Exponent®

Special Publication SJ2004-SP1

Analysis of Avian Mortality at the North Shore Restoration Area of Lake Apopka in 1998–1999

Prepared for

St. Johns River Water Management District
Palatka, Florida

Exponent®

Analysis of Avian Mortality at the North Shore Restoration Area of Lake Apopka in 1998–1999

Prepared for

St. Johns River Water Management District
4049 Reid Street
Palatka, FL 32177

Prepared by

Exponent
15375 SE 30th Place, Suite 250
Bellevue, WA 98007

August 2003

Doc. no. 8601529.001 0701 0403 NG04

Contents

List of Figures

List of Tables

Acronyms and Abbreviations

BSAF	biota-sediment accumulation factor
FWS	U.S. Fish and Wildlife Service
GABA	gamma-aminobutyric acid
GIS	geographic information system
LOAEL	lowest-observed-adverse-effect level
NDV	Newcastle disease virus
NOAEL	no-observed-adverse-effect level
NSRA	North Shore Restoration Area
SJRWMD	St. Johns River Water Management District
TOC	total organic carbon
TRV	toxicity reference value
USGS	U.S. Geological Survey

1 Introduction

Between mid-December 1998 and early March 1999, an outbreak of avian mortality occurred at the North Shore Restoration Area (NSRA) of Lake Apopka in central Florida. Dead birds of numerous species were recorded, although in terms of total numbers, the American white pelican (*Pelecanus erythrorhynchos*) was the most affected species. More than 400 mortalities were recorded for this species at the NSRA, and similarly high mortality was seen at offsite locations.

The NSRA lies on former agricultural land that was used for the production of vegetables and market crops. As a result of past agricultural pest control practices, soils of the NSRA contain elevated residues of organochlorine pesticides, including toxaphene, DDT and its metabolites, and dieldrin. When the agricultural lands were acquired by the St. Johns River Water Management District (SJRWMD) post-harvest in 1998, they remained flooded. The shallow-water habitat thus created was attractive to resident and migratory birds, and resulted in a huge influx of many species of birds to the NSRA. When the mortality outbreak began, it was believed by wildlife agencies that the elevated organochlorine pesticide levels in flooded soils, particularly toxaphene levels, likely caused the mortality.

Exponent was retained by SJRWMD to investigate the mortality event, and specifically to:

- Analyze the underlying causes of the bird mortality at the NSRA
- Determine the levels of pesticides in muck soils that are protective of birds, if pesticides are the cause of mortality
- Determine the most effective management options for preventing such mortality events from occurring in the future.

This technical report addresses the first objective and summarizes our review of key components of the mortality event to analyze the evidence that supports or refutes organochlorine pesticides, specifically toxaphene, as the causal agent of the mortality. As no single approach or measurement endpoint is definitive for assigning causality, this report uses a weight-of-evidence approach that relies on several different components to evaluate causality. The components that are evaluated include:

- Avian population census and mortality data
- Food-web modeling to evaluate exposure of avian species to organochlorine pesticides
- Body residues of organochlorine pesticides in dead birds
- Etiology and pathology of the mortality event.

Each of these components is addressed separately in the following sections, and results are summarized in the *Conclusions* section, where an epidemiological approach is used to evaluate causality.

2 Avian Census and Mortality Data

2.1 Analysis of Census Data

Bird counts were conducted by SJRWMD staff at the NSRA of Lake Apopka (Units 1 and 2 and the Sand Farm) between August 15, 1998, and August 13, 1999. During that period, 89 censuses were performed at frequencies of 1–3 times per week. Data were compiled as the total number of individuals of each species that were observed, and were not subdivided by unit or flooding block.

Exponent condensed the count data provided by SJRWMD to assist in comparing species counts with mortality patterns. Data were grouped into 2-week intervals, which were arbitrarily selected to conclude on a Sunday. If multiple censuses were performed within any 2-week period, then an average count for that period was calculated for each species based on estimates from all censuses. Data from all census periods were evaluated, but this report focuses only on censuses conducted between August 15, 1998, and the week ending on April 11, 1999, because:

- Bird mortalities were not recorded after the middle of April
- The combination of habitat changes (drying of fields), departure of migratory species, and bird-hazing practices had resulted in greatly reduced species abundances by that time.

Count data are summarized by time interval in Table 2-1.

2.2 Analysis of Mortality Data

Avian mortality data were provided to Exponent in three formats. Mortalities occurring between December 12, 1998, and January 9, 1999, were summarized by Dr. Gianfranco Basili of SJRWMD in a table that listed the total number of dead birds by species and date, and provided comments describing the locations (flooding blocks) where these birds were collected. From January 10, 1999, onward, SJRWMD staff recorded all bird mortalities on a standardized report form that listed species, number of dead and sick birds, and in many cases, the age of birds (adult or juvenile). Locations where birds were collected were denoted on a map inset on the collection form. Additionally, the U.S. Fish and Wildlife Service (FWS) compiled mortality information from late February through early April 1999, and provided that information to SJRWMD in a summary spreadsheet in 2002.

Exponent tallied all dead and sick birds listed in the various data sources and classified the time of occurrence according to the same time intervals as used to group census data. The total numbers of dead and sick individuals recorded for each species are presented in Table 2-2. The total number of birds reported in Table 2-2 differs from numbers reported in earlier reports, such as the chronology in the Report to Governor Bush (SJRWMD 1999a) for several reasons. First,

the mortality totals in the Governor's report did not include the FWS data reported here. Second, the data reported in Table 2-2 include dead and sick birds whereas the Governor's report listed only dead birds. There may be some double-counting of birds in the summary presented in this report as a result of the partial overlap in the time period when both SJRWMD and FWS personnel were recording mortalities and when both groups may have observed and recorded the same birds. However, from the mortality information available, it is not possible to determine if such double-counting occurred, and it is assumed that all mortalities reported here represent unique events.

2.3 Species Abundance Trends

The four species with the highest reported mortality were white pelican, wood stork (*Mycteria americana*), great blue heron (*Ardea herodias*), and great egret (*Casmerodius albus*). Population trends for these species are shown in Figure 2-1. As illustrated by the graph, wood storks and great egrets were most abundant in mid- to late November, well before the peak abundance of white pelicans, which occurred in late January. Wood storks and great egrets are resident species in the area, and early abundance peaks for these species may reflect local movements and aggregations of birds that were attracted to the flooded fields of the NSRA. White pelicans are migratory, and the more pronounced peak in abundance for this species may represent the arrival of migrants. Great blue herons, a resident species, did not display any population peak, and numbers tended to be constant throughout the period of mortality.

Summary population graphs for those species where mortality was not observed, or for which fewer individuals were found dead, are included in Figures 2-2 through 2-4. As shown on the graphs, many of these species were as abundant, or more abundant, than the species primarily involved in the mortality event, and they were present on the NSRA at the same time. Wading bird species (Figure 2-2) showed relatively minor fluctuations in population size, except for the cattle egret (*Bubulcus ibis*). This species prefers open dry areas for foraging more than the other wading bird species, and fluctuating population sizes for this species likely reflect a response to changing water levels in the flooding blocks. Although not shown on the graph, the population of this species on the NSRA increased to about 2,500 individuals in July 1999, when all flooding blocks were dry.

Most shorebirds also maintained fairly consistent population sizes (Figure 2-3), although their different periods of peak abundance likely reflect different arrival and departure times for migratory species (e.g., short-billed and long-billed dowitchers, *Limnodromus griseus* and *L. scolopaceus*, Figure 2-3). Killdeer (*Charadrius vociferous*) numbers increased later in the study period. This species also prefers open, dry fields for foraging, which may explain its higher abundance later on when fields were drying out. Some waterfowl species, such as ring-necked duck (*Aythya collaris*), blue-winged teal (*Anas discors*), and green-winged teal (*Anas crecca*) were extremely abundant (Figure 2-4), but had a well-defined peak of abundance, which likely reflects the arrival and departure times of these migratory species.

2.4 Comparison of Population and Mortality Trends

Species with the highest number of recorded mortalities (white pelican, wood stork, great egret, and great blue heron) were examined for relations between abundance and mortality. Because of the large variations in population sizes among these species, mortalities are not expressed on an absolute basis, but rather as a percentage of the maximum population recorded during any census count. This method is subject to uncertainty because the census counts may not enumerate all birds present at the NSRA, and thus mortality rates could be overestimated. Alternatively, if not all bird mortalities were recorded, the rate may be an underestimate.

As a percentage of their maximum estimated population, white pelicans and great blue herons exhibited the highest mortality rate, at about 16 percent of the population; however, the patterns of mortality differ between the two species. For white pelicans, there appear to be two peaks of mortality, one occurring in late December, and a lag of two months before another peak in mid-February. The initial incidence of mortality occurred at a time when the census count for this species was low relative to later periods. However, in the previous two-week period the average census count was 1005 individuals (Table 2-1), and variation in census counts may reflect local movement of white pelicans from the NSRA to other regional locations. At the time of this initial mortality event, there is no evidence that increased mortality was observed for other species. For great blue herons, the peak mortality did not occur until mid-March, when census counts indicate that heron numbers were declining and when pelican mortality was declining. For wood stork and great egret, mortality rates, expressed as a percentage of the maximum population, were about 5–6 percent, and mortality occurred at about the same time as great blue heron mortality was observed.

2.5 Conclusions

Analysis of the population and mortality data indicates that:

- Mortality was not observed for the majority of wading bird, shorebird, and waterfowl species present at the NSRA, including many with high abundance. The intensive carcass searching and recovery efforts that were conducted during the mortality event suggest that at least several individuals of these species would have been located if pronounced mortality had occurred.
- Four species (white pelican, wood stork, great egret, and great blue heron) experienced elevated mortality, up to 16 percent of the total population may have been affected for white pelican and great blue heron, and about 5–6 percent of the population for wood stork and great egret.
- The patterns of mortality differed for pelicans and great blue herons, with an early mortality event occurring in the former species, but mortality only occurring late in the season for the latter species, at a time of decreasing population abundance.

3 Evaluation of Organochlorine Brain Residue Levels

Several researchers have examined residue levels of organochlorine compounds in avian brain tissue to determine threshold concentrations that are diagnostic of mortality. These threshold concentrations can be used for comparison with levels measured in the brains of birds collected at the NSRA. It must be emphasized, however, that the distribution of the collected samples examined in this section represents birds that died and is hence not representative of the distribution of concentrations for birds resident to the NSRA. Therefore, the proportional distributions represented here cannot be applied to the probability of given organochlorine concentrations among all the birds. Rather, it is the distribution of organochlorine concentrations in birds that died in the NSRA regardless of attributable cause of death.

There are analytical results for 25 birds collected March and April 1999. Tissues from 15 of those birds were analyzed at the USGS Patuxent laboratory, 1 at EPA's Athens laboratory, and 9 at En Chem, Inc. in Wisconsin. The 15 Patuxent analyses were performed in two different laboratory runs, with 9 analyses reported in Catalog #4030049 and 6 reported in Catalog #4030063.

Determination of a critical threshold for DDT is complicated because residues in the brains of experimental and wild animals killed by DDT are a variable mixture of DDT and the breakdown products DDD and DDE. To standardize the concentrations of these three compounds, Stickel et al. (1970) developed the concept of DDT-equivalent (DDTr) concentrations, based on analysis of lethal levels in the brains of birds exposed to the compounds individually. Stickel et al. (1970) determined that 1 mg/kg DDT is equivalent to 5 mg/kg DDD or 15 mg/kg DDE. In a review of published studies, Blus (1996) determined that the DDT equivalents in brains of birds dying from DDT poisoning were ≥18 mg/kg, and that most birds that die from DDT have equivalents >20 mg/kg. Therefore, 20 mg/kg DDT equivalents is considered a lower lethal threshold for DDT poisoning. The log-normal distribution of brain tissue residue data for birds that died at the NSRA is presented in Figure 3-1 as DDT equivalents (DDTr) and is compared with the diagnostic threshold. None of the American white pelicans had brain residue concentrations exceeding 20 mg/kg although the projected rate was predicted to be 2.36 percent. Among all avian species, 6.80 percent exceeded the diagnostic threshold. This strongly suggests that DDT concentrations were not sufficiently elevated to represent a significant cause of the avian mortalities.

The diagnostic concentration for dieldrin in brain tissue was based on the protocol developed for the Rocky Mountain Arsenal, based on the work of Stickel et al. (1969). An upper threshold of 9 ppm was considered to be lethal even in the absence of clinical signs. A lower diagnostic limit of 5 ppm was also used because it is considered to be the limit for dieldrin-induced mortality when associated with supporting clinical signs. The distribution of dieldrin concentrations in the brains of birds that died at the NSRA is illustrated in Figure 3-2. For all species, 13.8 percent of the collected birds exceeded the upper threshold, while 21.1 percent exceeded the lower threshold (Figure 3-2).

The brain dieldrin level of the EPA bird was 1.6 mg/kg. For the other 24 birds, brain levels of dieldrin exceeded the 9 mg/kg threshold 9 times. Two of the above-threshold values were coded "U" (En Chem). One bird (SJRWMD #48) analyzed by En Chem exceeded the 9 mg/kg limit at 10 mg/kg. The remaining six high values came from data provided by the Patuxent lab, and all were from a single lab run, Catalog #4030049. The brain dieldrin levels reported in that lab run were generally so high (10, 11, 36, 34, 24, 20 mg/kg) that closer inspection of other data from that catalog number seems warranted.

The temporal distribution of dieldrin brain concentrations showed that most of the birds that exceeded either the upper or lower thresholds died in March and April 1999: more than 3 months after the NSRA had been drained to historic levels (Figure 3-3). For the collected American white pelicans, one bird (1.8 percent of the pelicans) exceeded the upper diagnostic threshold for dieldrin (9.5 mg/kg; SJRWMD #58; collected January 30, 1999) while 8.8 percent exceeded the lower threshold. Unlike the other avian species tested, observations exceeding the lower threshold were only observed in January and February of 1999 (Figure 3-4). However, the low rate of occurrence suggests that dieldrin exposure was an insignificant factor for the white pelican.

Although it is possible that brain levels of dieldrin might have increased in March and April to the extent reported in the Patuxent data, presumably other physical characteristics, such as percent lipid and brain weight, would remain fairly constant. From the mortality data set, the average of 50 values of percent brain lipid measured by three different laboratories (excluding Patuxent lab) was 8 (std dev 1.7). An average of 11 brain lipid measurements analyzed by the Patuxent lab from two different lab runs (catalog #s 4030045 and 4030037) was 6 percent (std dev 1.9). In contrast, 12 brain lipid results reported under Catalog #4030049 averaged 30 percent (std dev 12)—five-fold higher than their own data, and a four-fold difference from the other labs. The brain weights of the birds analyzed under Patuxent laboratory's Catalog #4030049 were also exceedingly high, compared both to brain weights recorded under their other catalog numbers, and to brain weights measured by other laboratories. For instance, of four great egrets analyzed, brain weights of two of them reported in Patuxent Catalog # 4030049 were 83.42 g and 83.48 g. Those two weights are about 20 times greater than the 4.62 g great egret brain weight reported in Patuxent Catalog #4030045 and the 3.9 g great egret brain weight reported by Dr. Spalding, University of Florida.

A diagnostic threshold for toxaphene concentrations in bird brain tissue was not located in the literature for comparison with levels reported in birds from the NSRA. Only a few studies have examined toxaphene levels in avian brains, and the reported concentrations are much lower than levels measured in birds from the NSRA. Haseltine et al. (1980) conducted a dosing study using black ducks (*Anas rubripes*) fed toxaphene in the diet at 0, 10, or 50 ppm for 19 months. Even at that prolonged exposure, toxaphene was undetected in brains of birds fed the lower dose level (detection limit was 0.1 mg/kg), and ranged between 0.68 and 1.4 mg/kg in birds on the higher dose level. Keith (1966) did not detect toxaphene residues in brains of white pelicans and western grebes (*Aechmophorus occidentalis*) found dead on Tule Lake in California. The birds were part of a mortality event that occurred in marsh lands adjacent to farmlands that were treated regularly with toxaphene and DDT. Niethammer et al. (1984) reported undetectable levels of toxaphene in brains of yellow-crowned night herons (*Nycticorax violacea*) and residues of 0.54 mg/kg in green heron (*Butroides striatus*) brains for birds collected in

northeastern Louisiana. In the absence of any suitable avian data, and for the purpose of this analysis only, given its inherent uncertainties, a threshold of 4 mg/kg was used based on the analysis of brain concentrations in pigs lethally poisoned with toxaphene (Mount et al. 1980).

Toxaphene concentrations in bird brains from birds collected in the NSRA were typically higher than other organochlorine pesticides, and exhibited much greater variability among individuals. Toxaphene levels in avian brains ranged from 0.05 to 288 mg/kg with 81.3 percent of analyses from all species exceeding the 4 mg/kg threshold and 80.2 percent of the white pelicans exceeding the threshold (Figure 3-5). The temporal distributions of toxaphene concentrations (Figures 3-6 and 3-7) were similar to those seen with dieldrin (see Figures 3-3 and 3-4). Across all avian species, the highest brain concentrations for toxaphene were seen in March and April of 1999 while the highest toxaphene concentrations in the white pelicans were observed in January and February 1999, and declined from there (Figure 3-7).

It appears on the surface that toxaphene may have played a major role in the avian mortality observed in the NSRA. The dead or dying birds removed from the NSRA possessed toxaphene concentrations in their brains that were one to two orders of magnitude higher than any reported in the scientific literature. Independent analysis undertaken by Indiana University and performed on a selection of six avian liver samples and one brain tissue sample confirmed the toxaphene concentrations reported by En Chem (Hites 2001).

However, although the sampled birds did appear to have unprecedented toxaphene concentrations in their brain tissues, it is impossible to be conclusive as to the physiological implication of such observations because of the lack of an adequate toxicological benchmark. Toxaphene is not a single compound, but rather a mixture of like-congeners of polychlorinated borane. If the mammalian acute threshold benchmark of 4 mg/kg developed for fresh technical toxaphene is applicable to both avian receptors and to the type of weathered toxaphene measured in the birds, then in theory, these birds should never have had such high brain concentrations because they would have died at lower toxaphene brain concentrations. The reality that they had survived to accumulate such levels suggests that the assumptions associated with the toxicity of toxaphene may not be valid. If the benchmark of 4 mg/kg is not representative of an acute tissue burden for the type of toxaphene that the birds had accumulated, and assuming the birds did die of toxaphene toxicosis, then the observations suggest that the threshold value must be higher than 4 mg/kg, perhaps around the median measured concentration of 33 mg/kg in brain tissue. Unfortunately, the current understanding of toxaphene toxicity is not sufficient to provide an estimate of avian toxicity on a congener-specific basis.

This reasoning should be considered highly speculative because, aside from the high measured toxaphene concentrations in the bird's brain tissue, there is no evidence to indicate that the birds died of acute toxaphene poisoning. Because a definitive benchmark cannot be defined at this time, the high toxaphene concentrations in the sampled birds, while noteworthy, cannot be considered as indicative of the cause of the avian mortality observed in the NSRA.

Comparison of measured concentrations of DDT and its metabolites in bird brains with acute toxicity thresholds reported in the literature showed that levels in birds that died at the NSRA were too low to for these compounds to be a likely cause of mortality. The incidence of dieldrin

concentrations exceeding the threshold was predicted to be 14–21 percent in all species, but only 2–9 percent in American white pelicans. This suggests that dieldrin may have affected the pelican populations, but at an average rate of less than 10 percent of total mortalities. If the birds died of organochlorine poisoning, the most likely cause was exposure to toxaphene because of the high measured residue concentrations relative to past surveys from other locations. However, the assumption of organochlorine poisoning is circumstantial and because the residue levels observed could not be compared with a realistic diagnostic threshold, it is not possible at this time to conclude that the high toxaphene concentrations observed in the dead birds found around the NSRA are indicative of toxaphene poisoning.

4 Food-Web Models for Organochlorine Pesticide Exposure

To determine whether pesticides may have been a causal factor for the avian mortality recorded in 1998–1999, potential organochlorine pesticide exposure was modeled using probabilistic methods for seven bird species: American coot (*Fulica americana*), American white pelican, double-crested cormorant, great egret, great blue heron, least sandpiper (*Calidris minutilla*), and wood stork. These species were examined because a) they were present and abundant in the NSRA during the period in question, b) they were species with differing degrees of mortality, from none to high, and c) they use different foraging behaviors (e.g., benthic feeder or piscivore) and have different diets, and therefore, would have been exposed to organochlorine pesticides at different rates or through different exposure routes. The objectives of this analysis were to use available data on pesticide concentrations in fish and soil/sediments to determine whether the white pelican had greater exposure to pesticides than other bird species, and whether this difference could explain the variable mortality rates among species on the NSRA.

4.1 Ecology of Modeled Avian Receptors

The ecology of the bird species chosen for food-web exposure modeling is reviewed in this section. The ecological parameters used to parameterize the food-web model simulations are presented in the context of species' life histories. A summary of the respective parameters is provided in Table 4-1.

4.1.1 American Coot

The American coot is a diving and dabbling rail that weighs an average of 0.56 and 0.72 kg for females and males, respectively. The average of female and male body weight, 0.64 kg, was used as the body weight in the exposure model. The American coot can be a year-round resident in Florida and prefers foraging in marshes and vegetated ponds (Udvardy 1988). The American coot is mainly an herbivore, but will occasionally eat invertebrates (Bogiatto 1990). Pondweeds, watermilfoil, algae, and submerged aquatic plants are their preferred food items (Allen 1985). Because the accumulation rate of organic pesticides in plants is very low, it was conservatively assumed in the exposure model that the American coot consumes only invertebrates, which accumulate organic pesticides to a greater extent than do plants (McEwen and Stephenson 1979). The ingestion rate for the American coot was estimated using the allometric scaling equation developed by Nagy (1987) for estimating food mass intake based on body weight:

$$\text{Intake Rate}\left(\frac{\text{g Dry Weight}}{\text{Day}}\right) = 0.648 \times \text{W}^{0.651}$$

where W is the body weight in grams.

Using the average body weight of 0.64 kg, a prey ingestion rate of 69 g (dry matter) per kilogram of body weight per day is estimated and applied in the exposure model.

4.1.2 Double-Crested Cormorant

The double-crested cormorant is a diving bird that weighs an average of 1.54 kg for females, and 1.81 kg for males (Dunning 1993). The average body weight for both sexes (as applied in the exposure model) is 1.68 kg, based on observations reported by Dunning (1993). The double-crested cormorant breeds in Florida and prefers to forage in large, deep water bodies (Udvardy 1988; Robbins et al. 1983). Preferred forage fish range between 2.6 and 27.5 cm long and were assumed to constitute the cormorant's entire diet (Campo et al. 1993). A maximum fish length of 28 cm was applied in the exposure model. The prey ingestion rate assumed for the double-crested cormorant was 48 g (dry weight) per kilogram of body weight per day, based on observations of Campo et al. (1993).

4.1.3 Great Blue Heron

The great blue heron is a large wading bird that weighs 2 to 2.5 kg (Butler 1992; Dunning 1993). Butler (1992) reviewed numerous studies that measured body weights of great blue heron adults and reported that the average combined-sex body weight—based on studies that sampled more than one individual—is 2.26 kg. Based on Butler (1992), a body weight of 2.3 kg was assumed for the great blue heron in the exposure model. The great blue heron is a year-round resident of Florida and is very adaptable to various habitats, although it prefers vegetated wetland habitats (Butler 1992; Udvardy 1988). The great blue heron primarily eats fish but will also consume amphibians, invertebrates, reptiles, and mammals (Kushlan 1978; Palmer 1962). Because fish are the primary food item in its diet, and no estimates of residue concentrations are available for other prey sources specific to the NSRA, it was assumed for the exposure model that the great blue heron's diet is composed entirely of fish. The prey ingestion rate of the great blue heron was estimated using the allometric equation developed by Kushlan (1978) for wading birds:

$$\text{Intake Rate}\left(\frac{\text{g Wet Weight}}{\text{Day}}\right) = 10^{0.966 \log W - 0.64}$$

where W is the body weight in grams.

The food ingestion rate for application in the exposure model that is estimated using the above formula, and a wet weight-dry weight conversion factor that assumes fish are 75 percent water (U.S. EPA 1993), is 44 g (dry matter) per kilogram body weight per day.

4.1.4 Great Egret

The great egret is a wading bird that ranges in size from 0.7 to 1.5 kg (del Hoyo et al. 1992). The average body weight of adult great egrets was reported by Palmer (1962) to be 0.92 kg. Based on Palmer's study and his own unpublished data, Smith (1997) modeled great egret

populations on Lake Okeechobee, Florida using a body weight of 1 kg, and this was the body weight assumed for the exposure model.

Like the great blue heron, the great egret is a permanent resident of Florida. Its preferred foraging habitats are the shallow waters of streams, ponds, marshes, and mudflats (Robbins et al. 1983). Great egrets feed primarily on fish but will also consume invertebrates, amphibians, reptiles, and small mammals (Hoffman 1978). Frederick et al. (1999) reported that the diet of great egret nestlings was approximately 95 percent fish, based on field observations made in the Florida Everglades over a 4-year period. For the purposes of the exposure model, it was assumed that the great egret consumes a diet composed entirely of fish. Willard (1977) reported the maximum prey length consumed by the great egret is 37 cm, and this maximum prey length was used in parameterizing the exposure model. The food ingestion rate of the great egret was estimated using the allometric equation developed by Kushlan (1978). The prey ingestion rate, based on an average body mass of 1 kg, was 45 g (dry matter) per kilogram body weight per day.

4.1.5 Least Sandpiper

The least sandpiper is a small, migratory shorebird that winters in Florida (Udvardy 1988). Cooper (1994) reported body weights for adult least sandpipers ranging from 16.0 to 33.2 g, depending on location and time of year. In North Carolina, birds captured during the fall migration period weighed between 19.0 and 33.2 g, with an average weight of 25.9 g (Post and Browne 1976, as cited in Cooper 1994). Because avian mortality at the NSRA occurred in the late fall and early winter, the migratory average weight (25.9 g) was determined to be appropriate for application in the exposure model. The least sandpiper winters in Florida and prefers to forage on mudflats and around grassy pools. In winter, it is often found foraging in flooded fields (Udvardy 1988). The majority of its diet is composed of invertebrates (Cooper 1994). The prey ingestion rate for the least sandpiper is estimated using the allometric equation specific for non-passerine birds (Nagy 1987):

$$\text{Intake Rate}\left(\frac{\text{g Dry Weight}}{\text{Day}}\right) = 0.301 \times \text{W}^{0.751}$$

where W is the body weight in grams.

