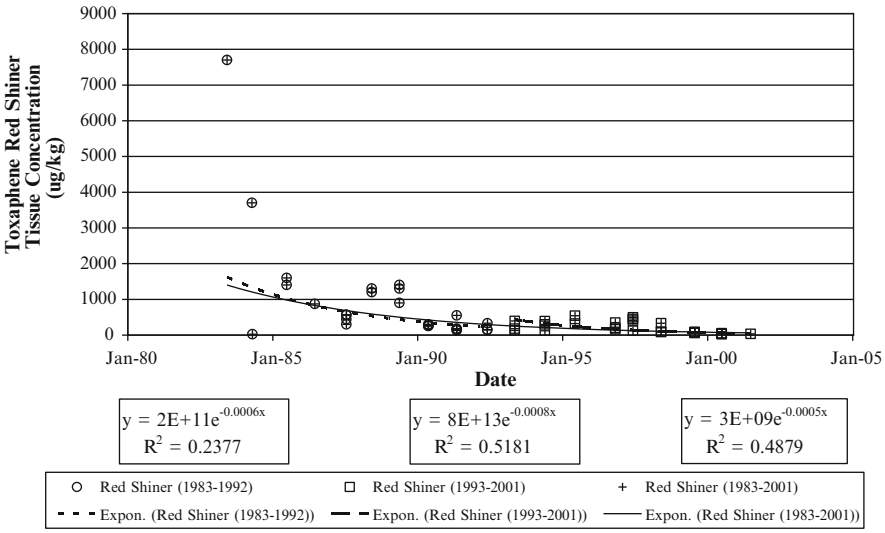

Fig. 29 Toxaphene concentrations in red shiner, San Diego Creek and Peters Canyon Wash (1983–2001). *Source*: California Toxic Substances Monitoring Program (1983–2002). Red shiner data from 2002 to the present are not available

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 data from Newport Bay for the period 1980 through 2000 do not show any statistically significant trends in wet weight toxaphene concentrations over time (Fig. 31). However, toxaphene concentrations in 84 out of the 111 samples (76%) collected over the 21-year period were below analytical detection limits, and the frequency of non-detect results was consistent over time. Nondetect samples were not plotted in Fig. 31.

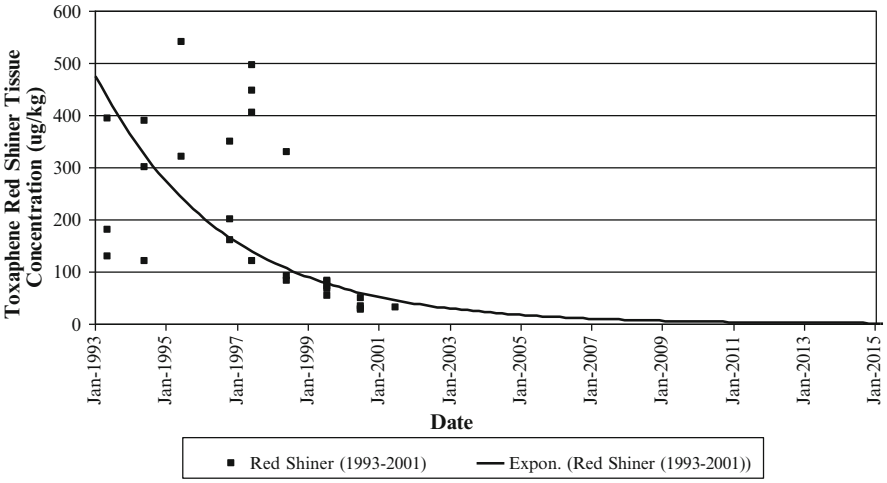

Fig. 30 Toxaphene concentrations in red shiner, San Diego Creek and Peters Canyon Wash, projected through 2015. Data from California Toxic Substances Monitoring Program (1983–2002). Red shiner data from 2002 to the present are not available