The estimated food ingestion rate of the least sandpiper, assuming a body weight of 25.9 g, was 134 g (dry matter) per kilogram of body weight per day.

4.1.6 American White Pelican

The American white pelican is a relatively large bird that ranges from 4.5 to 13.6 kg (Behle 1958); however, typical body masses range between 5.4 and 9.0 kg (Evans and Knopf 1993). Because the range of body weights is wide and males are heavier than females (Evans and Knopf 1993), the more conservative average female body weight of 5.4 kg, as reported by Evans and Knopf (1993), was applied in the exposure model. The American white pelican is a migratory species that winters in Florida. Unlike the brown pelican (*Pelecanus occidentalis*),

the American white pelican rarely dives while hunting. Rather, it prefers to hunt while swimming on the surface and capturing prey by darting its head and neck underwater to scoop up shallow swimming fish. The American white pelican's preferred foraging habitats are open but shallow water bodies where fish are plentiful (Udvardy 1988). Its diet is composed almost entirely of fish that range from <1 to 40 cm long (McMahon and Evans 1992; Derby and Lovvorn 1997). Evans and Knopf (1993) reported a daily food ingestion rate of 1.8 kg (wet weight basis). This was considered to be a more accurate estimate of pelican intake rates because a) it is based on measured observations for the species of consideration, and b) the body mass of the white pelican places it at the very high end of the range in the regression model proposed by Nagy and therefore is highly uncertain. Using a body weight of 5.4 kg and assuming a food moisture content of 75 percent, the dietary intake rate used to characterize the white pelican was determined to be 83.3 g (dry matter) per kilogram of body weight per day.

4.1.7 Wood Stork

The wood stork is one of the largest wading bird species, weighing from 2 to 3 kg (Palmer 1962). The average body weight for female and male adult wood storks was reported by Dunning (1993) to be 2.1 and 2.7 kg, respectively. For the exposure model, an average combined body mass of females and males (2.4 kg) was assumed. The wood stork resides year-round in Florida and prefers foraging in marsh and swamp habitat, particularly in wooded habitats such as mangrove swamps (Udvardy 1988; Coulter et al. 1999). Wood storks have been reported to consume amphibians, reptiles, insects and plants, but their primary food item is fish (Coulter et al. 1999). The fish typically consumed by the wood stork measure between 2.5 and 25 cm long (Kahl 1964; Ogden et al. 1976). Therefore, the maximum prey length applied in the exposure model was 25 cm. The food ingestion rate of the great egret was estimated using the allometric equation developed by Kushlan (1978). Assuming a body weight of 2.4 kg, the food ingestion rate was estimated to be 44 g (dry matter) per kilogram of body weight per day.

4.2 Sediment Ingestion Rates

Precise estimation of incidental soil or sediment ingestion rates for the modeled bird species is extremely difficult because of a lack of published studies. The only major survey available is by Beyer et al. (1994), who estimated sediment ingestion rates for various bird species, including ducks, geese, and sandpipers. Estimates ranged from less than 2 percent to 30 percent of the total dietary ingestion rate (on a dry weight basis). Of the species considered in the exposure models described here, direct observations of incidental soil or sediment ingestion are available only for the least sandpiper, which has been determined to consume sediment at a rate of 7.3 percent of the food ingestion rate (Beyer et al. 1994). A slightly more conservative rate of 10 percent of the food ingestion rate for least sandpiper was selected for use in the exposure model, to account for potential uncertainty in this estimate.

For the other bird species, best professional judgment was used to estimate ingestion rates based on comparisons of their foraging methods with species listed in Beyer et al. (1994), such that errors would result in reasonable overestimation of exposure. Beyer et al. (1994) estimated

sediment ingestion rates for species (ducks, geese, and sandpipers) that do not share the same feeding strategies as wading and diving birds (great egret, great blue heron, wood stork, double-crested cormorant). Some ducks and geese do not hunt fish but prefer to forage on the bottom of water bodies for various prey items, including aquatic and terrestrial plants and invertebrates. Sandpipers practice a different foraging strategy than piscivorous birds (benthic feeding) and are likely to incidentally consume relatively more sediment than wading or diving birds. Therefore, the upper range of the estimates for duck and goose sediment ingestion rates is considered to be a conservative estimate for the wading and diving birds.

The two highest sediment ingestion rates estimated by Beyer et al. (1994) for ducks and geese were 11 percent of the food ingestion rate for the wood duck, and 8.2 percent for Canada goose. The rounded average of these two rates (10 percent of the prey ingestion rate) was assumed to be a conservative estimate of the sediment ingestion rate for all wading and diving bird species in the exposure model. The species that was estimated to have the greatest potential exposure to incidental sediment ingestion was the American coot. Because this species forages almost exclusively within submerged aquatic vegetation, its incidental sediment ingestion rate is likely higher than that of wading and diving birds. The range of sediment ingestion rates estimated by Beyer et al. (1994) for four sandpiper species, also benthic feeders, was 7.3 to 30 percent of the food ingestion rate. The average of the two highest sediment ingestion rates (18 percent for the western sandpiper; 30 percent for the semipalmated sandpiper) is 24 percent of the food ingestion rate. Thus, the American coot was conservatively estimated to ingest sediment at 24 percent of total ingested food.

4.3 Food-Web Models

Daily exposure to the three organochlorine pesticides that are potential causal agents (DDTr [DDT plus its metabolites], dieldrin, and toxaphene) was estimated using a probabilistic exposure modeling approach. The three compounds have been detected in the fish and sediments of Lake Apopka (SJRWMD 2000) and are known to bioaccumulate in wildlife (Verschueren 1983). Ingestion of food and incidental sediment ingestion are considered the primary modes by which birds were exposed to these compounds. Sediment ingestion is included in the exposure model because of the high organochlorine concentrations known to be in the sediments. Exposure through surface-water ingestion was not evaluated because pesticides in Lake Apopka surface waters were undetected even in unfiltered samples. Because the pesticides under consideration possess very low water solubilities (Verschueren 1983), the exclusion of exposure through ingestion of surface water is not likely to significantly alter exposure model results.

The exposure model was designed to estimate exposure of the seven bird species to the above-mentioned organochlorine pesticides. The structure of the relationship used in this model was as follows:

$$f(E) = \int IR_{prey} \times \left(f([OC]_{prey}) + A_{Sed} \times f([OC]_{Sed}) \right) \frac{d[OC_{prey}]}{dP} \frac{d[OC_{sed}]}{dP}$$

where:

$f(E)$ = probability distribution of exposure (mg compound/kg bw/day vs. likelihood of occurrence within the population)

IR_{prey} = prey ingestion rate (mg/kg-bw/day)

$f([OC]_{prey})$ = probability distribution for the concentration of organochlorines in prey (mg/kg vs. likelihood of occurrence)

A_{Sed} = sediment ingestion rate (mg/kg-bw/day)

$f([OC]_{sed})$ = probability distribution for the concentration of organochlorines in sediment (mg/kg vs. likelihood of occurrence).

4.4 Distributions for Sediment Organochlorine Concentration

Because the spatial extent of inundated areas of the NSRA changed over time during the avian mortality event, the specific sediments that were available for incidental ingestion during foraging by the birds also changed. To account for these changes, flooding data were used to estimate water coverage on 10 discrete dates between September 9, 1998, and February 9, 1999. The dates were:

- September 9, 1998
- October 27, 1998
- November 2, 1998
- November 9, 1998
- November 24, 1998
- December 2, 1998
- December 7, 1998
- December 18, 1998
- January 25, 1999
- February 9, 1999.

An ArcView® geographic information system (GIS) coverage of the Lake Apopka site was created based on data provided by the State of Florida. This coverage was delineated into nine flooding blocks, consistent with the divisions used by SJRWMD, then further divided based on divisions created by drainage canals that traverse the flooding blocks. The sample stations were then posted onto this coverage. With the exception of the area immediately surrounding the Lust Farm airstrip, these stations were used to generate a set of Thiessen polygons surrounding a single sampling station (Figure 4-1). The area inscribed by the polygon was then assigned the

same organochlorine concentration as the associated station. In the vicinity of the airstrip, the spatial sampling density was too great to permit the development of meaningful polygons. Therefore, polygons were developed based on the sampling location of SJRWMD's 1999 sampling and the representative sediment concentrations were assigned as the mean of all sampling stations encompassed by the given polygon.

The relative area of flooded land was determined for each flooding block for each time period based on interpretation of field survey observations recorded by Jim Peterson of SJRWMD (SJRWMD 1999b), and from maps created by interpretation of two aerial photographs taken September 9 and December 7, 1998. Once the extent of water coverage was determined for each of the 10 time periods (Figures 4-2 through 4-11), sediment/soil sampling stations were identified as having been either inundated or dry. If a station was inundated during the given time period, then it was included as a potential source of pesticide exposure to the foraging birds. If the site was dry, then it was excluded. These data were then overlaid on the GIS construct, such that the organochlorine pesticide concentrations could be associated with the areas of inundation at each of the particular time periods. This union was then used to define a probability density function of sediment contamination concentrations for the entire area using the following integral:

$$P([OC]_{Sed}) = \int Area_{(x,y)} \times [OC]_{(x,y)} \frac{d[OC]}{dP}$$

where:

P([OC]Sed) = the probability density function for organochlorine concentrations

area(x,y) = the area of the specific polygon

[OC](x,y) = the specific organochlorine pesticide concentration attributed to the polygon.

4.5 Exposure Distribution for Prey ingestion

The exclusive food item modeled for all bird species, except for the American coot and the least sandpiper, was fish. Organochlorine concentrations in fish were modeled based on an assumption of equilibrium partition between sediment concentrations and fishes by multiplying the soil/sediment concentrations by the ratio of the chemical-specific biota-sediment accumulation factor (BSAF). Probability density functions for toxaphene, dieldrin, and DDTr BSAFs were derived from collocated data collected by the SJRWMD in August 1999 (SJRWMD 2000). Four separate locations were sampled (Duda West, Flow Way G, Zellwood East, and Zellwood South). All fish species were included with the exception of the Florida gar, which is outside the prey selection for all of the receptors considered. The organochlorine concentrations were standardized based on lipid mass in fish and total organic carbon (TOC) mass in sediments prior to generating the probability functions. For samples where any of the pesticides were undetected in either fish or sediment, a uniform distribution between zero and the detection limit was applied as an estimate of the actual concentration. The concentrations

were averaged for replicate samples. DDTr was calculated by summing DDD and DDE with DDT concentrations after application of DDT equivalence factors based on the relative toxic potency of the metabolites to the parent compound (Stickel et al. 1970). Frequency distributions were developed based on the likelihood of observing a given organochlorine concentration in sediment and a given organochlorine concentrations in fish. The BSAF distributions were then determined for each of the four locations using the following algorithm:

$$f(\mathrm{BSAF})_{\mathrm{Site}} = \frac{f([\mathrm{OC}]_{\mathrm{fish}})_{\mathrm{Site}}}{f([\mathrm{OC}]_{\mathrm{Sediment}})_{\mathrm{Site}}} \left(\frac{\mathrm{mg\,OC} / \mathrm{g\,lipid}}{\mathrm{mg\,OC} / \mathrm{kg\,TOC}} \right)$$

The model BSAF values between the four sampling locations (Duda West, Flowway G, Zellwood East, and Zellwood South) were regressed against mean sediment concentrations in order to develop the correlation coefficients necessary to account for interactions between the two variables. Standard correlation coefficients were determined to be r = –0.513, r = –0.383 and r = –0.377 for DDTr, dieldrin and toxaphene, respectively (Figure 4-12). The overall distribution of BSAF values was defined as the union of all four sampling locations and was determined as follows:

$$f(\mathrm{BSAF})_{\mathrm{Overall}} = P\left(\bigcup_{n=1}^{i} \mathrm{BSAF}_{\mathrm{Site\,n}}\right) = \sum_{n=1}^{i} p(\mathrm{BSAF})_{\mathrm{Site\,n}}$$

Where:

$$f(\mathrm{BSAF})_{\mathrm{Site\,n}} \left(\frac{\mathrm{mg\,OC} / \mathrm{g\,DW}}{\mathrm{mg\,OC} / \mathrm{kg}} \right) = f(\mathrm{BSAF})_{\mathrm{Site\,n}} \left(\frac{\mathrm{mg\,OC} / \mathrm{g\,lipid}}{\mathrm{mg\,OC} / \mathrm{kg\,TOC}} \right) \times \left(\frac{\mu_{\mathrm{g\,lipid} / \mathrm{kg\,DW}}}{\mu_{\mathrm{kg\,TOC} / \mathrm{kg}}} \right)_{\mathrm{Site\,n}}$$

The probability distributions for DDTr, dieldrin, and toxaphene based on this derivation are provided in Figure 4-13.

There were two exceptions to the above parameterization approach. Both the American coot and the least sandpiper were assumed, for modeling purposes, to consume a diet exclusively of benthic macroinvertebrates. Site-specific data on the equilibrium partition between sediment and benthic macroinvertebrates were not available. Therefore, exposure from food ingestion was derived from the probability density functions to describe the sediment concentrations using static BSAF values derived from the literature. The values used were 0.019, 0.389, and 0.057 for DDTr, dieldrin, and toxaphene, respectively.

4.6 Model Execution

To estimate the total exposure of each bird species, the above model was run multiple times to develop the exposure probability density functions. The parameters were selected from the distributions by Monte Carlo simulations using Crystal Ball 2000 (Decisioneering, Inc.). The number of trials performed to develop the exposure distributions was ≥10,000 iterations. The

probability density functions were estimated based on the cumulative frequency of occurrence for all potential exposure rates during the simulation.

4.7 Food-Web Model Results

For all of the piscivorous species, the variation in total exposure concentrations of toxaphene, dieldrin, and DDTr did not change appreciably between September 9, 1998, and February 9, 1999. Figure 4-14 illustrates this trend for exposure of the American white pelican to toxaphene. The only variance was a slight increase in the exposure rates for the 10th to 60th percentile during the last two assessment periods (January 25 and February 9, 1999). Both these periods represent time periods after the first significant pelican die-off. Therefore, for the piscivorous birds, the exposure concentrations were assumed to be reasonably constant, and the date of December 18, 1998, which corresponds most closely with the start of the mortality event, was used for the relative comparisons discussed below.

The time-course patterns observed for the piscivorous birds did not hold for the two benthivorous species. Most of the exposure probability functions were similar, with the exception of the first one, corresponding to September 9, 1998, and last one, which corresponded to February 9, 1999. At these times, the exposure probability functions demonstrate a comparatively higher proportion of organochlorine pesticide exposure for these two species particularly between the 10th and 50th percentiles (as illustrated for the least sandpiper in Figure 4-15), the earlier increase was likely the result of the inclusion of upland regions, such as the airstrip, in the assessment area. These areas were the first to be dried down and therefore removed from future consideration. The later increase is believed to be due to a reduction in the extent of the inundated area. As the flooding blocks dried and were removed as sources of potential exposure, the overall flooded area of the NSRA was reduced. This decrease in flooded acreage increased the sensitivity of the exposure model to variations in organochlorine pesticide concentrations in the sediments and resulted in an increase in the proportion of the populations exposed to higher concentrations of organochlorine pesticides.

In comparing exposure estimates among all species, exposure to toxaphene was estimated to be higher for the American white pelican than for the other piscivorous bird species (Table 4-2), as well as the benthivorous species, American coot and least sandpiper (Figure 4-16, Table 4-2). Exposure to dieldrin was comparable among all species (Figure 4-17, Table 4-3). Therefore, provided birds are equally sensitive to acutely toxic effects, mortality rates at Lake Apopka for the American white pelican should have been higher than those observed for the other receptors modeled. Furthermore, Figures 4-18 and 4-19 show that the exposure for the American white pelican is higher than exposures for the other piscivorous species. This may account for the observations that many more pelicans died on the north shore of Lake Apopka than any other piscivorous species.

Similar findings were predicted based on exposure to DDT. The American white pelican was consistently found to have higher exposures than both the other piscivorous receptors (Figure 4-20) as well as the benthivorous receptors (Figure 4-21, Table 4-4).

Comparisons of the daily exposures for the American white pelican to DDT, dieldrin, and toxaphene indicated that the potential exposure to toxaphene was higher than the exposure to either DDT or dieldrin. The daily exposure for toxaphene at the 95th percentile was estimated at 12.56 mg/kg-day, compared to 0.75 and 0.22 mg/kg-day for DDT and dieldrin, respectively. All of these exposures are below the minimum avian LD_{50}s determined for these compounds, which were 23.7 mg/kg-day for toxaphene, 595 mg/kg-day for DDT, and 8.78 mg/kg-day for dieldrin (Hudson et al. 1984). These results argue against any single organochlorine pesticide being responsible for the acute avian lethality that occurred.

The highest concentrations of toxaphene in soil have been recorded in Flooding Block 6, adjacent to an airplane landing strip. The mean toxaphene concentration in this hot-spot area was 86.2 mg/kg, and the median concentration was 120 mg/kg. Aerial photographs taken on December 7, 1998, indicate that this area was not flooded at a time approximating the start of the mortality event. Earlier photographs, taken in September 1998, suggested that the region might have been inundated at this time (SJRWMD 2003). Although the American white pelican population at that time was very small, other avian species such as great egret, wood stork, and the small herons were in relatively high abundances (Figure 2-1). Thus, it is very unlikely that the pelicans would have been exposed to these high toxaphene concentrations while the great egrets, wood storks, tricolor herons, and little blue herons could very well have been exposed.

To determine the magnitude of this theoretical exposure, the models were calibrated to assume all of the receptors' exposure was derived from sediment and fish confined to the immediate vicinity of the airstrip (Figure 4-22). The exposure model indicated that the lowest LD_{50} would be exceeded at approximately the 60th percentile. However, similar results were reported for all piscivorous receptors. The benthivorous birds showed a lower risk, with the 7th percentile crossing the LD_{50}. This suggests the possibility that exposure to toxaphene soils in the hot-spot area could have resulted in acute mortality in 40 percent of the population of all piscivorous birds and 7 percent of the benthivorous birds exposed exclusively to the airstrip vicinity.

In the absence of sufficient organochlorine exposure rates to induce acute toxicosis, the potential that birds may have developed steady-state concentrations of toxaphene sufficient to cause lethality was examined. Biessman et al. (1983) measured the excretion of ^{14}C-labeled toxaphene in Japanese quail and determined that over half of the radioactive dose administered was excreted within 3 days. This can be considered a conservative half-life because it is based on total amount consumed versus total amount excreted, and therefore will not be affected by mechanistic considerations such as compartmentalization which would be a consideration if the half-life were based on changes in toxaphene blood concentrations. Using this half-life, the overall body burden for the white pelican was modeled using the following relationship:

$$[OC]_t = \left(\left([OC]_{intake} \times BW\right) + [OC]_{(t-1)}\right) e^{-kt}$$

where the body burden $[OC]_t$ was assumed to be the sum of the organochlorine pesticide intake rate ($[OC]_{intake} \times BW$), less the first-order elimination rate. The model was iterated until the overall body concentration was observed to reach steady state.

The comparative benchmarks used to evaluate the results of the steady-state model were the expected overall body burdens resulting from one-time exposures to toxaphene doses equivalent

to 23.7 mg/kg-day minimum, and 100 mg/kg-day maximum, which were the two lethal doses reported for white pelicans. The results of the steady-state modeling (Figure 4-23) show that the toxicity threshold would not be exceeded at the 75th percentile of exposure, but would at the 95th percentile. The actual proportion of the population that would exceed the lowest LD_{50} estimate would be 19 percent, which is similar to the estimate of 16 percent mortality in the white pelican population. No proportion of the pelican population exceeded the maximum estimated LD_{50}.

4.8 Uncertainty

The parameters used in the food-web model represent the major sources of uncertainty. The exposure model was parameterized with data from the literature. Where information on ecological parameters was incomplete or unavailable, conservative assumptions were made. This approach may have resulted in overestimates of exposure that must be considered in the comparative evaluation of the results.

The greatest source of uncertainty was in the assumption that the organochlorine concentrations in the fish tissue are proportional to the sediment concentrations on the north shore of Lake Apopka. This assumption was necessary, because the only fish tissue data available were acquired in August 1999, after the avian mortality event, and were used in this case to derive site-specific BSAFs. Time-course analysis of organochlorine concentrations suggests that DDTr concentrations in blue tilapia measured in January and February 1999 were consistent with those measured in August (Figure 4-26). However, similar time-courses for dieldrin and toxaphene organochlorine concentrations in tilapia had declined in the interval between January and August (Figures 4-27 and 4-28). This may have resulted in an under-prediction of the actual onsite BSAFs. Unfortunately, limitations in the dataset required a default assumption of linearity between fish and sediment concentrations. Also, none of the relationships demonstrated good fits, suggesting that factors other than sediment organochlorine concentrations contributed significantly to controlling organochlorine bioaccumulation in fish.

Another significant source of uncertainty in the prediction of onsite BSAFs resulted from the comparatively low sediment concentrations in the measured areas. This required the linear extrapolation of the fish body burdens, in some cases to over 100 times the actual observed relationship in order to predict organochlorine concentrations in the prey fish. Hence, there is very little confidence that the estimated organochlorine concentrations in the fish are representative of conditions at the time of the pelican die-off.

Another area of potential uncertainty involves the assumptions of prey intake by the receptors. No adequate estimates could be found for the selected receptors with the exception of the white pelican. Intake rates for the other species were based on weight-standardized power regressions developed from interspecies comparisons. This compounds associated observational error with model error which could be significant, particularly close to the maximum and minimum ranges of the comparison set.

Much of the uncertainty that could be quantified was incorporated into the exposure model as distributions, and therefore is represented in the exposure probability functions. Considering the

limitations of the available data, Exponent considered probabilistic exposure modeling to be the most accurate and informative method available.

4.9 Potential Synergism among Organochlorine Pesticides

One consideration in the analysis of avian mortality at the NSRA was potential interaction between different organochlorine pesticides. Numerous organochlorine pesticides were detected in the soils and sediments in this area and of these, three were detected in significant concentrations in resident fish tissues: dieldrin, DDT (and metabolites, denoted collectively as DDTr), and toxaphene. Predicting a quantitative toxicological response of a receptor exposed to more than one toxicant is extremely difficult in the current context of our understanding of toxicology. The potency and efficacy of a toxicant depend on numerous chemical and biological properties, including:

- Considerations of transport, such as availability for uptake from the environment
- Considerations of chemical interactions with biological constituents (i.e., protein binding, lipid accumulation, etc.)
- Considerations of compartmental deposition (adipose accumulation, bone deposition, etc.)
- Considerations of sensitivity at the site of action
- Considerations of relative detoxification and excretion rates.

Dieldrin, DDT, and toxaphene are all neurotoxic. Dieldrin is a cyclodiene and toxaphene is a mixture of bicyclomonenes whose mode of action involves antagonism of the gamma-aminobutyric acid (GABA) receptor in the central nervous system, which causes a hyperexcitability of post-synaptic membranes resulting in a disruption in signal conductance (Matsumura 1985). DDT (and to a lesser extent its metabolites) binds to sodium channels in both the central and peripheral nervous systems resulting in prolonged channel activation leading to an initial hyperexcitability, but ultimately inducing a loss in the ability of axons to propagate action potentials by inhibiting the axon's ability to maintain membrane potential (Ecobichon 1996). There is some evidence that DDT may also have limited activity at the GABA receptor (Matsumura 1985). Therefore, because all three of these toxicants are neurotoxic in that they affect nerve signal transmission, to a greater or lesser extent at the GABA receptor, there is the potential that simultaneous exposure to all three of these organochlorine pesticides could result in acute toxicity at concentrations lower than exposure to any of them individually.

To model the potential for additivity, the concentrations of DDTr, dieldrin, and toxaphene were combined to determine whether together, they could represent sufficient exposure to induce mortality in white pelicans. Comparisons of intake concentrations obtained from the exposure models developed for the north shore of Lake Apopka indicate that the highest exposure rates resulted from toxaphene (Figure 4-24). Exposure rates determined for toxaphene represent

7.6 percent (at the 50th percentile) to 46 percent (at the 95th percentile) of the minimum LD_{50} of 23.7 mg/kg-day for this organochlorine. The second highest rates were from exposure to DDTr. However, because the minimum LD_{50} for DDTr is 595 mg/kg-day, this exposure rate represents only 0.0002 to 0.001 percent of the LD_{50} at the 50th percentile and maximum exposure rates, respectively. Dieldrin had the lowest exposure rates. However, it also has the lowest minimum estimate for the avian LD_{50} (8.78 mg/kg-day). The 50th percentile estimates of population exposure equaled 0.0046 percent of the minimum LD_{50}, while the 95th percentile exposure concentration equated to 0.025 percent.

In order to combine these compounds, concentration estimates were weighted based on their comparative toxicity relative to toxaphene. This was done by multiplying the estimated exposure concentration by the ratio of the minimum toxaphene LD_{50} by the minimum LD_{50} for the specific organochlorine compound. An example for dieldrin is as follows:

$$[\text{Dieldrin}]\left(\frac{\text{mg}}{\text{kg}-\text{day}}\text{ Toxaphene equivalents}\right)=[\text{Dieldrin}]\left(\frac{\text{mg}}{\text{kg}-\text{day}}\right)\times\frac{\text{LD}_{50}\text{ Toxaphene}}{\text{LD}_{50}\text{ Dieldrin}}$$

The highest potential for adverse effects is a result of toxaphene exposure (Figure 4-25). However, dieldrin has a higher potential for causing adverse effects than DDTr because of its relatively high acute toxicity. Exposure concentrations of DDTr, expressed as toxaphene equivalents, were extremely low; the range of potential impact was three orders of magnitude less than toxaphene itself. When the three rates were combined, the result, expressed as toxaphene equivalents, was not very different from that of toxaphene alone. Fiftieth percentile concentrations at the T9 time interval increased from 2.08 to 2.43 mg/kg-day and the 95th percentile increased from 12.56 to 18.59. These increases represent 8.9 and 68.1 percent of the minimum acute avian LD_{50} for toxaphene at the 50th and 95th percentile exposure concentrations, respectively.

The 95th percentile toxaphene exposure concentrations estimated for birds at the NSRA were less than half the minimum avian LD_{50} for this organochlorine pesticide. Simultaneous exposures to dieldrin and DDTr, on a weighted relative toxicity basis, had a very limited effect on the estimates of potential for impact. If this multiple exposure did result in acute toxicity in the white pelicans, then exposure to the combination of dieldrin and DDT would had to have potentiated the effects of toxaphene twice the levels resulting from pure additivity. This is theoretically possible and, hence, it is not possible at this time to conclude that the suspected toxicity was not the result of synergism between the organochlorine pesticides.

Currently, there is no adequate means to justify the uncertainty associated with this estimate of acute toxicity. The estimate is based on the assumption that these three organochlorine pesticides are acting upon the white pelican independent of each other in proportion to their toxicities if applied individually. It is possible that these three agents may very well interact at various physiological levels. At the site of action, it would be expected that these pesticides may compete for binding sites and could result in a reduction in the acute response to the most toxic constituent by reducing its resident time at the receptor. For example, if dieldrin possesses a greater potency than toxaphene at the GABA receptor, then dieldrin would be potentiating to toxaphene, but toxaphene would be mitigating for dieldrin. This is a well-known phenomenon and the basis for many toxicological antidotes. Likewise, exposure to multiple agents could

induce detoxification mechanisms such as the mixed function oxidases, which would also result in a reduced toxic response. Alternatively, agents working at different sites, such as toxaphene and DDT, could result in a potentiation of the response by inducing compounding disruptions. Unfortunately, there is no way to be certain as to the nature of the interactions. The estimate of toxicity provided above, therefore, represents an accurate assessment, providing the agents act as if they were independent of each other. Verification of this estimate would require performing multiple dosing experiments specific to the white pelican.

4.10 Conclusion

The results of the exposure model indicated that the American white pelicans, compared to the other receptors modeled, experienced the highest organochlorine exposure rates. However, the differences in daily exposures were apparently not large enough to explain the differences in mortality rates observed between the bird species during the avian mortality event at Lake Apopka, unless pelicans are much more sensitive than other species to organochlorine pesticide toxicity. Great blue herons also displayed high mortality rates relative to other species, yet daily exposure estimates for this species did not differ from estimates for other fish-eating wading birds. This could indicate greater sensitivity in great blue herons, or perhaps more prolonged exposure to organochlorines, as mortality primarily occurred in mid-March, after the peak of pelican mortality.

Analysis specific to the white pelican strongly suggests that the maximum daily exposure for the three detected organochlorine pesticides, either singularly or in summation, was not sufficient to induce mortality, even assuming that the sensitivity of this species was represented by the most sensitive avian species ever tested. However, steady-state modeling did indicate that a significant proportion of the white pelican population (19 percent) could potentially have accumulated sufficient toxaphene concentrations to cause death.

5 Etiology and Pathology of the Mortality Event

5.1 Introduction

To better characterize the potential causes of the mortality event, reports from necropsies performed on different bird species were examined. The results from these reports were compared to published historical accounts that described mortalities known to be caused by organochlorine pesticide poisoning. The objective was to determine the weight of evidence in support of the hypothesis that organochlorine pesticides were directly involved as the cause of death in any of these cases.

5.2 Etiology

Conditional etiology in the context of this discussion is defined as the signs and symptoms presented by a bird just prior to death. The etiology of organochlorine pesticide poisoning is reasonably specific, because it is the result of targeted impacts on nerve signal transduction. Post (1951) performed a detailed study on the acute effects of toxaphene and chlordane on the Galliformes (pheasants, grouse, and partridge). His description of the etiology of mortality was as follows:

> The first symptom noted was locomotor effects. There was an unsteadiness in walking with a drooping of the wings until the wing feathers touch the ground to give aid in walking. Ocular symptoms were noted at this time. The eyes were closed and opened apparently in an attempt to clear the vision. There were periods of apparent total blindness as the finger could many times touch the cornea before the eye would be closed. There was usually much clucking and fluffing of the feathers. At all times there was much quivering and palsy. Shortly before death the birds had periods of extreme tetany followed by periods of relaxation and listlessness. Death nearly always occurred with extreme tetany, violent convulsions, and rigor of all skeletal muscles. Sage grouse usually died on the breast under the cover sagebrush. Pheasants sometimes died on the breast and sometimes on the back. Chukar partridge always died on the back. The legs, in all three species, were usually pushed back as far as possible as were the wings. The head was usually thrown back as far as possible.

The etiology described by Post (1951) is common in cases of specific poisoning that involves disruption of inhibitory neurotransmitters. Inhibition of GABA in the central nervous system will manifest as hyperexcitability, leading to tonic/clonic seizures. Animals thus affected almost always die in extreme tetany and usually in a hyperflexive orientation (Ecobichon 1996).

A reasonable level of expertise is required to discern diagnostic signs at time of death that are absolutely specific to organochlorine toxicoses. Convulsions at death do result from organochlorine poisoning; however, they are also common to numerous disease states, such as

Newcastle disease, chlamydiosis, duck plague, aspergillosis, or bacterial septicemia (Friend 1987). Other conditions such as extreme malnutrition and dehydration may also cause convulsions at the time of death. Indicative inferences may be drawn from the characteristics of a convulsive state. For example, asymmetric convulsion may indicate a chronic degeneration of the central nervous system caused by disease states such as Newcastle disease. Signs of toxaphene poisoning would be stress-inducible tonic/clonic seizures (i.e., an external stimulus, such as noise, would induce convulsions in an affected individual) that would affect all parts of the body equally, with hyperflexion immediately prior to and after death. The ability to discern subtle differences in the manifestation of a convulsive state requires expertise and should be used only as supporting evidence in association with other pathological findings.

A videotape produced by Harold Weatherman of SJRWMD on January 28, 1999, recorded 3 minutes, 55 seconds of a white pelican on dry land apparently undergoing a form of seizure. Personal communications with witnesses from SJRWMD suggest that the signs exhibited by the pelican during the taped seizure were common for many of the white pelicans that died at the NSRA (Stites 2000, pers. comm.). The commentary provided in the tape indicates that the bird was initially quiescent prior to initiating the recorded seizure. The recording is continuous, with the exception of one point where the recording was stopped for an unknown period of time. The record does not progress completely to the bird's death.

In the initial portion of the recorded seizure (as determined by comments attributed to H. Weatherman), the white pelican was resting on its sternum, with wings partially extended and neck and head resting on the ground. The disposition of the legs and feet could not be determined from the angle of the camera. The convulsive movements of the bird were apparently coordinated, as opposed to being tonic/clonic. Intermittent bill clapping was reported in the narrative and was heard on the tape. The apparent bill clapping occurred at a rate of approximately one to two per second, which is considered relatively slow for a tonic/clonic seizure. There may have been some voluntary movement by the bird that was obstructed by a lack of coordinated muscular control, but this is difficult to determine because the bird was not manually challenged. Toward the end of the recording, there appears to be an increase in the clonus of the seizure, particularly within the head and neck regions, but this did not appear to be manifested in the thoracic parts of the body nor in the wings. At the very end of the recording, there is a moderate flexion noted in the neck; however, it is not presented as a tonic/clonic seizure. This flexion may be a continuation of the earlier observed coordinated seizure, or may be the result of a voluntary flexion on the part of the bird as an attempt to raise its head by levering its beak against the ground. Tonus within the body trunk could not be determined visually and would have required palpation for confirmation. In either case, the extent of neck flexion could not be classified as either hyperflexive or hyperextensive, but rather as a moderate torticolis.

Other incidents of seizures were reported in field notes from both the Audubon Society and FWS (U.S. FWS 2001). Most of these reports were curt in nature and provide little in the way of details that could be applied to support or refute a finding of organochlorine toxicoses. There were some inconsistencies in the varied reports that were noteworthy. They were as follows:

- Hyperflexion was only specifically reported in two cases: U.S. Geological Survey (USGS) 16138-009 (diagnosis dieldrin poisoning) and 16138-010 (diagnosis dieldrin poisoning)
- A clonic state at time of death were reported for FWS Lab 1 (diagnosis suspected toxicosis) and Lab 2 (diagnosis suspected toxicosis)
- Non-descript convulsions for SJRWMD W99-210 (diagnosis suspected organochlorine toxicosis)
- Convulsions described as "twitching and head and eye tremors" were reported for SJRWMDC 99-97 and 99-90 (undetermined)
- Disorientation with no convulsions for 03-99 (diagnosis undetermined), W99-203 (diagnosis undetermined)
- No report of any seizures for SFWMD 64-99 A (diagnosis suspected toxicosis), W99-213 (diagnosis suspected organochlorine toxicosis).

Hyperclonus was only reported in two cases, both diagnosed as acute dieldrin poisoning. For other suspected cases, the types of neurological responses ranged from none noteworthy to mere disorientation to mild twitching. Of all the reports reviewed, only USGS 16138-009 and 16138-010 reported hyperflexive convulsions prior to death.

Other potential causes of death manifest signs similar to those described in anecdotal reports of the white pelican mortalities, as well as the above-mentioned videotape and field reports, that are completely independent of organochlorine poisoning. For example, descriptions of the etiology of death resulting from a particular strain called velogenic Newcastle disease are similar to those observed on the video record. A study reported by Meteyer et al. (1997) described the etiology of death from velogenic Newcastle disease in double-crested cormorants from the Dakotas and Nebraska. Clinical signs included ataxia, unilateral weakness of legs and/or wings, and torticolis (spasmodic contractions of the muscles of the neck resulting in the head being drawn to one side and usually rotated, similar to the behavior seen on the videotape). Kuiken et al. (1998) described the clinical etiology of velogenic Newcastle disease based on an outbreak of the disease in double-crested cormorants on Dore Lake in central Saskatchewan. The authors describe the initial clinical signs as being partial to complete paralysis of selected limbs (with the exception of the neck and head regions), inability to stand (resting on sternum), and partial to full wing spread (usually for balance). The signs progressed to include blindness, coordinated seizures, unilateral tremors, and torticolis. Other disease states such as chlamydiosis, botulism, and even West Nile Fever can cause similar types of convulsions. Other potential causes of torticoloid convulsions include electrolyte imbalance associated with dehydration, or extreme starvation.

5.3 Pathology

SJRWMD made available to Exponent a series of 31 post-mortem necropsy reports on white pelicans (25 individuals), wood stork (2), great blue heron (2), great egret (1), and bald eagle (1). The majority of the necropsies (22) were performed by Dr. Marilyn Spalding of the University of Florida. Of the remaining necropsies, six were performed by Dr. Joseph Gaydos and two by Dr. Todd Cornish, both of the University of Georgia, and one necropsy was performed by Dr. Gregory Bossart of the University of Miami.

Another 67 necropsy results were released by FWS and made available to Exponent in 2002 (U.S. FWS 2001). Of these additional reports, 59 were of sufficient post-mortem condition to provide adequate necropsy results and included wood storks (21), white pelicans (15), great blue heron (3), common egret (3), bald eagle (3), black-crowned night heron (2), short-billed dowitcher (2), ring-billed gull (2), yellow-crowned heron (1), great egret (1), herring gull (1), double-crested cormorant (1), snowy egret (1), pintail duck (1), Eurasian curlew (1) and a water snake (1). Dr. Carol Meteyer of the U.S. Geological Survey performed 53 of the examinations with the remaining 14 being performed by Dr. Richard Stroud of FWS. From both sets of necropsies, 46 listed the cause of death as resulting from, or potentially related to, organochlorine toxicosis. A matrix summarizing the results of the necropsies purportedly related to organochlorine exposure is provided in Table 5-1.

5.4 Gross Pathology and Histopathology

The review of gross pathology and histopathology is based on the aforementioned necropsy reports made available to Exponent by SJRWMD in 2000 and from FWS in 2002. These reports detailed post-mortem necropsies performed on white pelicans and other bird species present at the NSRA.

The gross pathology associated with toxaphene poisoning, as outlined by Post (1951) is as follows:

- **Cardiac:** Separation of muscle bundles, small petechial hemorrhages and hyperemia
- **Hepatic:** Pronounced hyperemia, predominant fatty infiltration, vacuolization, and cloudy swelling or albuminous degeneration; the degree of degeneration was reported to be dose-dependent
- **Renal:** Vacuolization, cloudy swelling or albuminous degeneration, moderate to severe hyperemia, and diffuse fatty infiltration particular to the proximal convoluted tubules
- **Spleen:** No pathological impact; normal architecture with cellular pulp

- **Gastrointestinal tract:** Varying degrees of exfoliation and desquamation, with large amounts of epithelium debris within the intestinal lumen
- **Neurological:** Severe vacuolization and clustered hyperemia.

Observations from a review study on the pathology and toxicology of aldrin and dieldrin, undertaken by the President's Advisory Committee and presented in 1965 (Hodge et al. 1967), are as follows:

- **Hepatic:** Tan liver with associated lobular degeneration; central lobular hypertrophy and hyperplasia in the liver with cytoplasmic oxyphilia and peripheral accumulation of basophilic granules
- **Pulmonary:** Pulmonary congestion and associated pneumonia
- **Renal:** Nephritis and tubular degeneration
- **Neurological:** Axonal degeneration and vacuolization particular to cerebellum and to a lesser extent in the medulla
- **Hematological:** Slight to moderate increase in serum cholinesterase activity.

Hodge et al. (1967) noted that the most common pathological manifestation associated with toxaphene poisoning was the vacuolization seen in the liver, kidney, and brain. He noted that the degree of this condition was dose-dependent, and in some cases, the organ was "so badly vacuolated that normal tissue was hard to find." An evaluation done with differential staining by the same author demonstrated that these vacuoles were not the result of fatty degeneration, because their contents were aqueous and not lipid in nature.

In examining the necropsy results from birds reported to have died from suspected toxicosis, 30 of 45 cases reported gastrointestinal contents. Of these 30, it was noted that 24 had little or no food content. A further 5 reported plant material in species that rely predominantly on a diet of fish. These observations suggest that most of the birds that were investigated suffered from a severe disruption in their feeding behaviors and had not been able to obtain food in at least 24–48 hours prior to death. Nine of the birds were reported in good nutritional state, suggesting that this condition was short term. However, 21 of these 30 birds were also reported to be in fair to poor nutritional states (denoted by depletion of fat stores), indicating that the inability to properly feed may have been conditional for approximately 1–3 weeks.

Chronic organochlorine exposure has been reported to cause a general wasting condition (Ecobichon 1996). Disruption in feeding behavior resulting in wasting is a very general condition and is usually manifest in most toxicological, physiological, and pathological conditions that result in disruptions in homeostasis. However, the lack of stomach and intestinal contents does indicate that if death were the result of a organochlorine toxicosis, it was not the result of an acute exposure because almost all of the birds were able to survive from days to weeks after their last substantial ingestion of food.

Little consistency can be found in the results of examination of the gross pathology and histopathology of the organ systems. The most commonly reported lesions included spleen lymphoid depletion and/or atrophy (24 of 31 individuals), kidney nephrosis (13/32), hepatic congestion/hepatitis (19/30), epicardial conditions such as hydropericardium, mild epicardial hemorrhaging and/or congestion (13/27), pulmonary congestion (11/22), adrenal hypertrophy/hyperplasia (9/13) and peritonitis/enteritis (8/27). Brain gross pathology remarks are available for 22 of the birds examined. Most were unremarkable with the exception of congestion or hemorrhage reported in two of the birds. Microscopic examinations were performed on the brain and other neurological tissues of only nine birds. All exhibited vacuole-like lesions: two reported clusters of objects resembling trophozoites (location unreported), two had dilated axonal sheaths specific only to the cerebellum, with intracytoplasmic vacuolization localized to the gray matter of the brain stem, and two reported undefined vacuolization in the brain stem, two reported undefined vacuolization in the optic regions, and one reported vacuolization in the thalamus.

Very few of the gross pathological manifestations identified by Post (1951) for toxaphene and Hodge et al. (1967) for dieldrin as being associated with toxaphene poisoning were noted in the available necropsy reports for birds from the NSRA. Noted comparisons were as follows:

- **Cardiac:** Epicardial hemorrhage was noted in only two of the 45 birds. The condition in W99-209 was described as mild and condition in W99-220 was purported to be secondary to avian cholera. The commonly reported hydropericardium appears inconsistent with toxaphene toxicosis. There were no reports of moderate to severe hemorrhage, nor any reported sign of muscle bundle separation.

- **Hepatic:** Only two birds were reported to have the mottled and tan liver associated with vacuolar degeneration (C99-1239 and 16188-001). One bird was reported to have tan foci, but no mention of vacuolization was made (16188-001). Hepatitis/inflammation was also reported in eight birds (W99-203, 99-90, 99-97, W99-208, W99-209, 16138-009, 16188-002, and 16188-006). Several other birds were observed to be suffering mild to moderate hepatic congestion and inflammation that is not directly consistent with toxaphene poisoning.

- **Renal:** Nephrosis was reported in seven of the birds. Most of these were reported to be manifest as a multi-focal necrosis with associated inflammation. None of the necropsy postmortems reported renal vacuolization consistent with that reported by Post (1951). Even pelican case number CP99-1239, which manifested the mottled and tan hepatic lesions with associated vacuolization, did not manifest the condition in the kidneys, but rather presented a general nephrosis and multifocal pyelitis that was inferred to be the result of an infection.

- **Spleen:** One of the most consistent observations noted in the necropsy record was severe to moderate lymphoid depletion. It was also noted that several birds were suffering splenic atrophy and pallor. These conditions are most closely associated with infection and are not cited as being consistent

with organochlorine toxicosis. It also strongly suggests that both the humoral and cellular immune cascades were active and not subject to organochlorine-induced suppression.

- **Gastrointestinal tract:** The most common pathology reported was associated with mechanical damage and associated inflammation and infection. There was no indication of the exfoliation and desquamation reported to be associated with toxaphene poisoning.

- **Neurological:** Vacuolization was reported in the four birds for which brain pathology was available. However, the histological manifestation was not consistent with that reported by either Post (1951) or Hodge et al. (1967). Hyperemia, manifest as congestion and consistent with organochlorine toxicity was reported in only one bird (Lab-6).

The comparison of reported pathology with that described by Post (1951) and Hodge et al. (1967) indicates that the majority of the birds for which post-mortem necropsies were available were not manifesting pathological lesions consistent with known acute toxaphene or dieldrin poisoning. Some specific lesions were present, but their manifestations were inconsistent and were usually not to the degree that would be expected in cases of acute toxicosis. Furthermore, the pattern of pathological signs reported in the necropsy results could be associated with conditions independent of organochlorine poisoning. For example, hydropericardium was a reasonably consistent lesion among the necropsied birds. This sign was also reported among the double-crested cormorants known to have died from Newcastle disease in central Saskatchewan (Wobeser et al. 1993). Likewise, two of the four white pelicans for which neurological pathology are available reported intracytoplasmic vacuoles associated with the gray matter of the brain stem, as well as axonal degeneration and aggregate intramylegenic edema specific to the cerebellum. Similar types of pathology were noted by Wobeser et al. (1993) in 12 of the 18 cormorants that were known to have died from Newcastle disease. Other signs such as hepatitis, nephritis, and depletion of the spleen were also observed to be associated with Newcastle disease (Meteyer et al. 1997) and reported in the necropsies from the birds found on the north shore of Lake Apopka.

Based on the comparative evidence between the available post-mortem necropsy results and published reports describing the manifestations of both gross and histopathology associated with toxaphene or dieldrin poisoning, a definitive attribution of cause of death to organochlorine toxicosis cannot be made for any of these birds. More likely, the birds succumbed to multiple, compounding, but independent conditions. Conclusive evidence, aside from measured organochlorine concentrations in brain tissue, that demonstrate a pattern of toxicosis resulting from exposure to toxaphene or any other organochlorine pesticide is absent based on a review of available information. Discussions of organochlorine concentrations in avian tissues are provided in Section 3.

5.5 Virology and Bacteriology

Thirty-three of the 45 birds necropsied underwent no disease testing whatsoever. Thirteen of the birds were subjected to liver culturing, which is a very preliminary screening protocol and will detect only bacterial infections. Of these birds, five were reported to culture positive, with *Clostridium sp.* being the most common pathogen identified. Seven individuals were titer tested for Newcastle disease, five for botulism, and one for cholera. Of these seven, five were reported to have positive titers for Newcastle disease at very low concentrations. Of the five birds tested for botulism, two were reported as equivocal and the rest as negative. The one bird tested for cholera was reported as positive.

5.6 Conclusions

Organochlorine pesticides produce a very specific toxic syndrome, because they are specific neurological agents that disrupt an organism's ability to control nerve signal transduction. These pesticides are also known to produce specific secondary toxic effects in tissues such as the liver and kidney by virtue of their lipophilicity and requirements for catabolic metabolism. The signs and symptoms of these effects were not consistent with the pathology observed in these birds, either as a group or within individuals. For example, bird CP99-1239 was reported to have hepatic lesions (mottled and tan vacuolization), which are consistent with toxaphene poisoning. However, it was the only bird to manifest this particular lesion. Other pathological manifestations associated with organochlorine toxicosis, such as kidney vacuolization or muscular hyperemia, were not reported for this bird. Likewise, bird 1-99 showed evidence of brain vacuolization, which may be consistent with toxaphene poisoning. However the same bird did not manifest any of the other pathological injuries known to be associated with exposure to acute concentrations of this organochlorine pesticide. In all, none of the birds manifested more than one of the signs or symptoms identified by Post (1951) or Hodge et al. (1967) as resulting from toxaphene or dieldrin poisoning.

Therefore, notwithstanding conclusions presented in the available necropsy records, there appears to be no singular, consistently identifiable cause of death. There is some evidence that a pathogen, such as velogenic Newcastle disease virus (NDV) may have been involved in some of the cases. The extremely limited information on the etiology of mortality for the birds from the NSRA suggests signs more consistent with the progression of Newcastle disease than with organochlorine pesticide poisoning. For example, five of the seven birds tested for Newcastle disease were positive for NDV antibody titers. This fact, by itself, is not indicative because NDV is commonly found in numerous individual birds that do not manifest the acute pathology, and manifestations of overt Newcastle disease have been reported to occur anywhere up to 22 weeks after the virus is no longer detectable (Wobeser et al. 1993). The presence or absence of NDV titer must be examined within the context of other pathological signs associated with Newcastle disease. For example, spleen depletion (noted in 12 of the 26 birds) and hydropericardium (in three birds) have been reported in double crested cormorants that are known to have died as a result of Newcastle disease (Wobeser et al. 1993). Furthermore, of the three birds reported to have had hydropericardium, all were also suffering severe spleen depletion.

Notwithstanding the presentation of the pathological conditions of the birds, there were very high toxaphene and dieldrin concentrations in brain tissues taken from some of the necropsied birds but not white pelican. In some cases, the reported concentrations of dieldrin were over five times the assumed toxic concentrations. This is highly indicative of potential pesticide toxicosis. However, there are numerous inconsistencies with these reports. For example, it is unclear how a bird could maintain such high brain concentrations when examinations of the gastrointestinal contents indicate that the individual had not ingested any foodstuffs for 24–48 hours. It is also unclear why the ratios of organochlorine concentrations in the adipose tissue and brain were so inconsistent. The implications of tissue residue concentrations are discussed in greater detail in Section 3.

6 Conclusions

The previous sections examined individual components of the mortality event to determine whether those components support the hypothesis that mortality was the result of organochlorine pesticide poisoning. In this section, all components are assessed jointly in a weight-of-evidence approach.

Fox (1991) proposed an epidemiological approach to determine whether a cause-effect relationship exists between an environmental factor and an observed effect. The approach for evaluating the relationship is based on seven criteria:

- Probability
- Time order
- Strength of association
- Specificity
- Consistency on replication
- Predictive performance
- Coherence.

These seven criteria are assessed below to examine the relationship between avian mortality at Lake Apopka and the likelihood that toxaphene (or another organochlorine pesticide) was the causal agent of that mortality.

Probability refers to a statistically significant relationship between the putative causal factor, organochlorine toxicity, and the observed response, mortality. No control birds were collected during the mortality event, so it is not possible to determine whether body burdens of organochlorine pesticides in birds that died at the NSRA were elevated in relation to levels in unaffected birds. Two white pelicans that died of trauma in January 2000 (case numbers W00-103 and W00-104) were analyzed for pesticide body burdens. However, these data cannot be used for comparison with data from birds that died during the mortality event because the sample size is too small and there is substantial variation in tissue residue levels between the two birds (i.e., two- to twenty-fold difference in concentrations of toxaphene, dieldrin, and 4,4′-DDE). Toxaphene levels in the brains of both birds were undetected, at a detection limit of 2.5 mg/kg. This detection limit, however, exceeds the levels reported in bird brains in the studies by Haseltine et al. (1980) and Niethammer et al. (1984). No prey (fish) species were collected during the mortality event for analysis of organochlorine concentrations, although some individuals were collected subsequent to the avian die-off. Therefore, it is also not possible to develop a statistical relationship between time-courses of organochlorine concentrations in prey and mortality in birds.

Time-order refers to the cause occurring before the effect. Migratory birds arriving at the NSRA were exposed to pesticides in sediments and fish before effects were manifested, but it is not possible to determine the latent period between initial exposure and onset of effects, primarily because it cannot be determined whether the same individuals were present at the NSRA over the entire mortality event, or whether there were arrivals and departures of migrants. For example, white pelicans were on site as of late September 1998, but their population did not peak until early January 1999, when mortality was occurring. Because residence times of individual birds were not tracked, it is not certain whether the birds that were dying had been present since September, and possibly were subjected to the longest period of exposure, or if they were from the population pulse that arrived in early 1999 and were subjected to a shorter exposure period. Resident species, such as great blue herons, were present on the NSRA throughout the mortality event, and as indicated in Table 2-1, some species, such as herons, were present at the NSRA prior to the arrival of pelicans. Although there could have been local movements of resident species from the NSRA to surrounding areas, it is presumable that residents had similar if not longer exposure to organochlorines as migrants. However, mortality in these species did not occur at high frequency until mid-to-late March 1999, at a time when their abundance was declining and pelican mortality was subsiding. As noted in Section 4, exposure modeling indicated that white pelicans experienced higher organochlorine exposure rates than other piscivorous species, which could explain the earlier occurrence of mortality in that species as they may have accumulated a lethal body burden faster. It cannot be determined whether this differential exposure is sufficient to explain different time-courses of mortality between migratory pelicans and resident great blue herons, which both suffered equivalent levels of mortality.