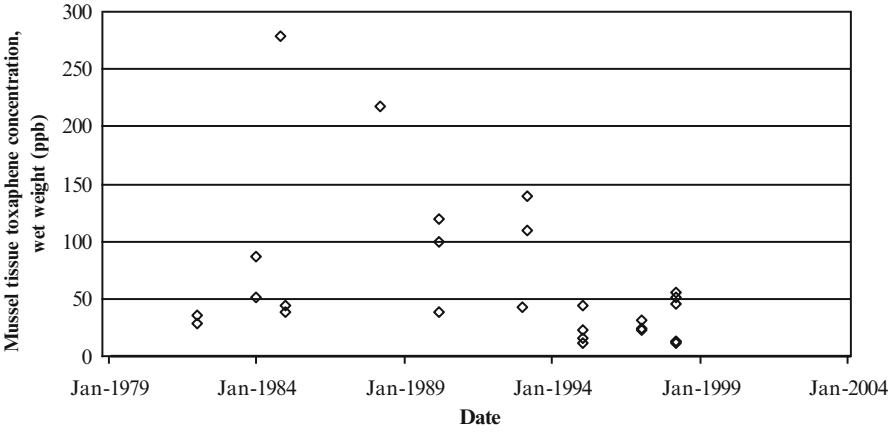

Fig. 31 Toxaphene concentrations in mussels from Newport Bay and Watershed. Data from the California Mussel Watch Program (1980–2000). Only data above detection limits were plotted. The Mussel Watch Program ended in 2000

5.1.5 Factors Affecting Decay of Toxaphene Residues

The U.S. EPA banned the use of Toxaphene in 1990. 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 to 14 years (US EPA 1999). The wide range is attributable to apparently differing degradation rates for toxaphene under aerobic and anaerobic conditions (US DHHS 1996). Under anaerobic conditions the half-life of toxaphene in soil and sediment has been reported to be on the order of weeks to months (Callahan et al. 1979). However, under aerobic soil conditions, Nash and Woolson (1967) reported a half-life of 11 years. Assuming that aerobic conditions are most common in the Newport Bay watershed suggests a half-life on the order of 11 years. At this rate and given that the use of toxaphene was banned in 1990 and excluding other loss mechanisms, the mass of toxaphene in the agricultural soils of Newport Bay and Watershed would have declined by at least 63% over the past 16 years due solely to natural removal. Assuming 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, most of 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.

5.2 NAS Fish Guidance to Protect Wildlife

The SARWQCB (2006) have decided to use the NAS (1972) guidance of 100 ppb toxaphene in fish to protect wildlife consuming fresh water fish in the Watershed. As is apparent from viewing the regression in Fig. 30, the current level in red shiners is well below this guidance. Even so, the SARWQCB has chosen to base impairment on the older fish data that exceed the guidance and to require a sediment target to achieve the fish target of 100 ppb.

5.3 New York State Sediment Guidance to Protect Wildlife

US EPA Region IX (2002) and SARWQCB (2006) staff have decided to use the New York State Department of Conservation (1998) screening level of 0.1 ppb as a TMDL target for toxaphene in sediments. The following is a detailed look at the scientific basis for this guidance.

New York State chose the equilibrium partitioning method for the derivation of a sediment screening level for toxaphene (New York State Department of Environmental Conservation 1998). The calculation began with a New York State water column criterion of 0.005 ppb (μg/L) toxaphene. Multiplication by a K_{oc} for

toxaphene gave the sediment OC concentration. Division by 100 gave the sediment screening level at 1% OC. The K_{oc} was assumed to be equal to a K_{ow} value of 1,995 L/kg (log K_{ow} = 3.3). Hence, the calculation of the screening level is 0.005 µg/L × 1,995 L/kg × 0.01 = 0.1 µg/kg (ppb).

A specific reference for the log K_{ow} of 3.3 could not be found in the New York State Department of Environmental Conservation (1998) guidance document. The K_{ow} used by New York State is 158-fold lower than the K_{ow} cited in the US EPA Region IX (2002) and SARWQCB (2006) reports. Using the EPA and SARWQCB-approved K_{ow} value in the New York State method gives a sediment screening level of 15.8 ppb.

The consequences of using the 0.1 ppb sediment target for toxaphene are major. The SARWQCB (2006) report estimated a Newport Bay Watershed loading capacity of 5.67 g of toxaphene per year and an existing load of 536 g of toxaphene per year. Assuming these loads to be correct, 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 one uses the 158-fold higher log K_{ow} of 5.5 published by both the US EPA Region IX (2002) and the SARWQCB (2006), the New York State sediment screening level would increase 158-fold. The sediment target would then be 15.8 ppb, resulting in a loading capacity of 896 g per year, a value greater than the estimated existing load of 536 g per year, thereby obviating the need for a TMDL.

6 PCBs

The need for a PCB TMDL was stated by the SARWQCB (2006) in their report on organochlorine TMDLs for Newport Bay and Watershed to be due to the level of PCB residues in sport fish in Newport Bay exceeding guidance levels of 20 ppb recommended by California EPA. This section will take a detailed look at the scientific basis for the impairment analysis. The starting point is a review of the PCB residue data in sport fish.