Strength of association refers to the degree to which the putative cause and response coincide in their distribution or the size of the effect produced by the presumptive cause. Food-web model results presented in Section 4 indicate that pelicans were most likely exposed to greater amounts of toxaphene and DDT than were other piscivorous birds, but not dieldrin. Furthermore, the estimated daily exposure to toxaphene at the 95th percentile for pelicans (16.44 mg/kg-day) was below the lowest known avian LD_{50} for this compound. However, steady-state modeling suggests that about 19 percent of the pelican population would exceed the lowest LD_{50} estimate. This value is comparable to the estimated 16 percent mortality rate in pelicans, suggesting that there could be good association between the degree of exposure and effect for that species, but the same does not apply for other piscivorous birds with elevated mortality rates.

Specificity refers to the precision of the association between the cause and the effect (i.e., does the cause lead only to that effect, and is that effect caused only by the putative cause?). Determination of specificity is difficult, because often the same agent can cause different effects through different biological mechanisms, or multiple causal agents can produce similar responses. Review of the signs and symptoms reported in dying birds at the NSRA (such as recorded on the Weatherman videotape) and review of necropsy data identified no consistent cause of death. Some clinical signs and pathological lesions observed in dead birds are consistent with the known effects of organochlorine poisoning, but these symptoms were not noted in all birds, and in some cases were seen only in a small subset of birds. Furthermore, some of the noted pathological symptoms described on necropsy reports are more consistent with effects of other causes, such as disease. The etiological and pathological evidence of the mortality event are not sufficient to identify the specific cause of death of the birds.

Consistency of replication refers to whether the association has been observed repeatedly in different places and at different times. Mass avian mortality events are relatively common. A nationwide summary of avian mortality incidences between 1985 and 1999 listed 147 such events involving 32 species of birds, including 36 events involving white pelicans (USGS 1999). One of the largest mortality events involving white pelicans killed 20,000 birds in the Salton Sea in California in 1996. That mortality event was attributed to botulism linked to tilapia that had been introduced into the sea.

The nationwide summary listed 46 known or suspected causes of avian mortality. The identified causes of mortality included toxicosis caused by several environmental contaminants, but organochlorine pesticides were not listed as a known or suspected causal agent. Keith (1966) reported a mortality event involving white pelicans and other piscivorous birds at Tule Lake in California, which was attributed to toxaphene poisoning. Terminal symptoms observed in some affected birds—including loss of coordination, tremors, and convulsions—appear similar to symptoms observed in birds at the NSRA. However, similar symptoms have also been observed in piscivorous birds during disease outbreaks (e.g., Wobeser et al. 1993).

Predictive performance requires that a hypothesis drawn from an observed association predicts a previously unknown fact or consequence. Analysis of predictive performance is not currently possible for the mortality event at the NSRA, because no follow-up studies have been completed that assess the potential relation between organochlorine pesticides in soils or fish and potential exposure to fish-eating birds.

Coherence refers to whether a putative relationship is theoretically and biologically plausible and consistent with the known facts of the event. Exposure of piscivorous birds to organochlorine pesticides in sediments and fish of the NSRA is plausible, and is supported by body burdens measured in tissues of dead birds, although it is theoretically possible that body burdens were accumulated following exposure to pesticides at other locations. Exposure via the food chain to pesticide doses sufficient to cause acute toxicity in piscivorous birds is also biologically plausible. However, several aspects of the factual evidence are not consistent with the hypothesis that pesticide exposure was responsible for mortality, including:

- The majority of wading bird, shorebird, and waterfowl species present at the NSRA, including many that were highly abundant, were not subject to mortality.
- No mortality was observed in resident birds or early migrants before the peak arrival of pelicans in early January 1999, despite the fact that conditions at that earlier time were similar and could have been sufficient to cause acute mortality if pesticide poisoning were the cause of death.
- Fish are known to be more sensitive to the acutely toxic effects of toxaphene than are birds (Saleh 1991), yet there were no reported mass die-offs of fish at the NSRA.
- Measured concentrations of DDT and metabolites, dieldrin, heptachlor epoxide, and oxychlordane in bird brains were below thresholds for acute mortality.

- Food-web models that are based on available prey data and use conservative exposure assumptions (e.g., 100 percent bioavailability of chemicals) indicate that the maximum daily exposure was below the minimum avian LD_{50}s for toxaphene, dieldrin, and DDT. These results argue against any single organochlorine pesticide being responsible for the avian mortality.

- Visual symptoms and necropsy results reported for dead birds, both within individuals and between individuals, were not consistent with the known effects of toxaphene toxicosis.

The results of the evaluation of epidemiological criteria for organochlorine pesticides being the cause of avian mortality at the NSRA are summarized in Table 6-1. While some criteria are plausible, several key criteria—notably specificity of association of observed causes with the putative effect, and coherence with factual evidence—are not unequivocally supported by available information on the mortality event. Overall, application of epidemiological criteria to the mortality event does not produce strong evidence either supporting or refuting the hypothesis that mortality was a result of exposure to organochlorine pesticides.

7 Management Recommendations

As part of a proposal submitted to SJRWMD, Exponent agreed to provide management recommendations to ensure that the avian mortality noted in the winter of 1998–1999 would not recur. At this juncture, Exponent feels that such recommendations are premature because there is inconclusive evidence that the organochlorine pesticide contamination associated with the soils of the north shore of Lake Apopka was solely responsible for avian mortalities. Furthermore, there are data gaps, particularly with regard to bioavailability, that need to be understood before informed management recommendations can be made. Without such data, the standard assumptions that are regularly used in ecological risk assessment could result in overly conservative predictions that could seriously affect the efficient allocation of management resources. Therefore, Exponent's primary recommendation at this time is to fully characterize transport, fate, and biological uptake of organochlorines at the NSRA. However, as a guide to SJRWMD, Exponent has performed calculations to determine the levels of organochlorine pesticides in soils that could be associated with potential adverse effects in birds. These calculations are described in the following section. Furthermore, until the uptake and distribution within the food web of the organochlorine pesticides within the north shore of Lake Apopka are better understood, it would be prudent not to inundate the flooding blocks, potentially creating a situation similar to that which occurred in the winter of 1998–1999.

8 Derivation of Threshold Pesticide Levels in Soils for Adverse Effects

8.1 Introduction

The food-web model results discussed in earlier sections of this report indicate that exposure to organochlorine pesticides may have been insufficient to elicit acute toxicity in birds present at the NSRA during 1998–1999. Alternatively, analyses of bird tissues indicated that pesticides, particularly toxaphene, had accumulated to high levels. Although the etiology of the observed mortality cannot be conclusively shown to be the result of pesticides, there is evidence that pesticides were a contributing factor. Therefore, Exponent performed risk-based calculations to determine the levels of organochlorine pesticides in muck soils that represent threshold concentrations above which adverse effects could occur for birds using the NSRA. Risk calculations were performed using the white pelican as the representative receptor, given this species' prominent role in the mortality event, even though this species is not a permanent resident at the NSRA. The evaluation focused on three chemicals that are present at high concentrations: toxaphene, dieldrin, and DDT and metabolites (referred to collectively as DDTr).

Threshold soil concentrations that could be associated with potential adverse effects can be determined by back-calculating from a toxicity reference value (TRV) for the chemical that represents either a no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL) in birds. This process involves several steps. First, based on a species' food ingestion rate, the TRV, which represents a daily dose to the bird, is converted into the corresponding body burden in the prey species. Second, the body burden in prey is converted to the corresponding soil concentration by applying an appropriate BSAF for the chemical. These two steps are described below following a description of the derivation of TRVs.

8.2 Derivation of Toxicity Reference Values

TRVs were determined for toxaphene, dieldrin, and DDT from studies reported in the scientific literature. Studies were selected if they examined ecologically relevant endpoints, such as survival, reproduction, and growth. Several studies are discussed for each chemical to reflect inter-species differences in sensitivity that must be considered when attempting to determine threshold concentrations in soil for all avifauna potentially present at the NSRA.

8.2.1 Toxaphene

Keith (1966) performed chronic toxicity tests with young white pelicans to evaluate toxaphene toxicity in relation to a suspected pesticide-induced mortality of pelicans in California. This is the only study of the effects of toxaphene on white pelicans. Flightless young birds were fed for 3 months with fish injected with toxaphene at 10 or 50 mg/kg. The paper provides few details of the experimental design and no detailed presentation of the results. The study was apparently

confounded by the fact that birds did not respond well to captivity with mortality occurring in all groups. However, Keith noted that birds in the 50 mg/kg group died earlier than pelicans in other groups. There was no apparent adverse effect (i.e., premature mortality) on pelicans eating food dosed at 10 mg/kg, which suggests that this was a no-effects level. Using an ingestion rate of 0.33 kg/kg BW-day (Evans and Knopf 1993), the LOAEL (50 mg/kg in food) would correspond to a TRV of 16.7 mg/kg BW-day, whereas the NOAEL (10 mg/kg in food) would correspond to a TRV of 3.3 mg/kg BW-day.

The most comprehensive study of chronic toxaphene toxicity in birds was conducted by Haseltine et al. (1980) who fed black ducks (*Anas rubripes*) 10 or 50 mg/kg toxaphene in the diet for 19 months. After this prolonged exposure, there were no significant differences in adult survival, egg production, fertility, hatchability, eggshell thickness, or growth and survival of young for birds receiving toxaphene relative to controls. There was evidence that young black ducks on both levels of the toxaphene diet had alterations in collagen and mineral content of backbones, but no evidence that this affected survival or growth to mature weight. Thus, 50 mg/kg toxaphene in the diet appears to be a no-effects level for reproductive effects in black ducks. Haseltine et al. (1980) did not report food ingestion rates for black ducks, but congeneric mallard ducks (*A. platyrhynchos*) are reported to consume 0.1 kg food/kg BW-day. Applying this ingestion rate would result in a NOAEL of 5.0 mg/kg-day.

Genelly and Rudd (1956) conducted experiments to assess the effects of toxaphene, DDT, and dieldrin (administered separately) on ring-necked pheasants (*Phasianus colchicus*). Birds were administered toxaphene in the diet at 89 and 243 mg/kg (nominally 100 and 300 mg/kg, respectively) for 8 weeks during egg-laying. Weekly rates of egg production declined significantly only at the higher dose, but survival of young was significantly decreased at both dose levels relative to control birds, which indicates that 89 mg/kg represents an unbounded effects level for reproductive effects in pheasants (i.e., not bounded by a no-effects level). Based on mean daily food consumption rates reported by Genelly and Rudd (1956) (47.4 g/bird/d for this dose group) and assuming an average female body weight of 953 g (Dunning 1993), this dietary concentration would correspond with a TRV of 4.4 mg/kg-day.

8.2.2 Dieldrin

Mendenhall et al. (1983) examined reproductive success in barn owls (*Tyto alba*) that were fed low levels of dieldrin. Only one dose level was used, 0.58 mg/kg (nominally 0.50 mg/kg), which was fed in the diet for 2 years. Breeding success was followed through egg laying, incubation, and natural rearing of the young. Dieldrin was associated with a slight, but significant decrease in eggshell thickness. However, there was no effect on the number of eggs laid or the number of eggs that hatched, or on the mean number of young fledged per pair. Therefore, because dieldrin did not reduce breeding success, the dose level of 0.58 mg/kg in food can be considered an unbounded NOAEL. Using an ingestion rate of 0.13 kg/kg BW-day (Sample et al. 1996), this NOAEL would correspond to a TRV of 0.078 mg/kg BW-day.

Genelly and Rudd (1956) administered dieldrin in the diet to ring-necked pheasants at 22 and 42 mg/kg (nominally 25 and 50 mg/kg, respectively) for 8 weeks during egg laying. Weekly rates of egg production and survival of young significantly decreased at both dose levels relative

to control birds, which indicates that 22 mg/kg represents an unbounded effects level for reproductive effects in pheasants. Based on mean daily food consumption rates reported by Genelly and Rudd (1956) (44.6 g/bird/d for this dose group) and assuming an average female body weight of 953 g (Dunning 1993), this dietary concentration would correspond with a TRV of 1.03 mg/kg-day.

8.2.3 DDT

Anderson et al. (1975) examined reproductive success in brown pelicans (*Pelecanus occidentalis*) breeding on islands offshore of southern California and northwestern Baja California between 1969 and 1974. Fledging rates improved substantially during this time interval, from 0.004 young fledged/nest in 1969 to 0.922 young fledged/nest in 1974. The paper maintains that despite this increase, the fledging rate was still below the rate of 1.2–1.5 young/ nest required to maintain a stationary population, based on the eastern race of the species. The study also measured DDT and DDE levels in eggs and in anchovies, which were claimed to be the primary food source of pelicans. Concentrations of other chemicals were not measured as eggshell thinning effects were attributed wholly to DDE. Concentrations of DDT+DDE in anchovies exhibited a continuous decline from 4.27 mg/kg in 1969 to 0.15 mg/kg in 1974. These concentrations were considered to be representative of the concentrations in the prey of pelicans. U.S. EPA (1995) used these results to infer that 0.15 mg/kg DDT and metabolites in food (the level measured in 1974) constitutes an unbounded LOAEL for reproductive effects in pelicans, as the fledging rate in 1974 was insufficient to maintain a stable population. Using a body weight of 3.5 kg (Dunning 1993) and a dry weight food ingestion rate of 0.04 kg/kg BW-day (Nagy 1987), this concentration in food equates to a TRV of 0.027 mg/kg-day.

There are several problems with use of the study by Anderson et al. (1975) to derive an avian TRV for DDT. First, because the study did not assess the potential effects of other contaminants that may have been present in food and eggs, it is not possible to determine if the decrease in reproductive success was solely due to the egg-shell thinning-induced effects of DDE, or whether other contaminants may have contributed. Second, there appeared to be an increase in anchovy populations concurrent with the increase in fledging success, as fish biomass, expressed as thousands of schools per census in a fixed area, increased from a low of 70 in 1970 to 355 in 1974. Therefore, it is not clear whether the changes in nestling fledging success are related to changes in chemical concentrations or, more simply, to changes in food abundance. These factors related to the use of an uncontrolled field study for deriving a TRV make it highly uncertain whether the DDT concentration measured in anchovies represents the true effects level. If other chemicals or low food abundance contributed to decreased fledging success, then the actual effects concentration could be much higher. Nevertheless, the TRV determined from this study is used to represent a conservative level for potential adverse effects.

Genelly and Rudd (1956) administered DDT in the diet to ring-necked pheasants at 90 and 355 mg/kg (nominally 100 and 400 mg/kg, respectively) for 8 weeks during egg laying. Weekly rates of egg production for both dose groups were statistically indistinguishable from controls, but survival of young was significantly lower at both dose levels. The decrease in chick survival indicates that 90 mg/kg represents an unbounded effects level for reproductive effects in pheasants. Based on mean daily food consumption rates reported by Genelly and Rudd

(57.2 g/bird/d for this dose group) and assuming an average female body weight of 953 g (Dunning 1993), this dietary concentration would correspond with a TRV of 5.4 mg/kg-day.

In an examination of the toxicity of DDT to avian species, U.S. EPA (1995) reviewed a number of studies, and reported LOAELs ranging from 0.027 mg/kg-day to 5.8 mg/kg-day. The two studies discussed above nearly bracket this reported range of TRVs for DDT in birds, and thus capture the known sensitivity of birds to the chronic effects of DDT.

8.3 Conversion of TRVs to Body Burdens in Prey

The TRV represents a dose ingested by a bird on a daily basis, but this dose must be converted to a corresponding concentration in their prey (i.e., fish). This is accomplished by dividing the TRV by the food ingestion rate. As indicated in earlier sections of this report on food-web modeling, white pelicans are estimated to have a daily food ingestion rate of 0.33 kg food (wet weight)/kg BW-day. Using this value, wet-weight concentrations in food can be calculated, as presented in Table 8-1. Fish concentrations also need to be lipid normalized because BSAFs are calculated on a lipid-normalized basis. Lipid normalization was performed using the estimate that prey fish are 3.6 percent lipid, based on fish collected at the NSRA.

8.4 BSAF Determination

BSAF values can be obtained from the scientific literature, but published values can vary greatly, and are highly dependent on the conditions of the study, such as chemical bioavailability, exposure pathways, and the duration of exposure of prey species to the contaminated sediments. This uncertainty can be reduced if data are available to allow calculation of a site-specific BSAF. This approach is possible at Lake Apopka because concurrent fish and soil chemistry data were collected from four areas of the NSRA during August 1999. Samples were collected from Duda West, Flow-way Cell G, Zellwood East, and Zellwood South.

Soil chemistry data are presented in Table 8-2; fish tissue data are presented in Table 8-3 on a wet-weight basis and in Table 8-4 on a lipid-normalized basis. Undetected compounds are reported at half their detection limit. Concentrations are reported separately for DDT, DDD, and DDE, and summed as DDTr. BSAF values presented below are calculated using DDTr concentrations in soils and fish. Gar were also collected from Zellwood East, but this species was not included in the database because collected individuals are too long (> 40 cm) to constitute prey of wading birds. Additionally, this species was excluded from food-web models, as described earlier in this report.

The BSAF values (Table 8-5) are calculated by dividing the lipid-normalized tissue data for a fish by the average TOC-normalized soil concentration in the area where that fish was collected. As shown in Table 8-5, BSAF values for tilapia collected at Zellwood East were much higher than BSAF values for fish collected from other locations. The higher BSAF values at Zellwood East appear to be primarily the result of the lower lipid levels in tilapia collected from this location relative to other areas of the NSRA. In a previous risk assessment performed for the

Lake Apopka Muck Farm Wetlands Restoration, ATRA (1997) calculated BSAF values of 1.1–3.3 for DDT and toxaphene based on lipid-normalized fish tissue concentrations and TOC-normalized sediment concentrations. These values are similar to those calculated in this report excluding Zellwood East. The BSAF values for fish from Zellwood East are inconsistent with values calculated from other locations of the NSRA. The lower lipid content of these fish suggest that they were stressed, possibly by factors such as decreased food abundance or overcrowding. Therefore, because BSAF values for these fish do not appear to be representative of fish elsewhere on the NSRA, calculations of sediment thresholds are performed on all fish BSAF data except Zellwood East, as reported in Table 8-5.

8.5 Calculation of Soil Threshold Concentrations

The BSAF is calculated as the lipid-normalized fish concentration divided by the TOC-normalized soil concentration. Therefore, a threshold muck soil concentration for potential adverse effects can be determined by dividing the threshold concentration in fish by the BSAF value. The resulting value is then multiplied by the proportion of TOC in the soil to derive the threshold soil concentration. The following example describes the process for toxaphene. Based on the study by Haseltine et al. (1980), as described above, the lipid-normalized toxaphene concentration in fish that represents a no-effects threshold for chronic effects in birds is 421 mg/kg. The BSAF for toxaphene, based on NSRA-specific data is 1.4, therefore a threshold sediment concentration is 421/1.4, or 301 mg/kg on a TOC-normalized basis. If there is 20 percent TOC in soil, then the non-TOC normalized equivalent concentration is 301×0.2, or 60.1 mg/kg. The same procedure would be used to calculate soil concentrations for other chemicals. As indicated by this equation, the threshold toxaphene concentration depends on the TOC concentration. At 30 percent TOC in soil, the threshold toxaphene concentration would be 90.2 mg/kg. The range of threshold soil concentrations for toxaphene, dieldrin, and DDTr, as based on different combinations of TRVs and TOC levels in soil, is presented in Figure 8-1.

8.6 Uncertainty Evaluation

The results indicate the difficulty in attempting to derive a single value that represents a threshold soil concentration for any of these chemicals (i.e., a concentration that would not result in adverse effects in birds of any species chronically exposed to these soils or to soil-associated prey). There are three major sources of uncertainty that affect the calculated threshold concentrations, namely the TRVs, the calculated BSAF values, and the organic carbon content of soil, each of which is discussed below.

With the exception of the study by Keith (1966) that evaluated effects of toxaphene in white pelicans, there are no TRVs available for the avian species present at the NSRA. Therefore, TRVs had to be extrapolated from studies conducted with other species. This extrapolation introduces uncertainty because the relative sensitivities to adverse effects of the chemical for the test species and the species present at the NSRA are unknown. If species present at the NSRA are more sensitive, then the threshold soil concentrations would be lower than reported here, whereas if these species are less sensitive, then the threshold soil concentrations would be higher. There is no effective means of quantifying this uncertainty other than by applying

arbitrarily selected uncertainty factors based on the degree of taxonomic divergence between the test species and the species present at the NSRA.

Additional uncertainty is introduced when TRVs are unbounded (i.e., only a LOAEL or a NOAEL can be determined from a study, but not both). For example, with dieldrin, the study by Mendenhall et al. (1983) provides only a NOAEL, so the soil concentration at which effects would start to occur cannot be determined. Conversely, the study by Genelly and Rudd (1956) does not permit derivation of a no-effects threshold. The true effects threshold may fall somewhere in the interval bounded by these two studies, provided the test species used in the two studies have similar sensitivities. The uncertainty over the true effects threshold is greater for DDTr, because both studies used to derive TRVs for this compound did not determine a no-effects level, and therefore, a corresponding no-effects concentration in soil cannot be determined.

The BSAF values used to relate fish concentrations to soil concentrations were calculated as the average of BSAFs for all suitable prey species from all areas of the NSRA, excluding Zellwood East. There are several potential sources of uncertainty with this approach. First, the BSAF concept works on the assumption that concentrations in soils and in biota are in steady-state equilibrium. This assumption is likely not met in all areas of the NSRA because of the short duration of contact between fish and inundated soils in the flooding blocks. Second, the assumption is made that all fish species in all areas constitute potential prey for a receptor, which could mask species-specific or area-specific differences. For example, the average BSAF for toxaphene for all fish excluding those from Zellwood East is 1.4. However, for bullhead inhabiting Duda West, the average BSAF is 2.5, or approximately twice as high. If a bird foraged exclusively on bullhead from this location of the NSRA, then the threshold soil concentration would be approximately half the value estimated using the site-wide BSAF.

The determination of a threshold soil concentration is also influenced by the TOC content of soils. As indicated on Figure 8-1, as the TOC content of the soil increases, there is a corresponding increase in the soil concentration that is associated with the potential for adverse effects. The proportion of TOC in soils is not consistent over the entire NSRA; therefore, the threshold soil concentration will also vary over the site. This represents an additional uncertainty when attempting to derive a numeric criterion.

8.7 Conclusions

Concentrations of toxaphene, dieldrin, and DDTr in muck soils of the NSRA that may be indicative of a threshold response associated with either a NOAEL or LOAEL for chronic effects in birds as a result of trophic transfer were calculated. Because of the uncertainty primarily associated with determining appropriate TRVs for these compounds, it is not possible to provide a definitive numeric value. The TRVs that are determined from studies in the scientific literature are used as the basis for expressing the known sensitivity of birds to the adverse effects of these chemicals, and to calculate a range of soil concentrations that would represent a threshold for potential adverse effects in birds resident at the NSRA, provided that their sensitivity to the effects of these chemicals is similar to that of the species used to derive the TRVs.

9 References

Allen, A.W. 1985. Habitat suitability index models: American coot. U.S. Fish and Wildlife Service Biological Report 82(10.115). 17 pp.

Anderson, D.W., J.R. Jehl, Jr., R.W. Risebrough, L.A. Woods, Jr., L.R. DeWeese, and W.G. Edgecombe. 1975. Brown pelicans: Improved reproduction of the southern California coast. Science 190:806–808.

ATRA. 1997. Environmental risk assessment of a Lake Apopka muck farm wetlands restoration. Prepared for St. Johns River Water Management District by ATRA, Inc.

Behle, W.H. 1958. The bird life of Great Salt Lake. University of Utah Press, Salt Lake City, UT.

Beyer, W.N., E.E. Connor, and S. Gerould. 1994. Estimates of soil ingestion by wildlife. J. Wildl. Manage. 58:375–382.

Biessmann, A., A. Mohammed, and P. Slanina. 1983. Studies on the tissue disposition and fate of [14C]toxaphene in Japanese quail. Toxicology 28:155–165.

Blus, L.J. 1996. DDT, DDD, and DDE in birds. pp. 49–71. In: Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds). Lewis Publishers, Boca Raton, FL.

Bogiatto, II, R.J. 1990. Fall and winter food habits of American coots in the northern Sacramento Valley, California. Calif. Fish Game 76(4):211–215.

Butler, R.W. 1992. Great blue heron. Birds of North America, No. 25. American Ornithologists' Union.

Campo, J.J., B.C. Thompson, J.C. Barron, R.C. Telfair, P. Durocher, and S. Gutreuter. 1993. Diet of double-crested cormorants wintering in Texas. J. Field Ornithol. 64(2):135–144.

Cooper, J.M. 1994. Least sandpiper (*Calidris minutilla*). In: Birds of North America, No. 115. A. Poole and F. Gill (eds). Academy of Natural Sciences; Washington, DC, and American Ornithologists' Union.

Coulter, M.C., J.A. Rodgers, J.C. Ogden, and F.C. Depkin. 1999. Wood stork (*Mycteria Americana*). In: The Birds of North America, No. 409. A. Poole and F. Gill (eds). The Birds of North America, Inc., Philadelphia, PA.

del Hoyo, J., A. Elliott, J. Sargatal, and A. Collar (eds). 1992. Handbook of the birds of the world. Lynx Edicions, Barcelona.

Derby, C.E., and J.R. Lovvorn. 1997. Predation of fish by cormorants and pelicans in a cold-water river: a field and modeling study. Can. J. Fish. Aquat. Sci. 54:1480–1493.

Dunning, Jr., J.B. 1993. CRC handbook of avian body masses. CRC Press.

Ecobichon, D.J. 1996. Toxic effects of pesticides. pp. 643–689. In: Casarett & Doull's Toxicology, the Basis Science of Poisons. C.D. Klaassen (ed). Fifth Edition. McGraw-Hill, New York.

Evans, R.M., and F.L. Knopf. 1993. American white pelican (*Pelecanus erythrorhyncos*). In: The Birds of North America, No. 57. A. Poole and F. Gill (eds). The Birds of North America, Inc., Philadelphia, PA.

Fox, G.A. 1991. Practical causal inference for ecoepidemiologists. J. Toxicol. Environ. Health 33:359–373.

Frederick, P.C., M.G. Spalding, M.S. Sepulveda, G.E. Williams, L. Nico, and R. Robins. 1999. Exposure of great egret (*Ardea albus*) nestlings to mercury through diet in the Everglades ecosystem. Environ. Toxicol. Chem. 18(9):1940–1947.

Friend, M. 1987. Field guide to wildlife diseases. Volume 1. General field procedures and diseases of migratory birds. U.S. Fish and Wildlife Service Resource Publication 167, Washington, DC.

Genelly, R.E., and R.L. Rudd. 1956. Effects of DDT, toxaphene, and dieldrin on pheasant reproduction. Auk 73:529–539.

Haseltine, S.D., M.T. Finley, and E. Cromartie. 1980. Reproduction and residue accumulation in black ducks fed toxaphene. Arch. Environ. Contam. Toxicol. 9:461–471.

Hodge, H.C., A.M. Boyce, W.B. Deichmann, and H.F. Kraybill. 1967. Toxicology and no-effect levels of aldrin and dieldrin. Toxicol. Appl. Pharmacol. 10:613–675.

Hoffman, R.D. 1978. The diets of herons and egrets in southwestern Lake Erie. Wading birds. National Audubon Society Research Report 7: 365–369.

Hudson, R.H., R.K. Tucker, and M.A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. Second Edition. Resource Publication 153. U.S. Department of the Interior, Fish and Wildlife Service, Washington, DC.

Kahl, Jr., M.P. 1964. Food ecology of the wood stork (*Mycteria americana*) in Florida. Ecol. Monogr. 34:97–117.

Keith, J.O. 1966. Insecticide contaminations in wetland habitats and their effects on fish-eating birds. J. Appl. Ecol. 71–85.

Kuiken, T., F.A. Leighton, G. Wobeser, K.L. Danesik, J. Riva, and R.A. Heckert. 1998. An epidemic of Newcastle disease in double-crested cormorants from Saskatchewan. J. Wildl. Dis. 34(3):457–471.

Kushlan, J.A. 1978. Feeding ecology of wading birds. Wading birds. National Audubon Society Research Report 7:249–297.

Matsumura, F. 1985. Toxicology of insecticides. Plenum Press, New York, NY.

McEwen, F.L., and G.R. Stephenson. 1979. The use and significance of pesticides in the environment. John Wiley & Sons, New York, NY.

McMahon, B.F., and R.M. Evans. 1992. Foraging strategies of American white pelicans. Behaviour 120:69–89.

Mendenhall, V.M., E.E. Klaas, and A.R. McLane. 1983. Breeding success of barn owls (*Tyto alba*) fed low levels of DDE and dieldrin. Arch. Environ. Contam. Toxicol. 12:235-240.

Meteyer, C.U., D.E. Docherty, L.C. Glaser, J.C. Franson, D.A. Senne, and R. Duncan. 1997. Diagnostic findings in the 1992 epidemic of neurotropic velogenic Newcastle disease in double-crested cormorants from the upper midwestern United States. Avian Dis. 41:171–180.

Mount, M.E., V. Traffas, R.J. Milleret, and F.W. Oehme. 1980. An unusual occurrence of toxaphene poisoning in swine. J. Am. Vet. Med. Assoc. 177(5):445–447.

Nagy, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Monogr. 57:111–128.

Niethammer, K.R., T.S. Baskett, and D.H. White. 1984. Organochlorine residues in three heron species as related to diet and age. Bull. Environ. Contam. Toxicol. 33:491–498.

Ogden, J.C., J.A. Kushlan, and J.T. Tilmant. 1976. Prey selectivity of the wood stork. Condor 76:324–330.

Palmer, R.S. 1962. Handbook of North American birds. Volume 1. Yale University Press, New Haven, CT.

Post, G. 1951. Effects of toxaphene and chlordane on certain game birds. J. Wildl. Manage. 15(4):381–386. (not seen, as cited in Cooper 1994)

Post, W., and M.M. Browne. 1976. Length of stay and weights of inland migrating shorebirds. Bird-Banding 47:333–339. (not seen, as cited in Cooper 1994)

Robbins, C.S., B. Bruun, and H.S. Zim. 1983. A guide to field identification: Birds of North America. Golden Press, New York.

Saleh, M.A. 1991. Toxaphene: chemistry, biochemistry, toxicity and environmental fate. Rev. Environ. Contam. Toxicol. 118:1–85.

Sample, B.E., D.M. Opresko, and G.W. Suter II. 1996. Toxicological benchmarks for wildlife: 1996 revision. Risk Assessment Program, Health Science Research Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

SJRWMD. 1999a. Chronology of events surrounding the avian mortality episode at the Lake Apopka North Shore Restoration Area—September 1998 to March 1999. St. Johns River Water Management District, Palatka, FL.

SJRWMD. 1999b. Water levels on the North Shore Restoration Area at Lake Apopka during fall 1998 and winter 1999. St. Johns River Water Management District, Palatka, FL.

SJRWMD. 2000. St. John's River Water Management District, chemical analysis data on website at http://sjr.state.fl.us/news/TAG_info/data_updates/. Last accessed May 2000.

SJRWMD. 2003. Review comments for: Analysis of avian mortality at the North Shore Restoration Area of Lake Apopka in 1998–1999. St. Johns River Water Management District, Palatka, FL.

Smith, J.P. 1997. An energy-circuit population model for Great Egrets (*Ardea alba*) at Lake Okeechobee, Florida, U.S.A. Ecol. Model. 97:1–21.

Stickel, W.H., L.F. Stickel, and J.W. Spann. 1969. Tissue residues of dieldrin in relation to mortality in birds and mammals. pp. 174–204. In: Chemical Fallout. M.W. Morton and G.G. Berg (eds). Charles C. Thomas, Springfield, IL.

Stickel, W.H., L.F. Stickel, and F.B. Coon. 1970. DDE and DDD residues correlated with mortality of experimental birds. pp. 287–294. In: Pesticides Symposia, Seventh Int. Am. Conf. Toxicol. Occup. Med. W.B. Deichmann (ed). Helios and Assoc., Miami, FL.

Stites, D. 2000. Personal communication (conversation with C. Mackay and N. Gard, Exponent, Bellevue, WA, on January 22, 2000, regarding videotaped incident of white pelican mortality). SJRWMD, Palatka, FL.

U.S. EPA. 1993. Wildlife exposure factors handbook. EPA/600/R-93/187a. U.S. Environmental Protection Agency, Washington, DC.

U.S. EPA. 1995. Great Lakes water quality initiative criteria documents for the protection of wildlife—DDT, mercury, 2,3,7,8-TCDD, PCBs. EPA 820-B-95-008, Office of Water, U.S. Environmental Protection Agency, Washington, DC.

U.S. FWS. 2001. Data relating to legal case USFWS INV #403000457: Errata sheet for submissions. NWHC Case #'s 16138, 16188 and 16196. Dr. Carol U. Meteyer, March 15, 2001. U.S. Fish and Wildlife Service, Washington, DC.

Udvardy, M.D.F. 1988. The Audubon Society field guide to North American birds, Western region. Alfred A Knopf, Inc., New York. 852 pp.

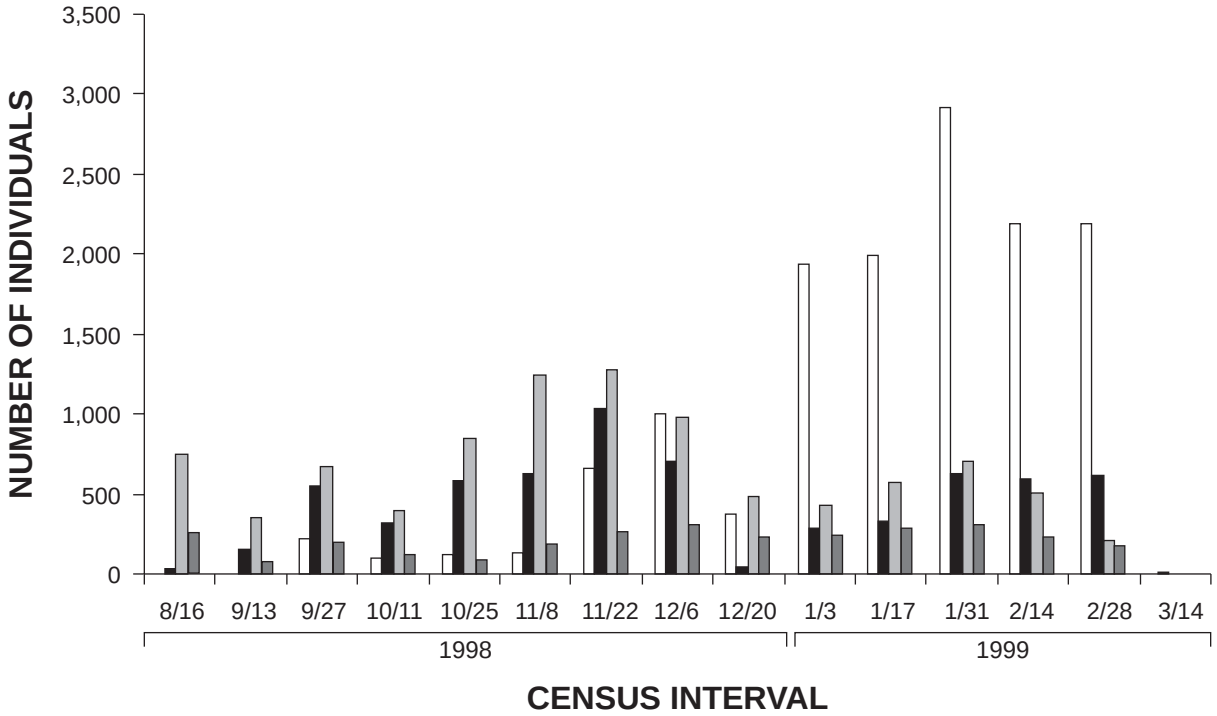
USGS. 1999. USGS NWHS additional mortality database info.xls web page http://sjr.state.fl.us/news/TAG_info/ERG_info/data/, accessed on November 19, 1999. U.S. Geological Survey.

Verschueren, K. 1983. Handbook of environmental data on organic chemicals. Van Nostrand Reinhold, New York.

Willard, D.E. 1977. The feeding ecology and behavior of five species of herons in southeastern New Jersey. Condor 79:462–470.

Wobeser, G., F.A. Leighton, R. Norman, D.J. Myers, D. Onderka, M.J. Pybus, J.L. Neufeld, G.A. Fox, and D.J. Alexander. 1993. Newcastle disease in wild water birds in western Canada, 1990. Can. Vet. J. 34:353–359.

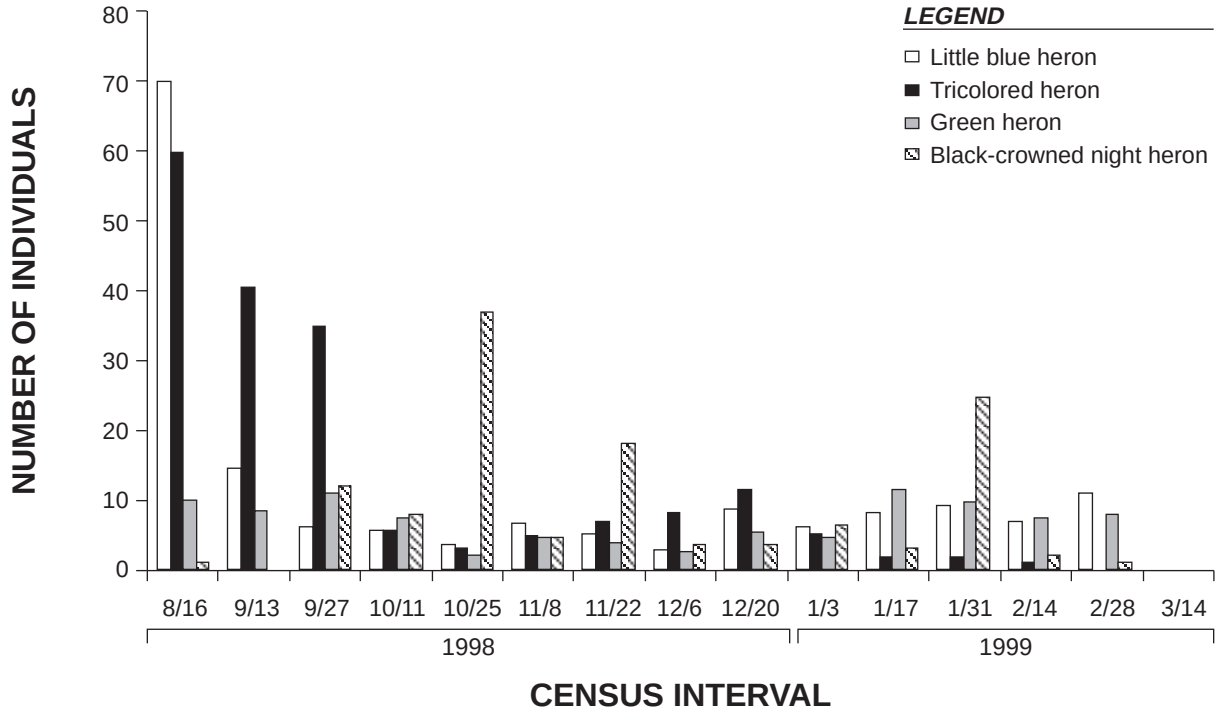
Figures



LEGEND

- American white pelican
- Wood stork
- Great egret
- Great blue heron

Figure 2-1. Abundance of species with highest recorded mortality rates at the North Shore Restoration Area of Lake Apopka



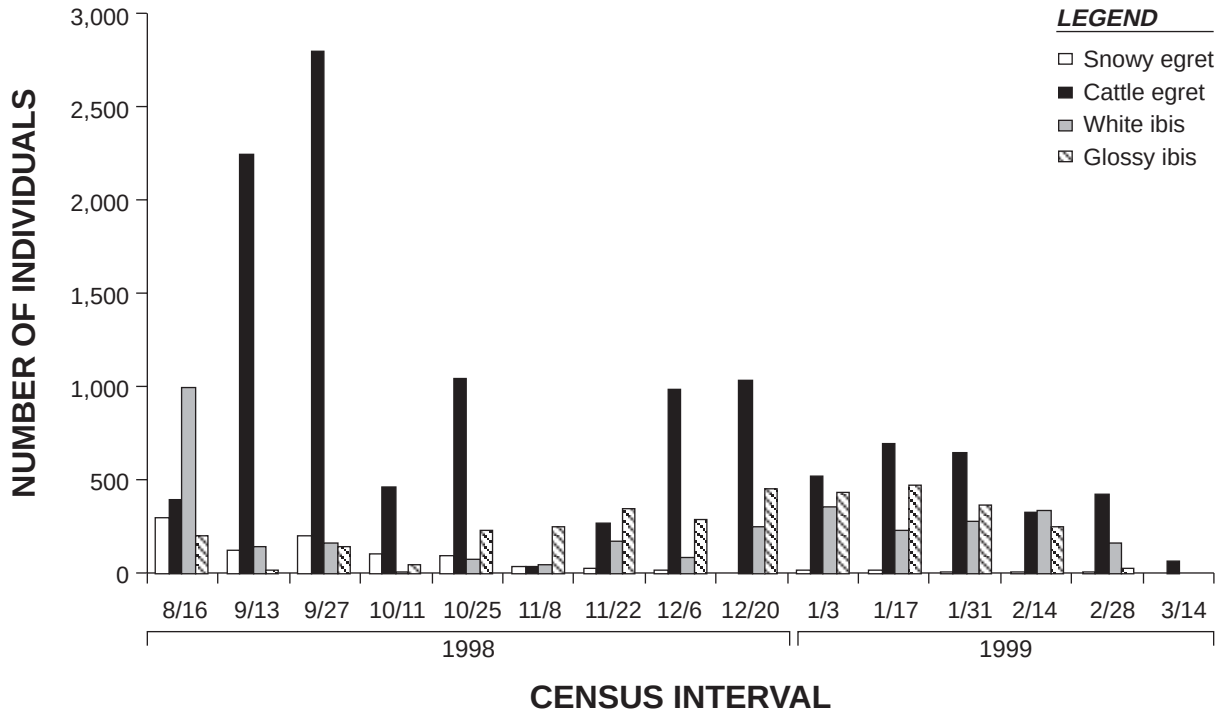
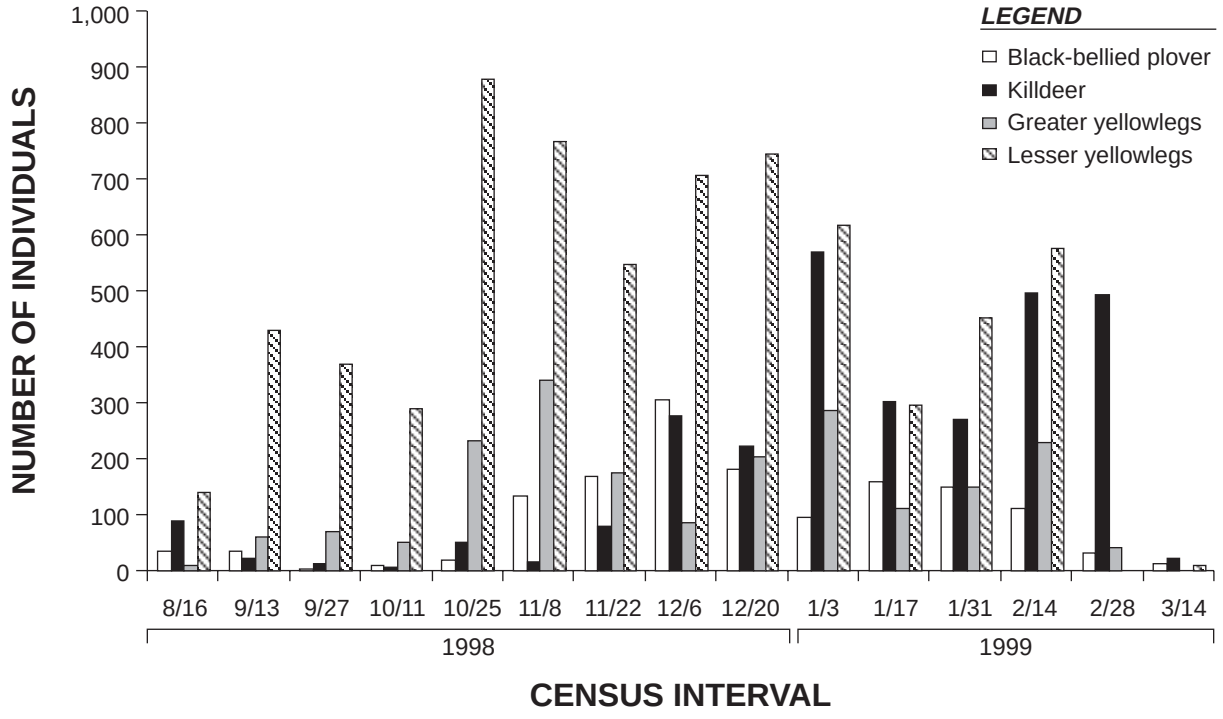


Figure 2-2. Wading birds abundance at the North Shore Restoration Area of Lake Apopka



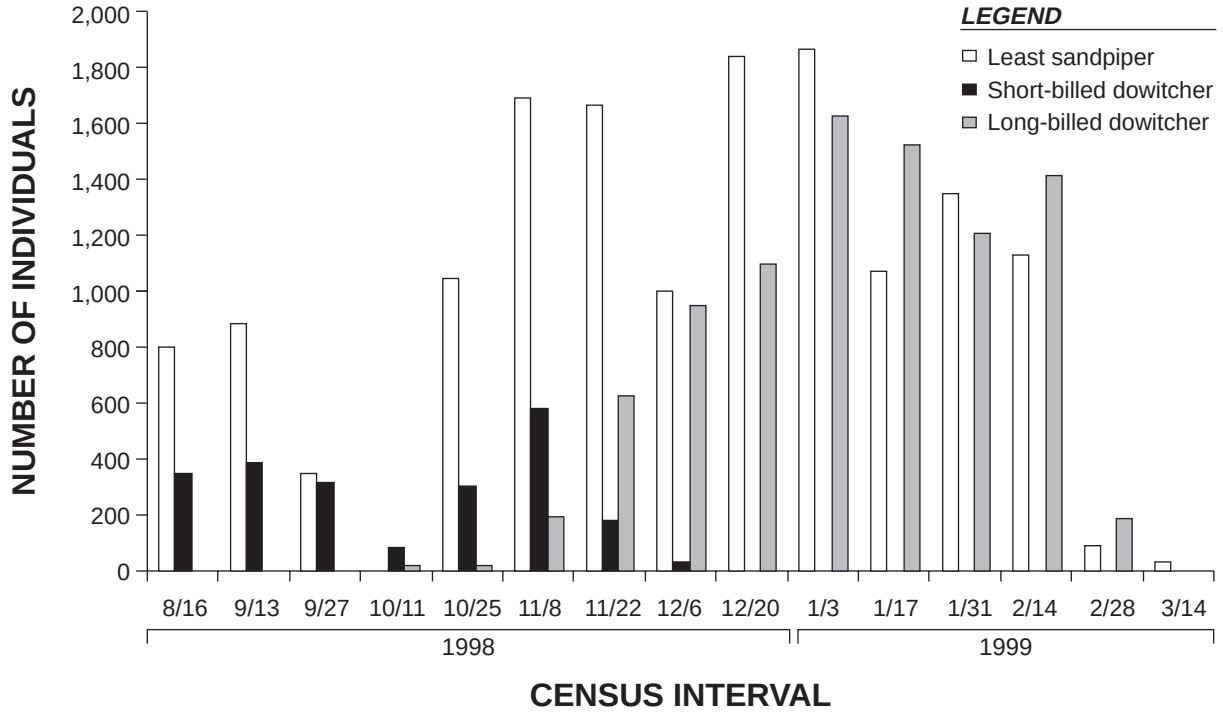
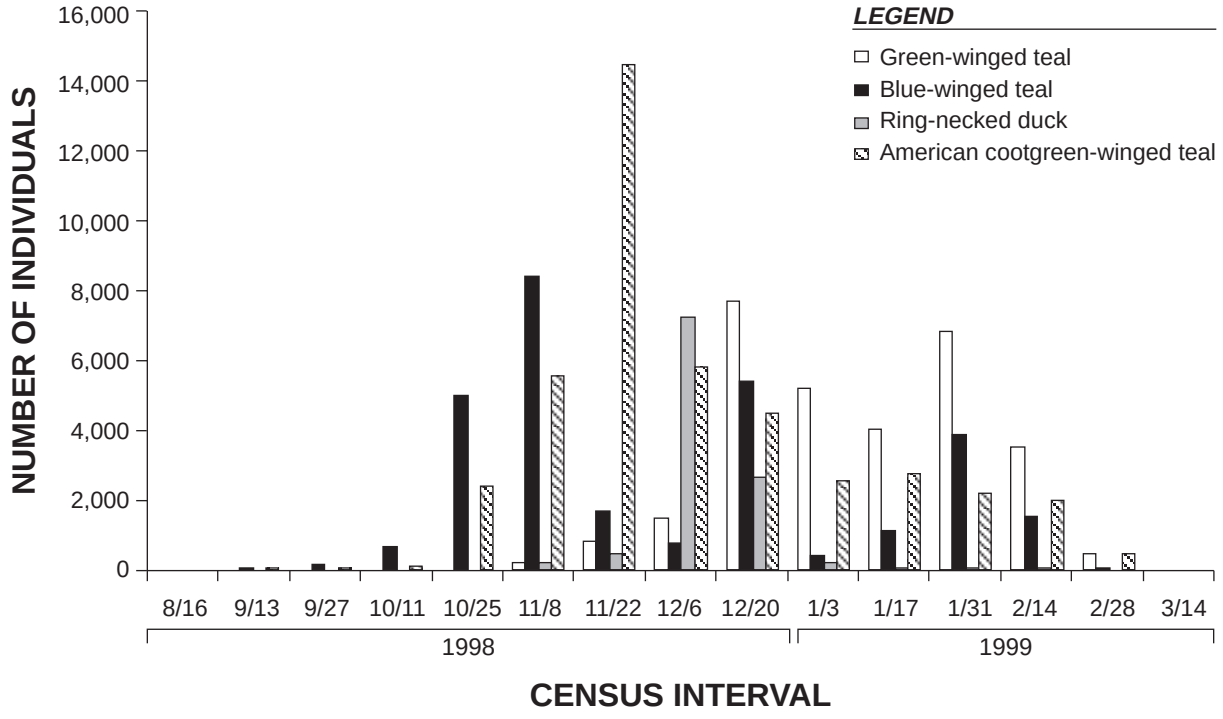


Figure 2-3. Shorebird abundance at the North Shore Restoration Area of Lake Apopka