6.1 Levels in Sport Fish

The SARWQCB organochlorine TMDL report (2006) contains the PCB data (Appendix A-1, Table 1) for sport fish that were relied upon by the SARWQCB in their impairment analysis. The underlying data from the State Board is listed below in Table 33.

Of the 63 fish composites from 16 species of sport fish that were analyzed, 18 had measureable residues of PCBs. Two of these were at or below the reporting limit of 5 ppb, leaving 16 composites above the reporting limit. Nine of the fish

Table 33 Total PCB residues in fish fillets from Newport Bay, 1995–2002

Study	Location	Season	Year	Species	Fish per composite	PCBs (ppb)
CFCP	Lower NB	Summer	1999	Diamond turbot	5	5
CFCP	Lower NB	Summer	1999	Shiner surfperch	5	48
CFCP	Lower NB	Summer	1999	Shiner surfperch	10	94
CFCP	Lower NB	Summer	1999	Spotted turbot	5	11
CFCP	Lower NB	Summer	1999	Yellowfin croaker	5	30
TSMP	Lower NB	Summer	1995	Black croaker	2	≤5
TSMP	Upper NB	Summer	2002	Striped mullet	2	172
TSMP	Upper NB	Summer	2002	Spotted sand bass	1	84
TSMP	Upper NB	Summer	1999	Orangemouth corvina	1	21
TSMP	Upper NB	Summer	2000	California halibut	1	18
TSMP	Upper NB	Summer	2001	Round stingray	1	10
TSMP	Upper NB	Summer	1997	Diamond turbot	3	≤5
TSMP	Upper NB	Summer	1998	Brown smoothhound shark	1	≤5
SCCWRP	Lower NB	Winter	00/01	Barred sand bass	3	≤5
SCCWRP	Lower NB	Winter	00/01	Black perch	3	≤5
SCCWRP	Lower NB	Winter	00/01	Black perch	3	≤5
SCCWRP	Lower NB	Winter	00/01	Black perch	4	≤5
SCCWRP	Lower NB	Winter	00/01	California halibut	4	≤5
SCCWRP	Lower NB	Winter	00/01	California halibut	4	≤5
SCCWRP	Lower NB	Winter	00/01	C-O sole	4	≤5
SCCWRP	Lower NB	Winter	00/01	C-O sole	4	≤5
SCCWRP	Lower NB	Winter	00/01	Diamond turbot	6	≤5
SCCWRP	Lower NB	Winter	00/01	Diamond turbot	6	≤5
SCCWRP	Lower NB	Winter	00/01	Fantail sole	5	≤5
SCCWRP	Lower NB	Winter	00/01	Spotted sand bass	3	≤5
SCCWRP	Lower NB	Winter	00/01	Spotted turbot	4	≤5
SCCWRP	Lower NB	Winter	00/01	Spotted turbot	4	≤5
SCCWRP	Lower NB	Winter	00/01	Spotted turbot	4	≤5
SCCWRP	Lower NB	Summer	2001	Kelp bass	5	≤5
SCCWRP	Lower NB	Summer	2001	Yellowfin croaker	4	≤5
SCCWRP	Lower NB	Summer	2001	Yellowfin croaker	4	41.2
SCCWRP	Lower NB	Summer	2001	Yellowfin croaker	4	11.0
SCCWRP	Lower NB	Summer	2001	Yellowfin croaker	4	8.1
SCCWRP	Lower NB	Summer	2001	Yellowfin croaker	4	≤5
SCCWRP	Lower NB	Summer	2001	Black perch	3	≤5
SCCWRP	Lower NB	Summer	2001	Black perch	3	≤5
SCCWRP	Lower NB	Summer	2001	Black perch	3	≤5
SCCWRP	Lower NB	Summer	2001	Barred sand bass	3	≤5
SCCWRP	Lower NB	Summer	2001	Spotted sand bass	3	≤5
SCCWRP	Lower NB	Summer	2001	Spotted sand bass	3	10.4
SCCWRP	Lower NB	Summer	2001	Spotted sand bass	3	24.2

(continued)

Table 33 (continued)

Study	Location	Season	Year	Species	Fish per composite	PCBs (ppb)
SCCWRP	Lower NB	Summer	2001	California corbina	3	≤5
SCCWRP	Lower NB	Summer	2001	California corbina	3	57.8
SCCWRP	Lower NB	Summer	2001	California corbina	3	4.4
SCCWRP	Lower NB	Summer	2001	Diamond turbot	6	≤5
SCCWRP	Lower NB	Summer	2001	Spotfin croaker	6	≤5
SCCWRP	Lower NB	Summer	2001	Spotfin croaker	6	≤5
SCCWRP	Lower NB	Summer	2001	California halibut	4	≤5
SCCWRP	Upper NB	Winter	00/01	Black perch	3	≤5
SCCWRP	Upper NB	Winter	00/01	California halibut	4	≤5
SCCWRP	Upper NB	Winter	00/01	California halibut	4	≤5
SCCWRP	Upper NB	Winter	00/01	California halibut	4	≤5
SCCWRP	Upper NB	Winter	00/01	Diamond turbot	6	≤5
SCCWRP	Upper NB	Winter	00/01	Diamond turbot	6	≤5
SCCWRP	Upper NB	Winter	00/01	Diamond turbot	6	≤5
SCCWRP	Upper NB	Winter	00/01	Shiner perch	8	≤5
SCCWRP	Upper NB	Winter	00/01	Spotted sand bass	3	≤5
SCCWRP	Upper NB	Winter	00/01	Spotted turbot	4	≤5
SCCWRP	Upper NB	Summer	2001	Jacksmelt	6	≤5
SCCWRP	Upper NB	Summer	2001	Jacksmelt	6	9.9
SCCWRP	Upper NB	Summer	2001	Jacksmelt	6	≤5
SCCWRP	Upper NB	Summer	2001	Diamond turbot	6	≤5
SCCWRP	Upper NB	Summer	2001	Diamond turbot	6	≤5

Personal communication from Randall Yates (2006) at the California State Water Resources Control Board. Fish residue data spread sheets from the Coastal Fish Contamination Program

composites exceeded the guidance of 20 ppb. Of these nine fish composites, only two, representing one species, spotted sand bass, were from a species considered to be resident to Newport Bay. The remaining seven composites above 20 ppb were from coastal species, i.e., those that migrate up and down the coast. Coastal species could have received exposures from higher PCB levels outside of Newport Bay. Even the spotted sand bass migrates off shore in the winter, creating the possibility of exposure to PCBs outside of Newport Bay.

6.2 California Sport Fish Guidance to Protect Human Health

The SARWQCB (2006) report states that three resident species from upper Newport Bay displayed residue levels in excess of the guidance, whereas the underlying data cited in Appendix A-1 states that only one resident species, spotted sand bass, from upper Newport Bay, was in excess of the guidance. Review of the cited studies

supports the finding of just one resident species, spotted sand bass, with residues above 20 ppb.

Another important issue is the State Board's TMDL listing policy. The policy requires 6 exceedances for 63 measurements for listing and the initiation of a TMDL (Table 2-3 in SARWQCB 2006). The policy also advises Regional Boards to not consider migratory (coastal) fish because the residues can come from other watersheds. This concept would be particularly true for Newport Bay because of coastal migration of fish from the very highly contaminated Palos Verdes Shelf, only 30 miles to the north. With only two composites from one species of resident fish in excess of the guidance, the listing criteria is not met, meaning that a TMDL for PCBs would not be recommended by the State Board for Newport Bay.

More important than meeting the State Board's listing policy for a TMDL, the health risks of PCB from ingestion of resident sport fish caught in Newport Bay can be shown to be insignificant. The average residue in six composites of spotted sand bass was 21 ppb. This average assumes PCB concentrations at one-half the detection limit of 5 ppb in composites where PCBs were not detected. All other residential sport fish species were below 20 ppb, with most being below the detection limit of 5 ppb. Since sport fishermen eat several different species of resident fish, the average residue of PCBs ingested will be well below the guidance of 20 ppb. The average PCB residue in all 63 resident and coastal sport fish composites is 12 ppb, a value below the guidance. In addition, the fish residue data are 11 years old or older, meaning that with no new sources of PCB input and further decay of residues, exposures are even lower today and will continue to decrease in the future.

7 Summary

DDT, chlordane, toxaphene and the PCBs are persistent organochlorines that are still found in aquatic environments of Newport Bay and Watershed (Orange County, California), decades after their use was discontinued. Under the Clean Water Act, organochlorines are regulated by a total maximum daily load (TMDL) to achieve levels that protect wildlife and human health. Stakeholders in the Newport Bay Watershed and an Independent Advisory Panel (IAP) requested by the Regional Board and administered by Orange County have questioned the quality of the science used to establish TMDL targets by US EPA Region IX and the Santa Ana Regional Water Quality Control Board. This review brings together a number of technical reports written by stakeholder consultants that address the scientific basis for the organochlorine TMDLs for Newport Bay and Watershed.