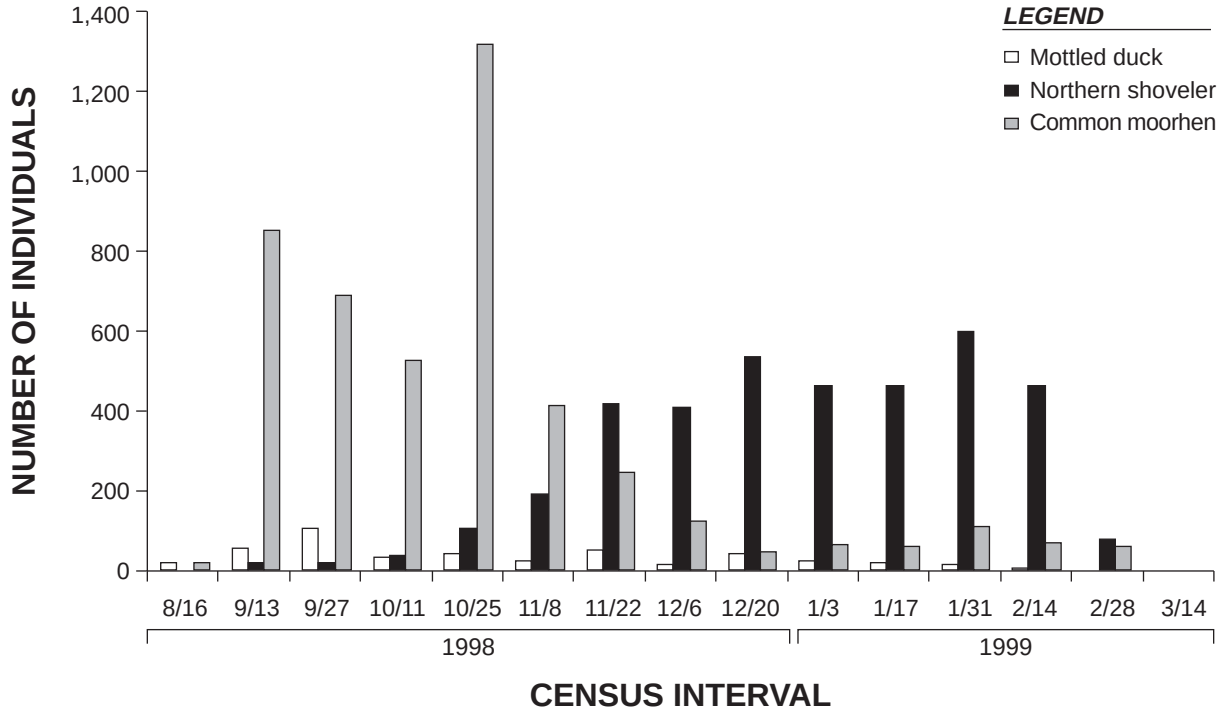
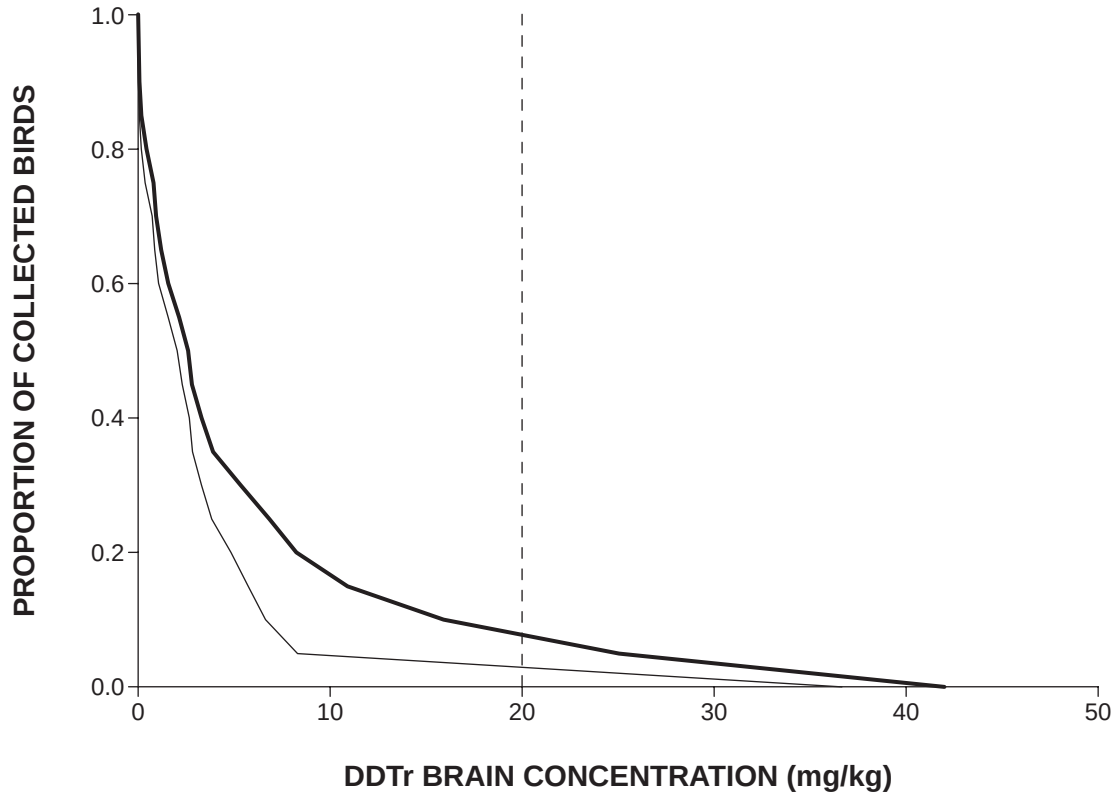


Figure 2-4. Waterfowl abundance at the North Shore Restoration Area of Lake Apopka

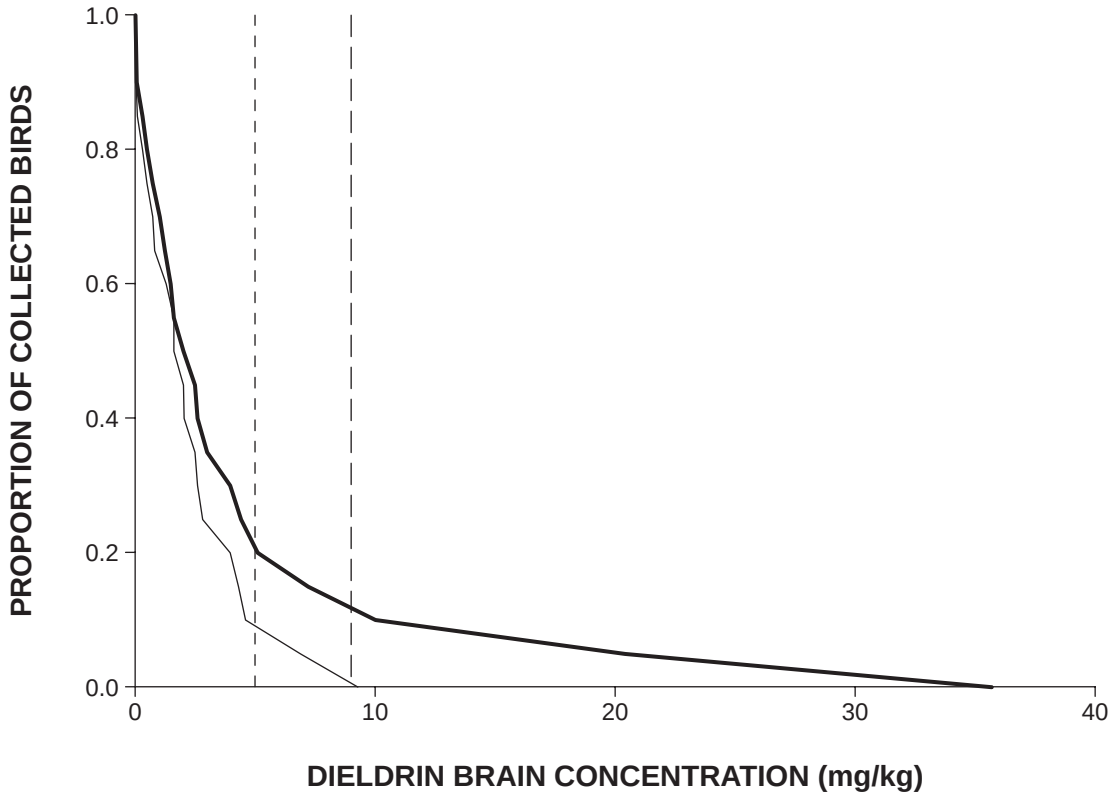


LEGEND

American white pelican
All species
DDTr threshold

Note: Vertical line represents the estimated toxicity threshold concentration.

Figure 3-1. Reverse cumulative distribution of reported DDTr concentrations in brain tissues from necropsied birds



LEGEND

American white pelican
All species
Upper dieldrin threshold
Lower dieldrin threshold

Note: Vertical lines represent the upper and lower estimated toxicity threshold concentrations.

Figure 3-2. Reverse cumulative distribution of reported dieldrin concentrations in brain tissue from necropsied birds

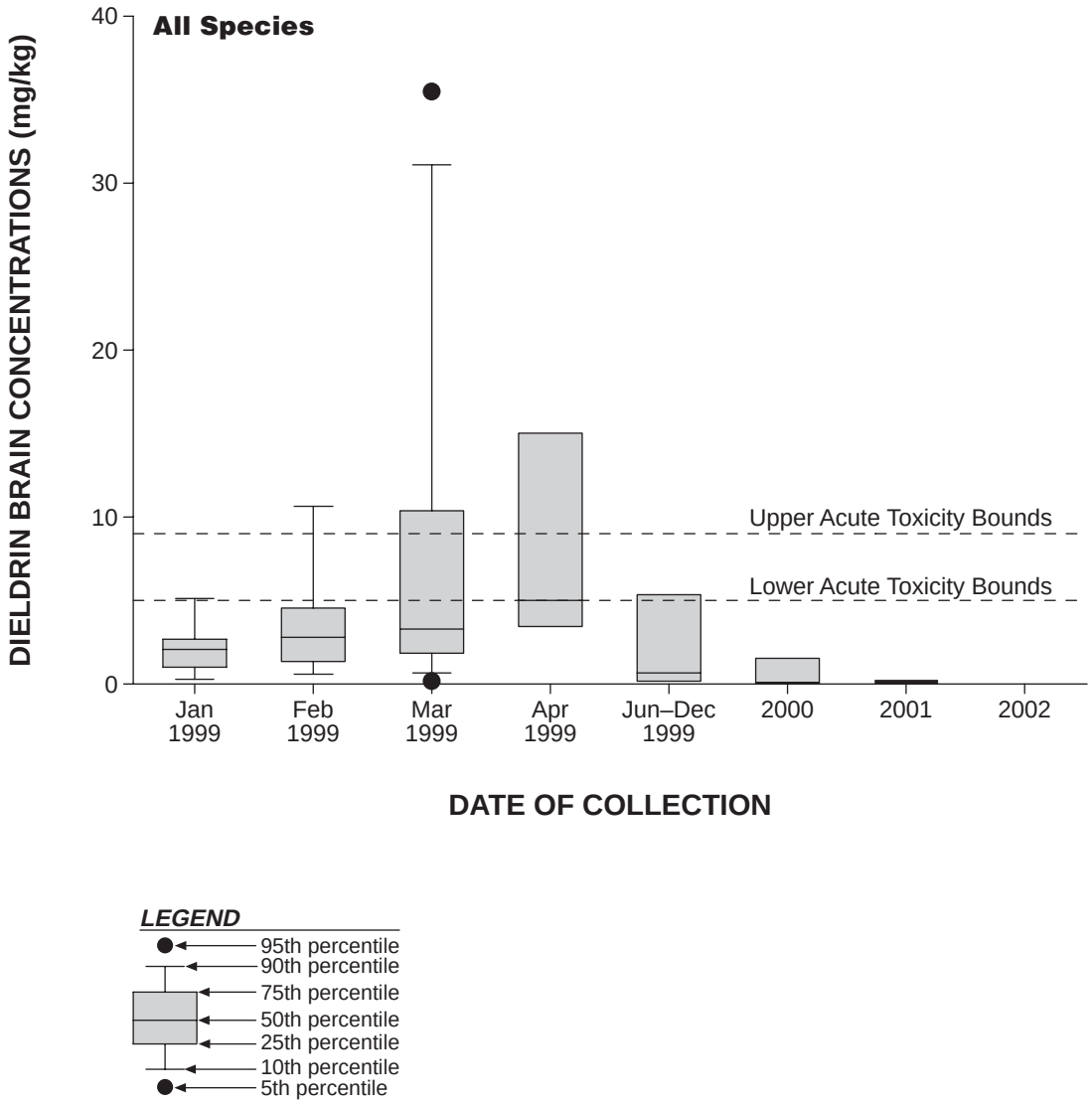


Figure 3-3. Temporal distribution of observed dieldrin concentrations in necropsied brain tissue from all avian species

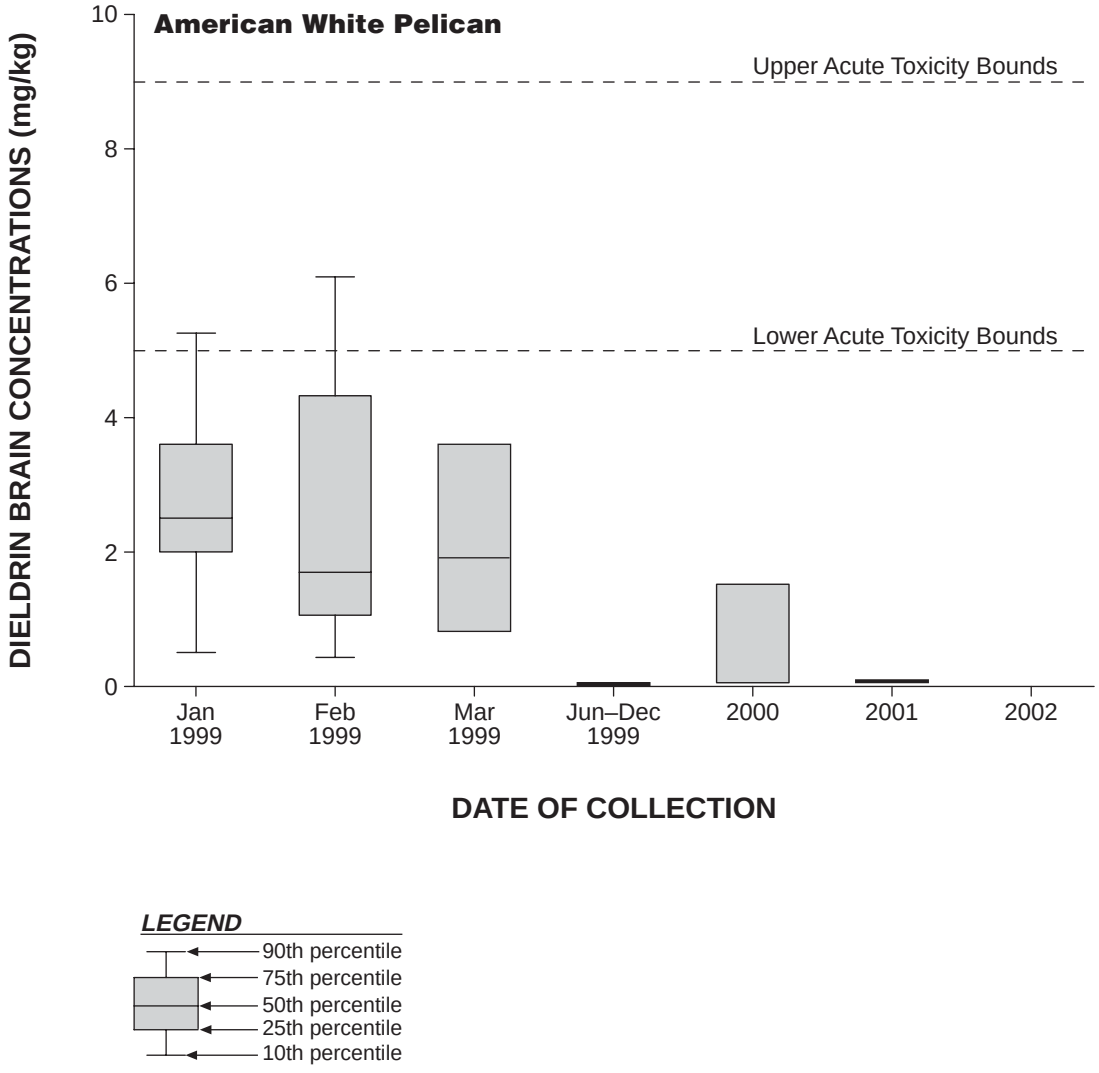
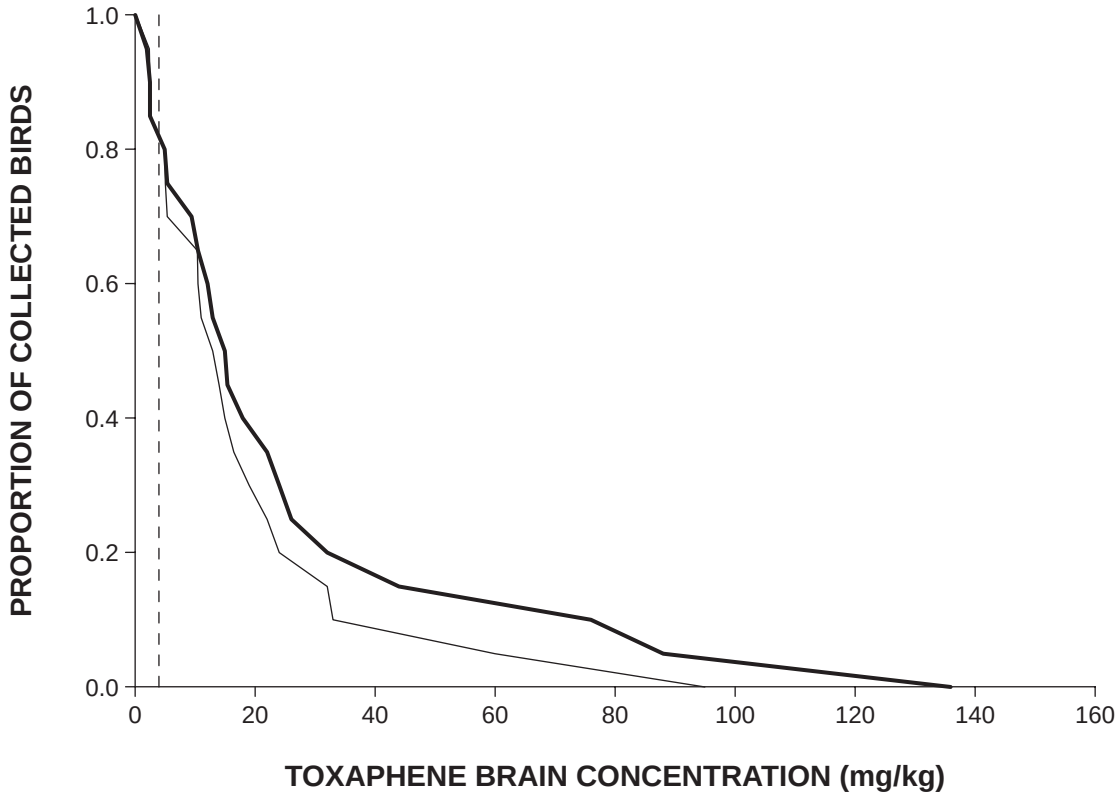


Figure 3-4. Temporal distribution of observed dieldrin concentrations in brain tissue of American white pelicans

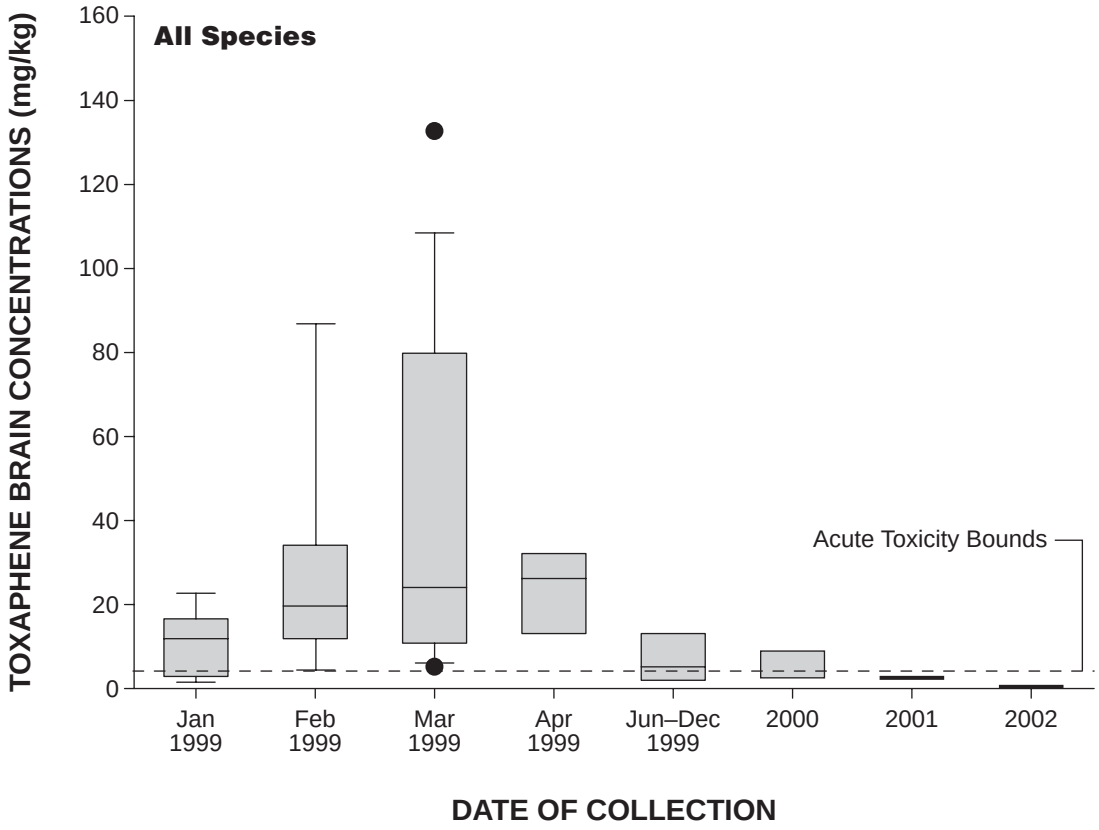


LEGEND

American white pelican
All species
Toxaphene threshold

Note: Vertical line represents the estimated toxicity threshold concentration.

Figure 3-5. Reverse cumulative distribution of reported toxaphene concentrations in brain tissue from necropsied birds



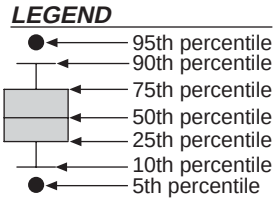


Figure 3-6. Temporal distribution of observed toxaphene concentrations in necropsied brain tissue from all species

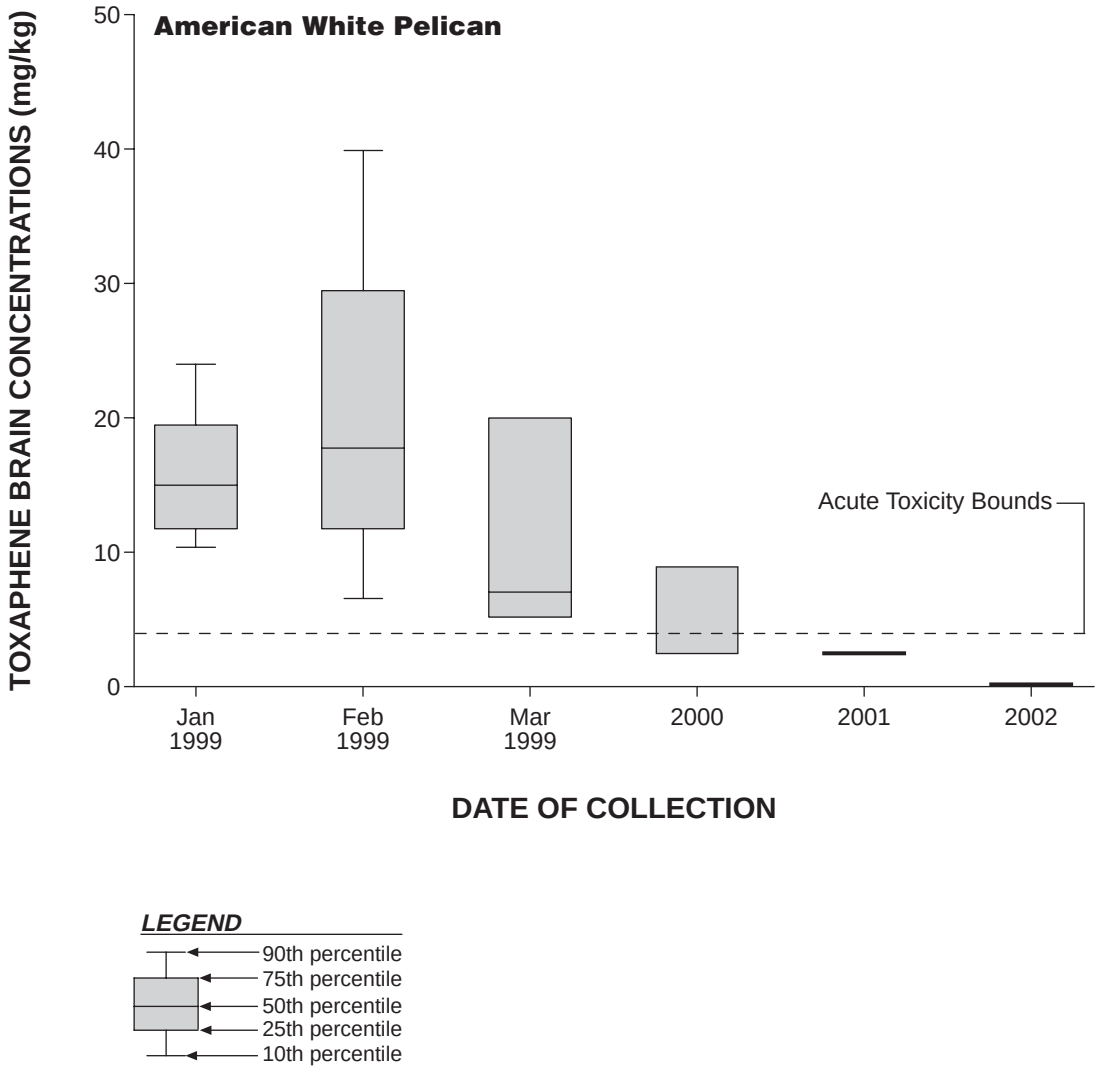


Figure 3-7. Temporal distribution of observed toxaphene concentrations in necropsied brains from American white pelicans

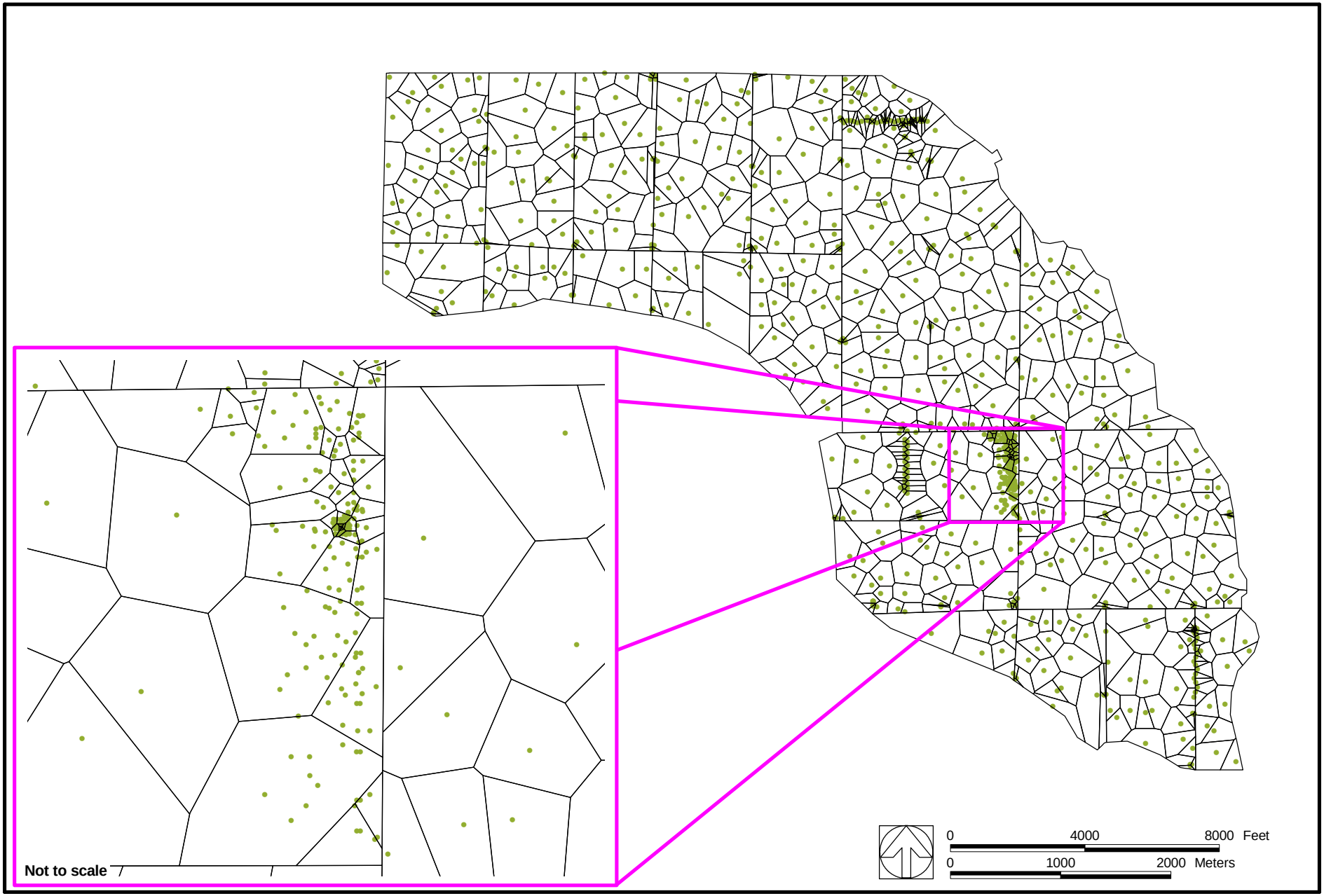


Figure 4-1. Thiessen polygon analysis of the North Shore Restoration Area of Lake Apopka used to develop the exposure distributions for sediment and prey items

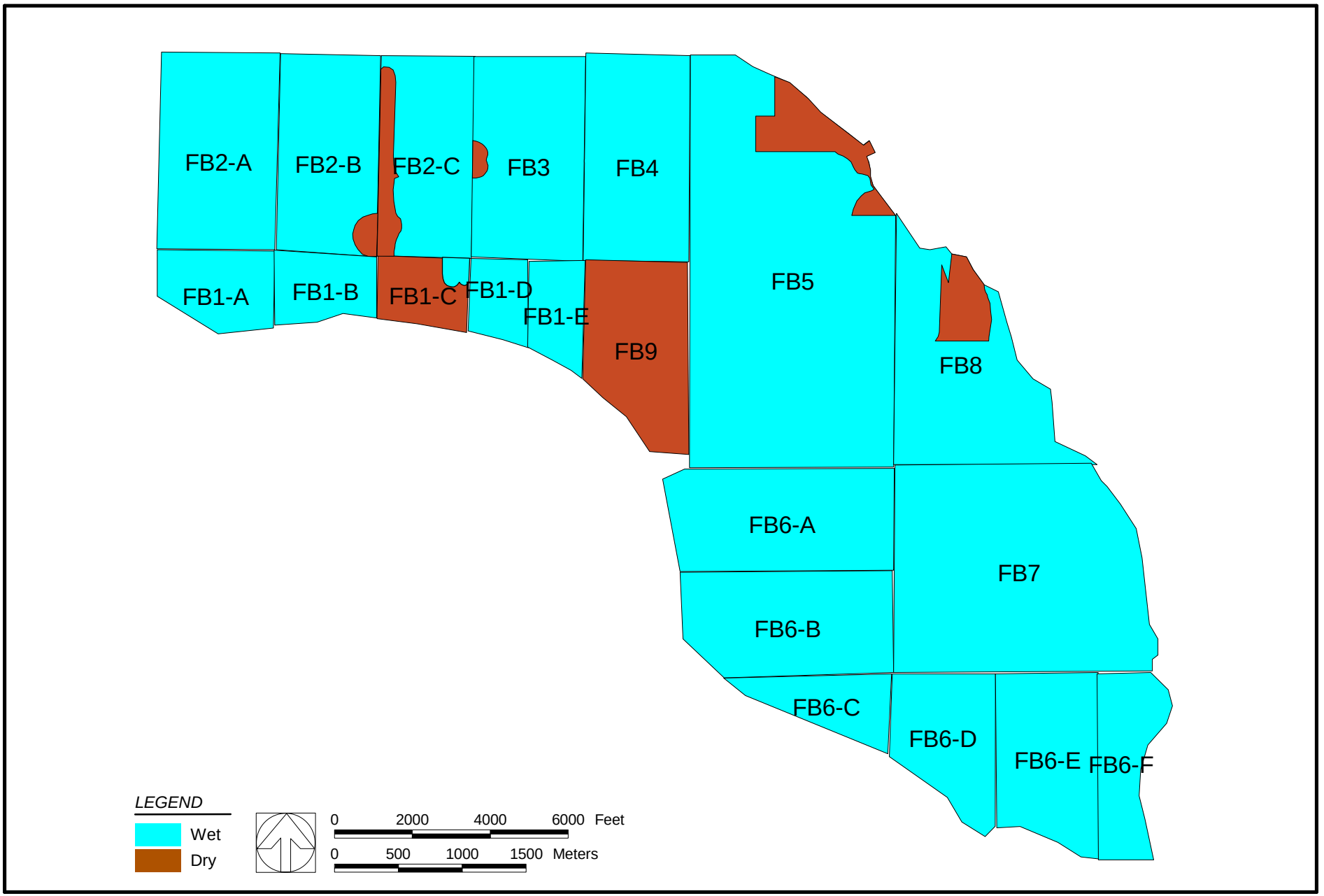


Figure 4-2. Flooding status of the North Shore Restoration Area of Lake Apopka on September 9, 1998

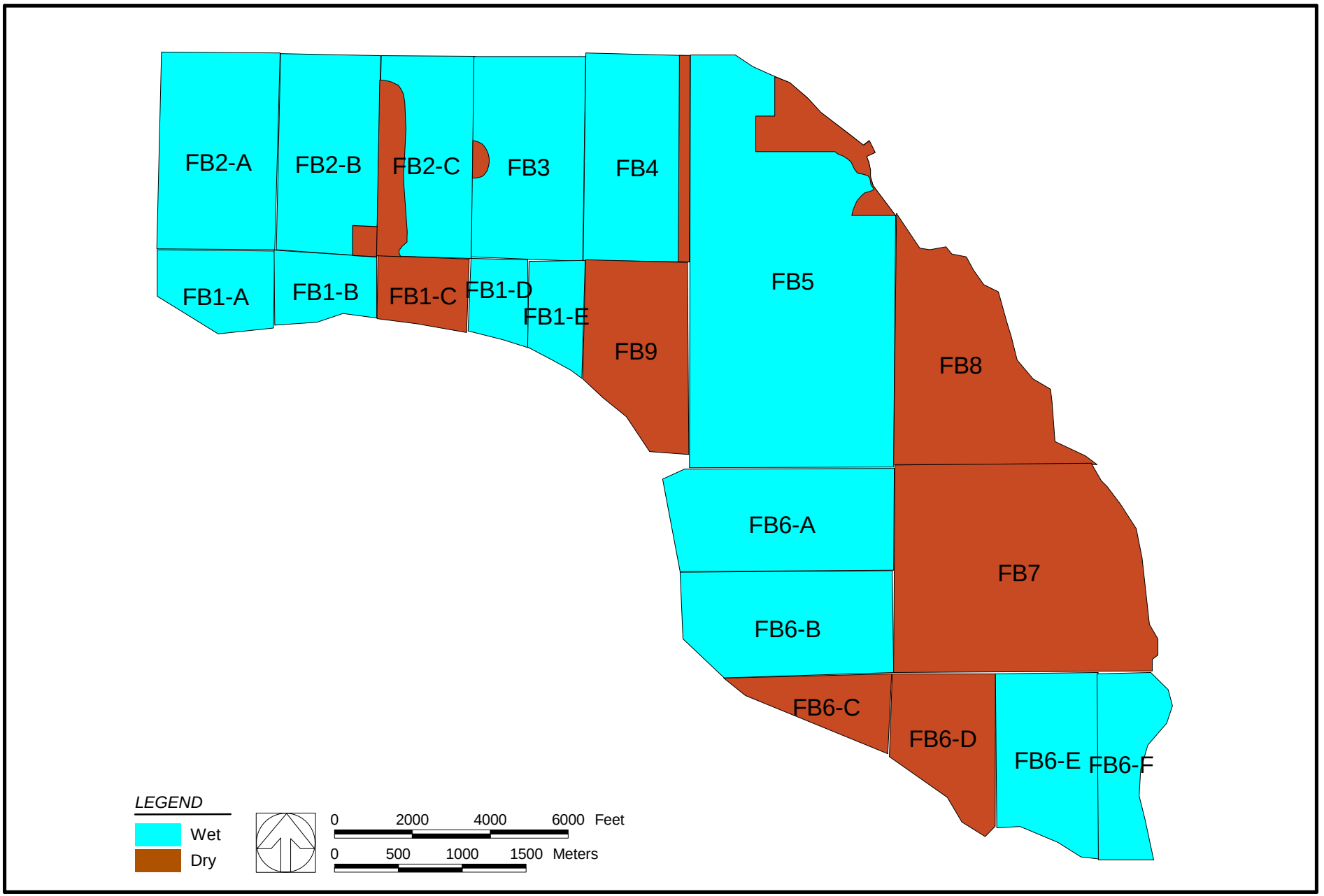


Figure 4-3. Flooding status of the North Shore Restoration Area of Lake Apopka on October 27, 1998

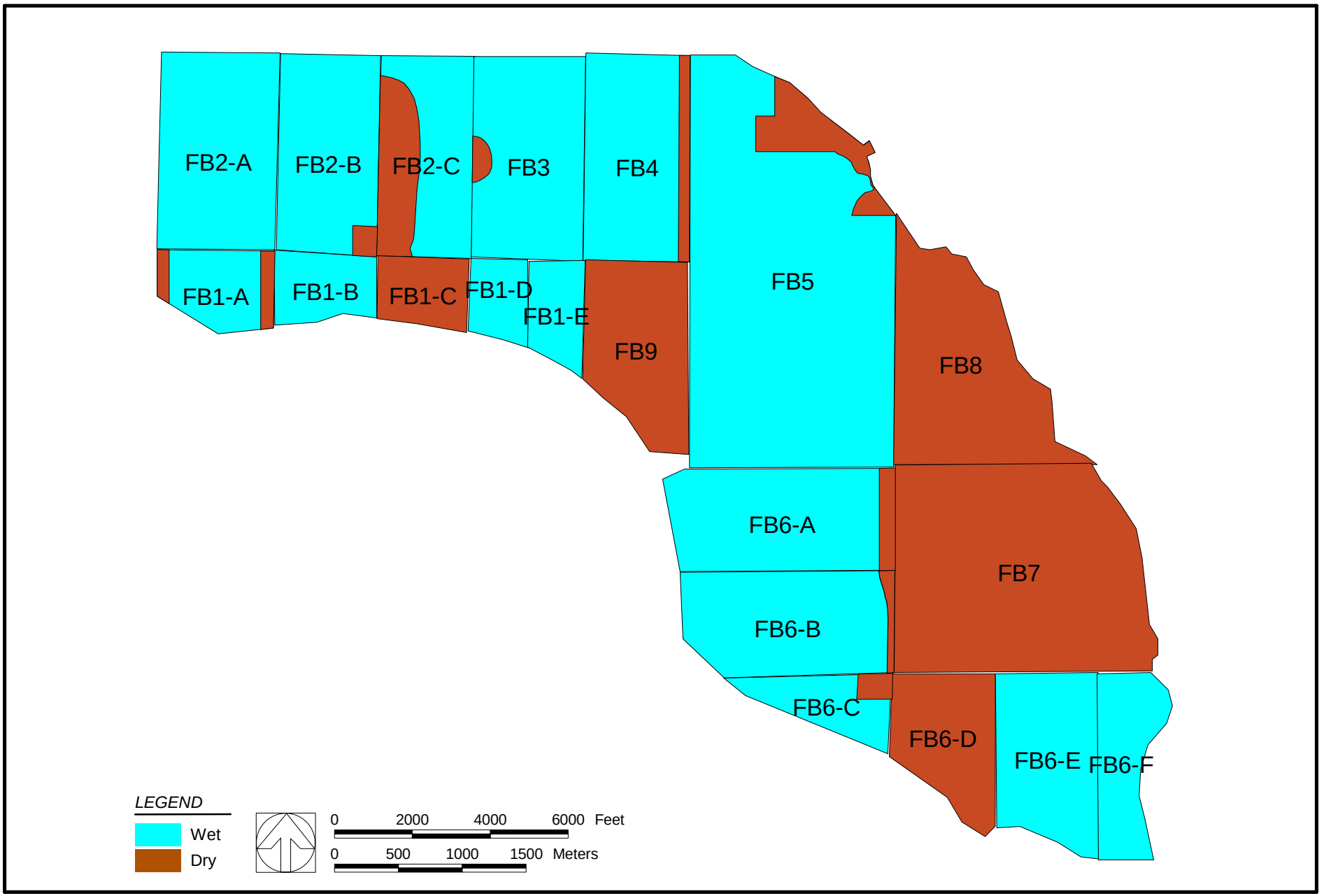


Figure 4-4. Flooding status of the North Shore Restoration Area of Lake Apopka on November 2, 1998

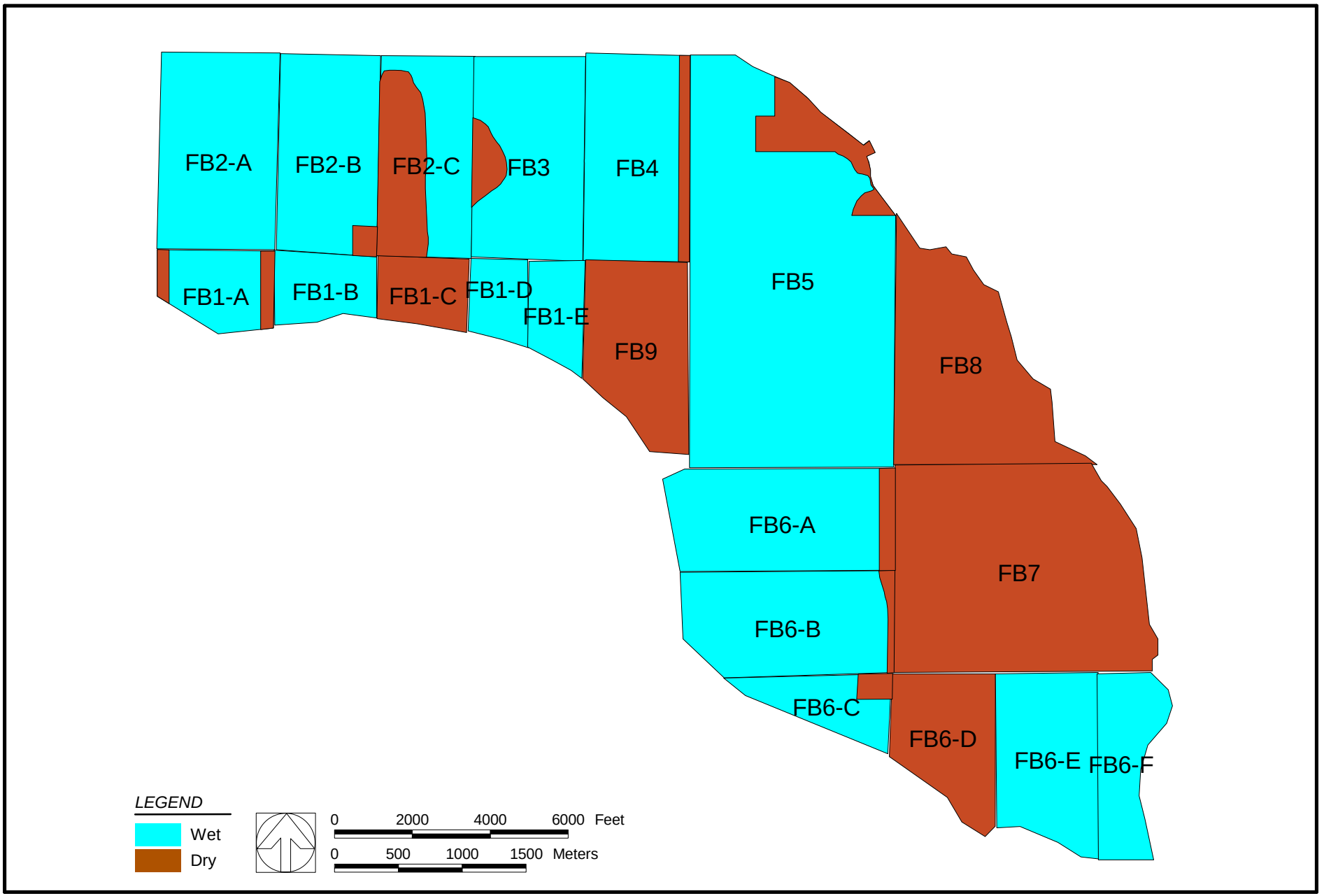


Figure 4-5. Flooding status of the North Shore Restoration Area of Lake Apopka on November 9, 1998

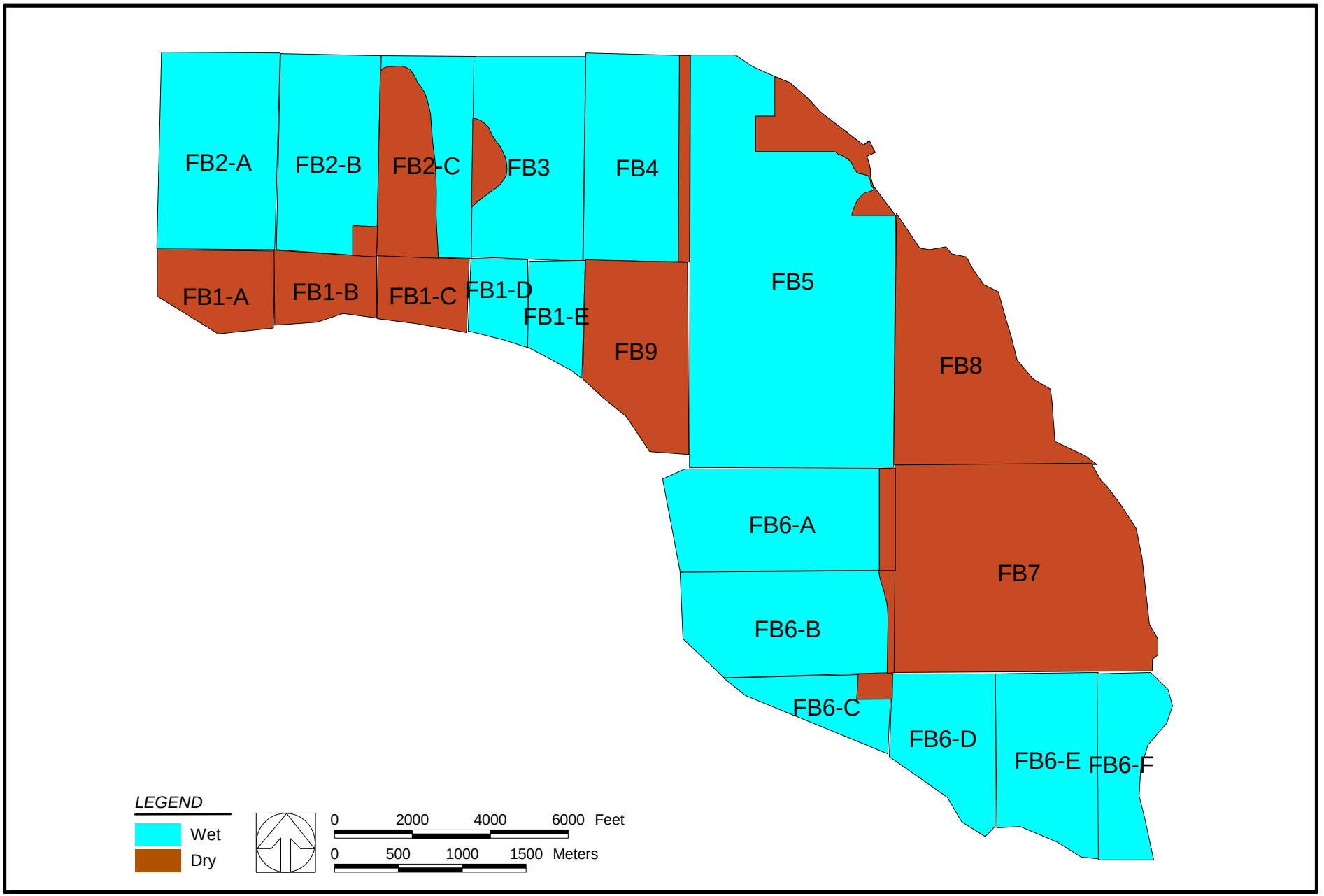


Figure 4-6. Flooding status of the North Shore Restoration Area of Lake Apopka on November 24, 1998

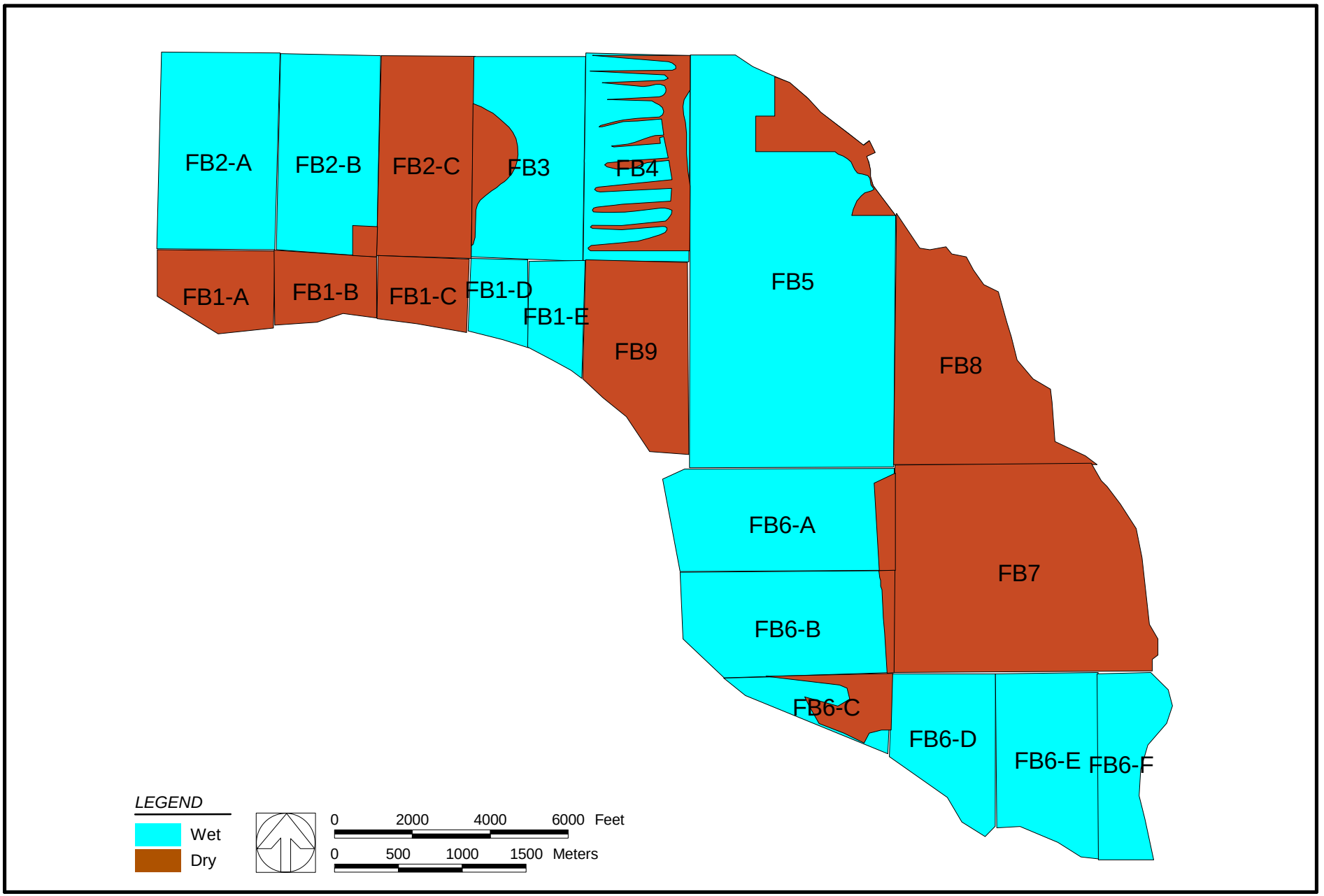


Figure 4-7. Flooding status of the North Shore Restoration Area of Lake Apopka on December 2, 1998