Urbanization of former agricultural lands has effectively capped soil organochlorine residues, reducing runoff into aquatic environments. Sediment controls have further reduced the movement of organochlorines from soil to aquatic environments. Residues in soil, water, sediments and biota are declining. For example, DDT in red shiner and mussels from Newport Bay and Watershed is declining exponentially with a half-life of 3.8 years and 5.2 years, respectively.

Sediment TMDL targets for total DDT, based on threshold effects levels (TELs), are inappropriate due to outdated, inaccurate and misinterpreted data. The TEL method ignores important dose-response relationships in the data sets used to calculate TELs. TELs for total DDT greatly overestimate thresholds for sediment toxicity, when compared to dose-response studies. The problem with using TELs as TMDL sediment targets is that risks are over-estimated, resulting in the assignment of resources disproportionate to risk and thereby not minimizing overall risk to humans and wildlife.

Terns, cormorants and several other avian species found in Newport Bay and Watershed are less sensitive to the reproductive effects of DDTs than ospreys and brown pelicans. Residues of DDE in eggs in excess of 10 ppm, resulting in eggshell thinning of 15% or more, appear to be necessary to produce significant hatching failure. The lack of a correlation between DDE levels in Forster's tern eggs and eggshell thickness indicates that reproduction in the closely related and threatened least tern probably will not be affected by DDT levels that currently exist in Newport Bay. The IAP has recommended the least tern as an indicator species for potential toxicity of DDT to wildlife in Newport Bay and Watershed.

The rare appearance of marine mammals in Newport Bay is unlikely to result in significant exposures to organochlorines. Worldwide, studies of marine mammals have disclosed a wide range of body burdens of DDT, but few if any impacts have been clearly delineated. With wildlife tissue levels clearly on the decline, impacts that might be identified in the future are unlikely.

Fish tissue targets to protect wildlife were adopted from the 1972 National Academy of Sciences recommendations. The 50 ppb target for DDT in marine fish is protective, but very extensive study since 1972 indicates that 150 ppb is also protective of sensitive avian species like the osprey. Successful osprey breeding began in Newport Bay in 2006 and has continued through 2013. The 150 ppb level in marine fish is also the basis for the national marine water criterion, a level that would be expected to protect the brown pelican. The 1,000 ppb target for DDT in fresh water fish is not protective and should be lowered to 150 ppb.

Two additional fish guidance reports were considered, but were not used as TMDL targets to protect wildlife. The fish guidance of 14 ppb by Environment Canada is highly protective from the assumption that minimal eggshell thinning is toxic and from having used an insensitive species to assess worst case ingestion rates. The fish guidance of 50 ppb by US EPA Region IX (Biological Technical Assistance Group, BTAG) relied on the same study in brown pelicans that is the basis for the national criterion for DDT in water (150 ppb in the fish diet). The BTAG guidance is threefold lower by having assumed a rapid equilibration of DDTs between dietary fish and brown pelican eggs, even though the underlying data indicates that equilibration may take several years.

The triad analysis, although one kind of weight-of-evidence analysis, is incomplete and flawed, when it was used to assess impairment of aquatic biota by chlordane in Newport Bay. Relying on the mere presence of chlordane, along with hundreds of other chemicals in toxic sediments, constitutes an incomplete weight-of-evidence analysis. The chlordane ERM is not a reliable measure of toxicity

thresholds and should not be used in a weight-of-evidence analysis to assess impairment of aquatic biota. Most important, one should consider the results of dose-response bioassays.

The New York State sediment guidance for toxaphene was promulgated as a TMDL freshwater target for the Watershed. The guidance relies on an unreferenced K_{ow} that is 158-fold greater than the K_{ow} recommended by the regulatory agencies promulgating the TMDL target. If the TMDL target is calculated with the recommended K_{ow}, the existing load of toxaphene in the Watershed is below the TMDL target.

TMDL fish targets for DDT and PCBs to protect sport fishermen in Newport Bay are based on California EPA guidance that was intended only as a risk tool and not as a TMDL standard. Even so, average residues of DDT and PCBs in sport fish collected in 2000 and 2001 from Newport Bay were below the guidance. More recent California EPA guidance weighs cancer and noncancer risks against the benefits of consuming fish. That guidance is not to exceed 520 ppb DDT in sport fish consumed at the rate of three 8 oz filets per week. Similar guidance for PCBs is 21 ppb. California EPA has not issued a sport fish advisory for Newport Bay.

Residues of organochlorines in fish have declined to levels that pose no known hazard to humans or wildlife. The margin of safety will continue to increase as residues in fish continue to decline.

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