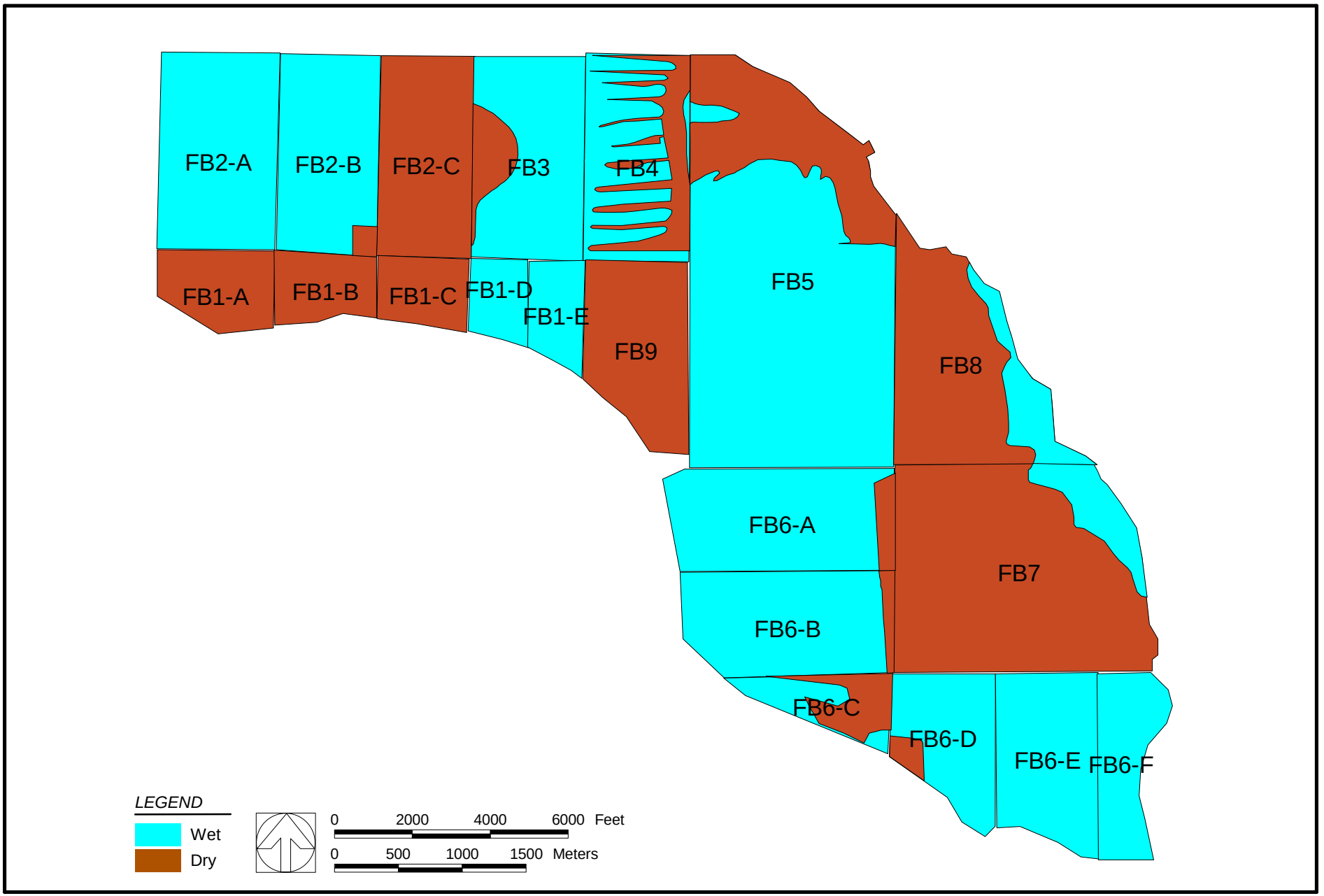


Figure 4-8. Flooding status of the North Shore Restoration Area of Lake Apopka on December 7, 1998

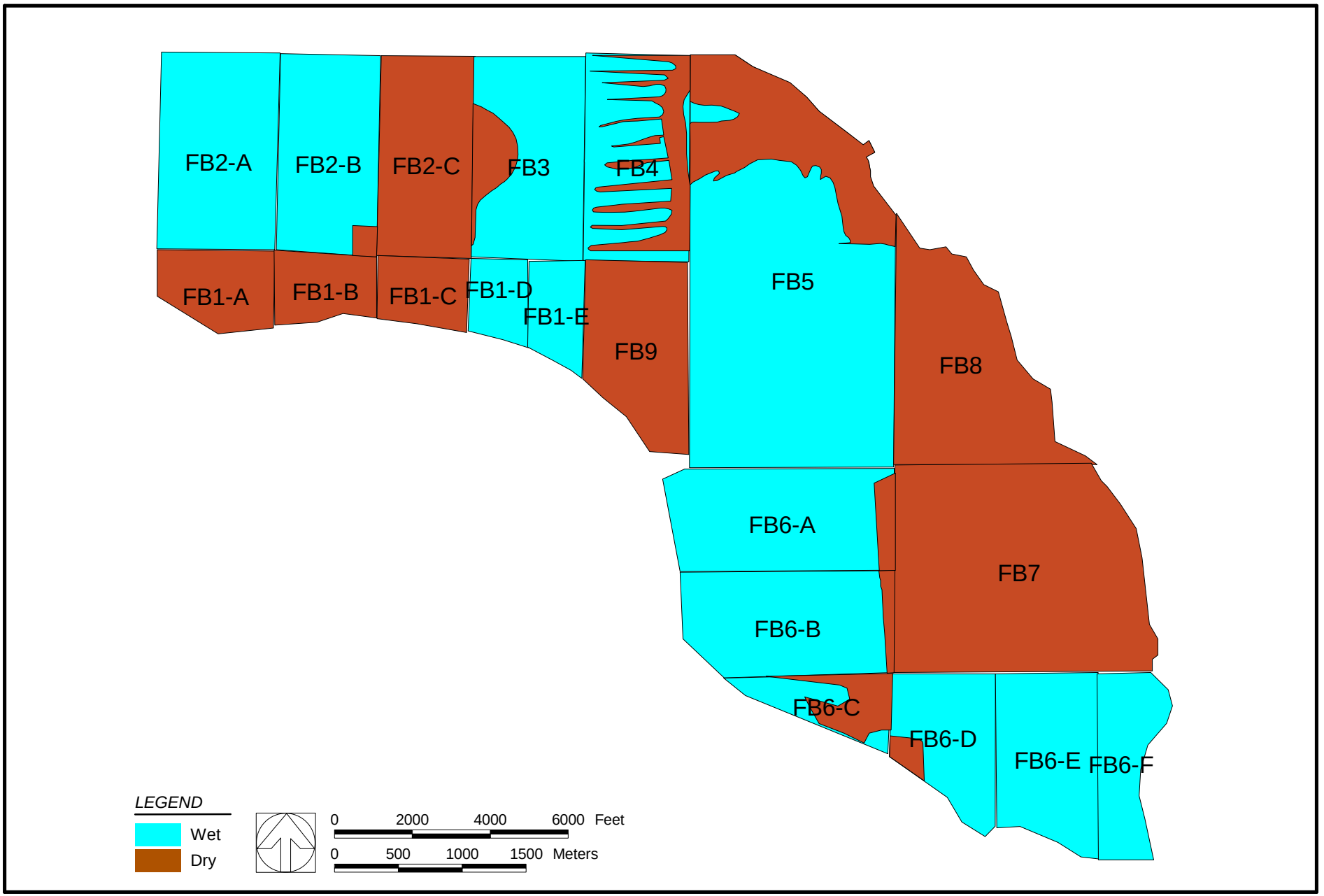


Figure 4-9. Flooding status of the North Shore Restoration Area of Lake Apopka on December 18, 1998

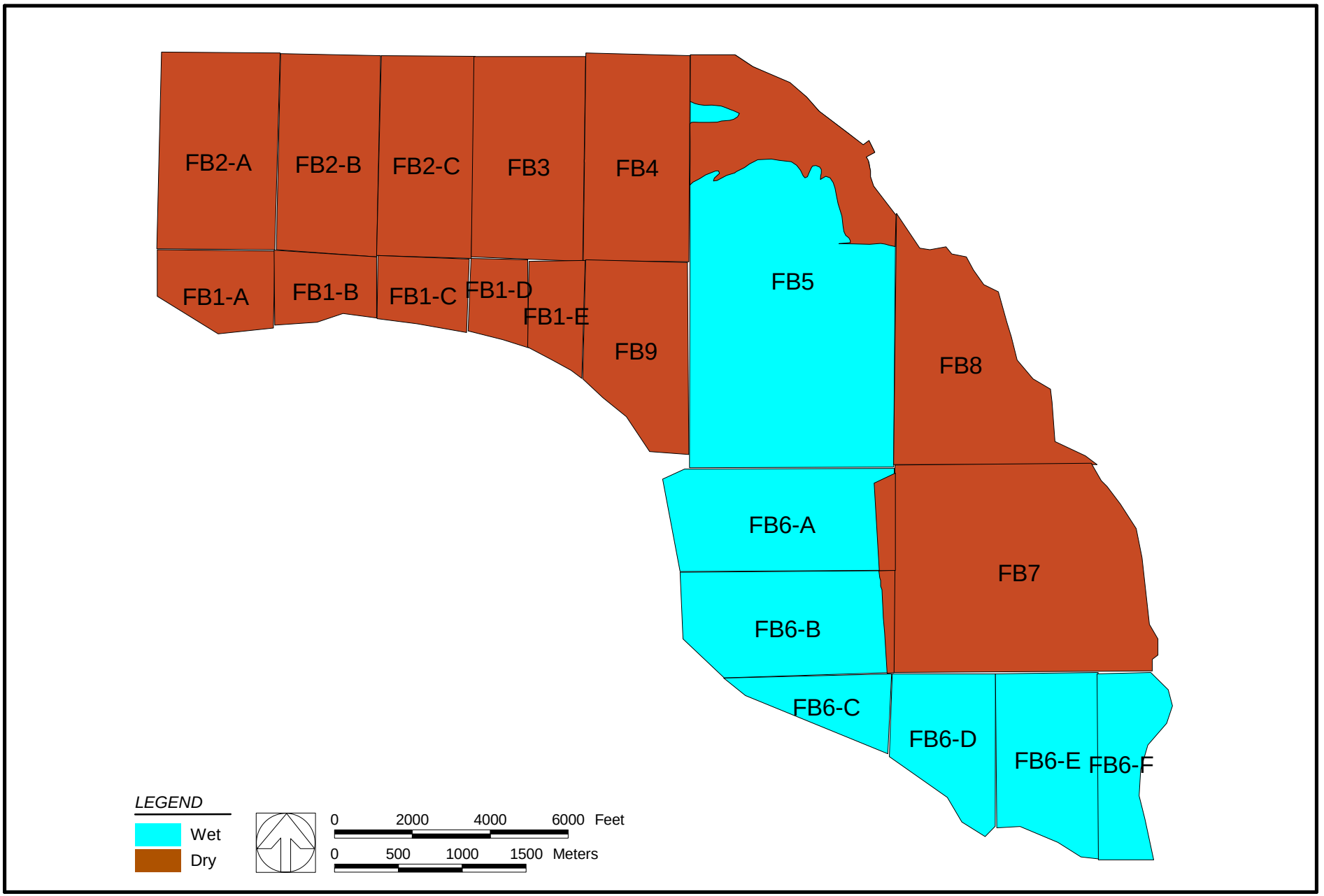


Figure 4-10. Flooding status of the North Shore Restoration Area of Lake Apopka on January 25, 1999

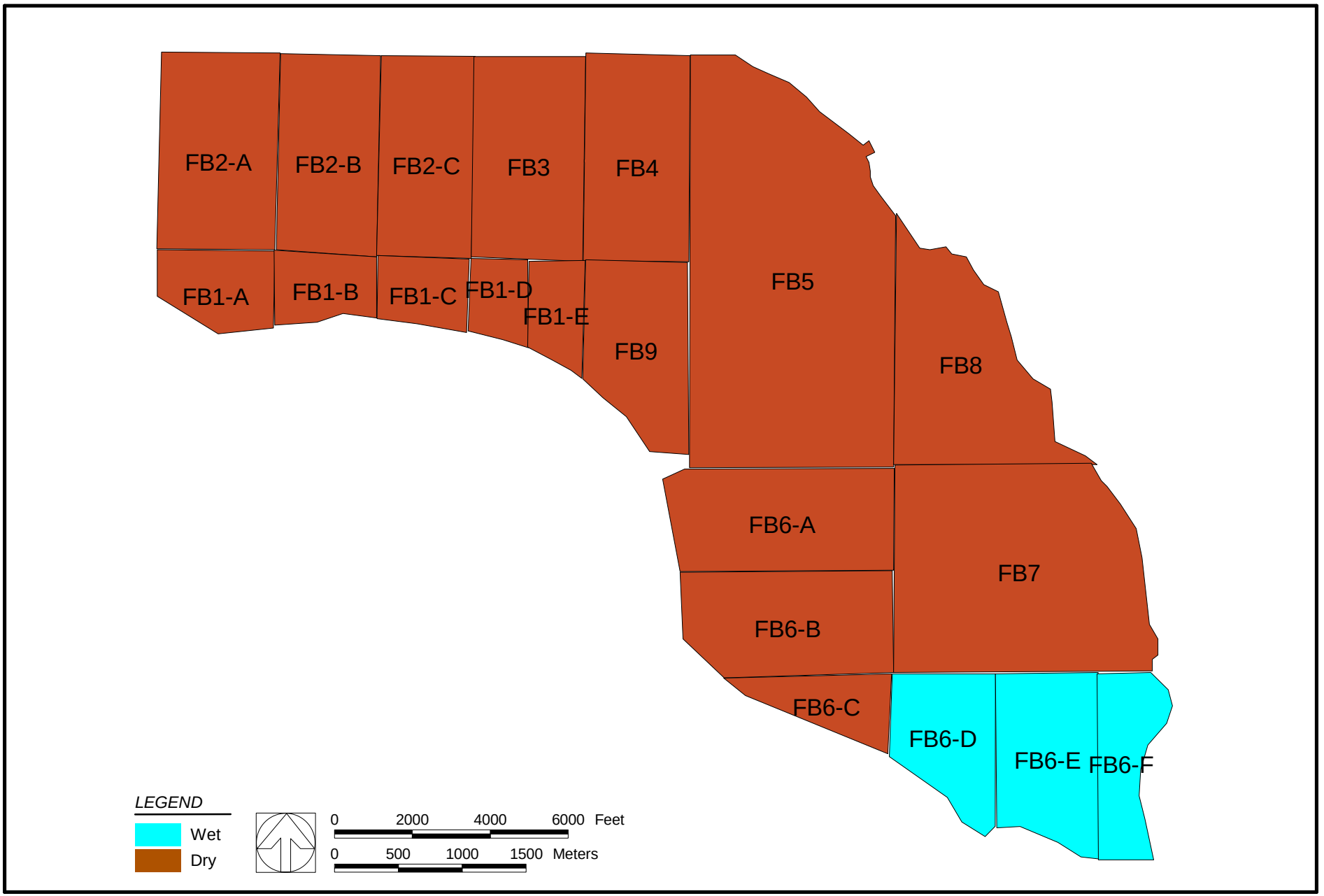
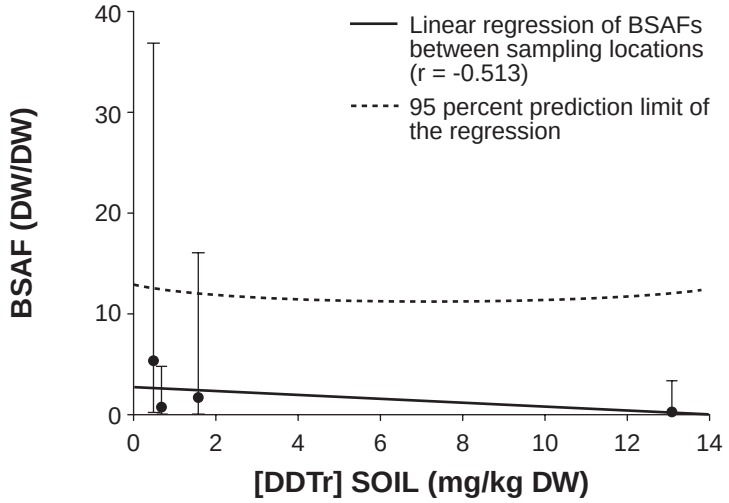
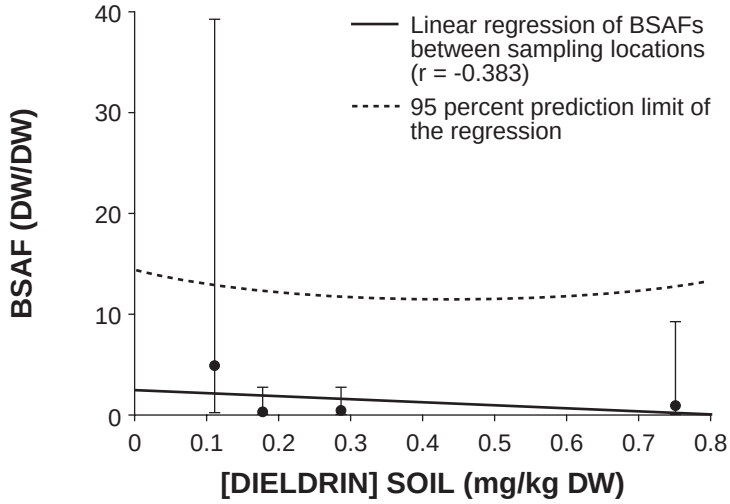
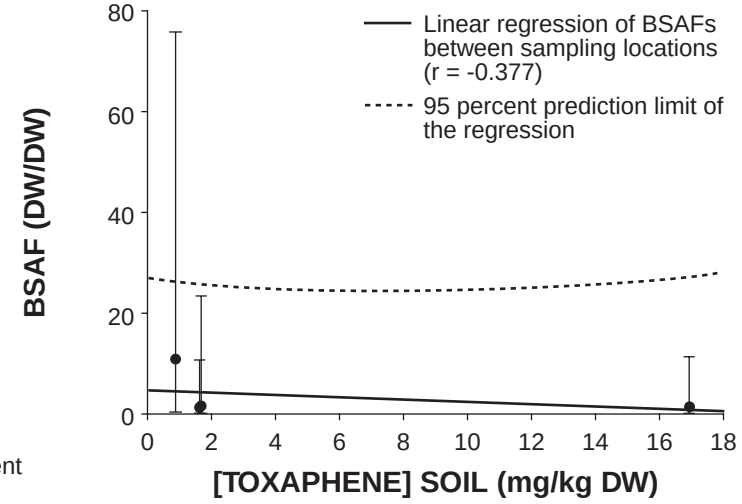


Figure 4-11. Flooding status of the North Shore Restoration Area of Lake Apopka on February 9, 1999

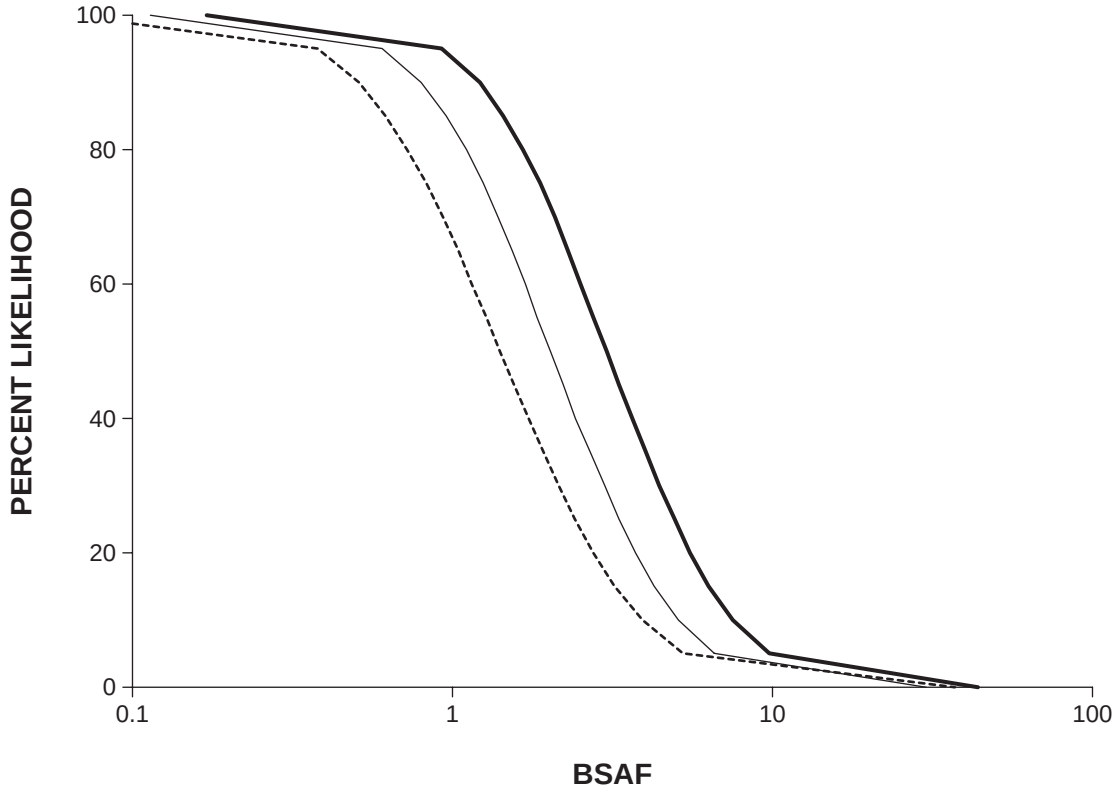






Note: BSAF–biota-sediment accumulation factor

Figure 4-12. Linear regressions of the relationship between organochlorine concentrations in sediment and BSAFs in teleost fish



Note: BSAF–biota-sediment accumulation factor

Figure 4-13. Probability distributions of BSAF values for prey fish

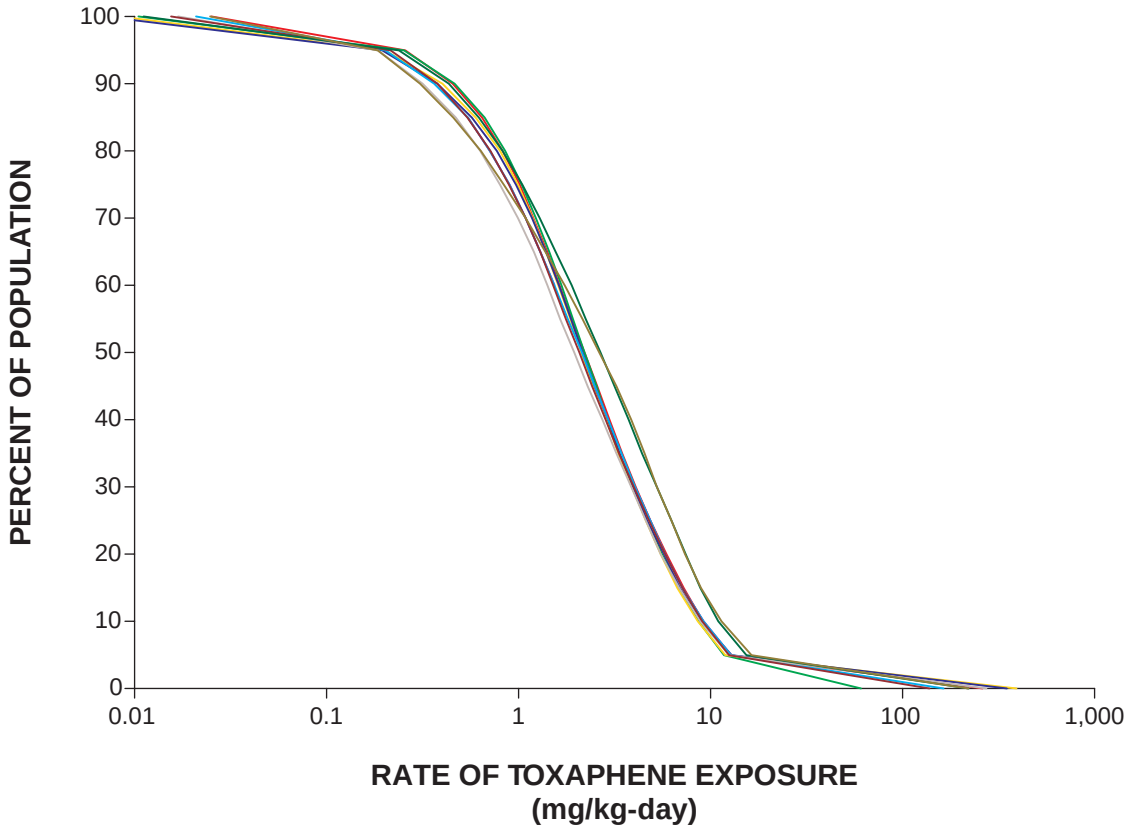


Figure 4-14. Variation in the proportion of the American white pelican population at the North Shore Restoration Area exposed to toxaphene at different time periods (i.e., different inundation scenarios)

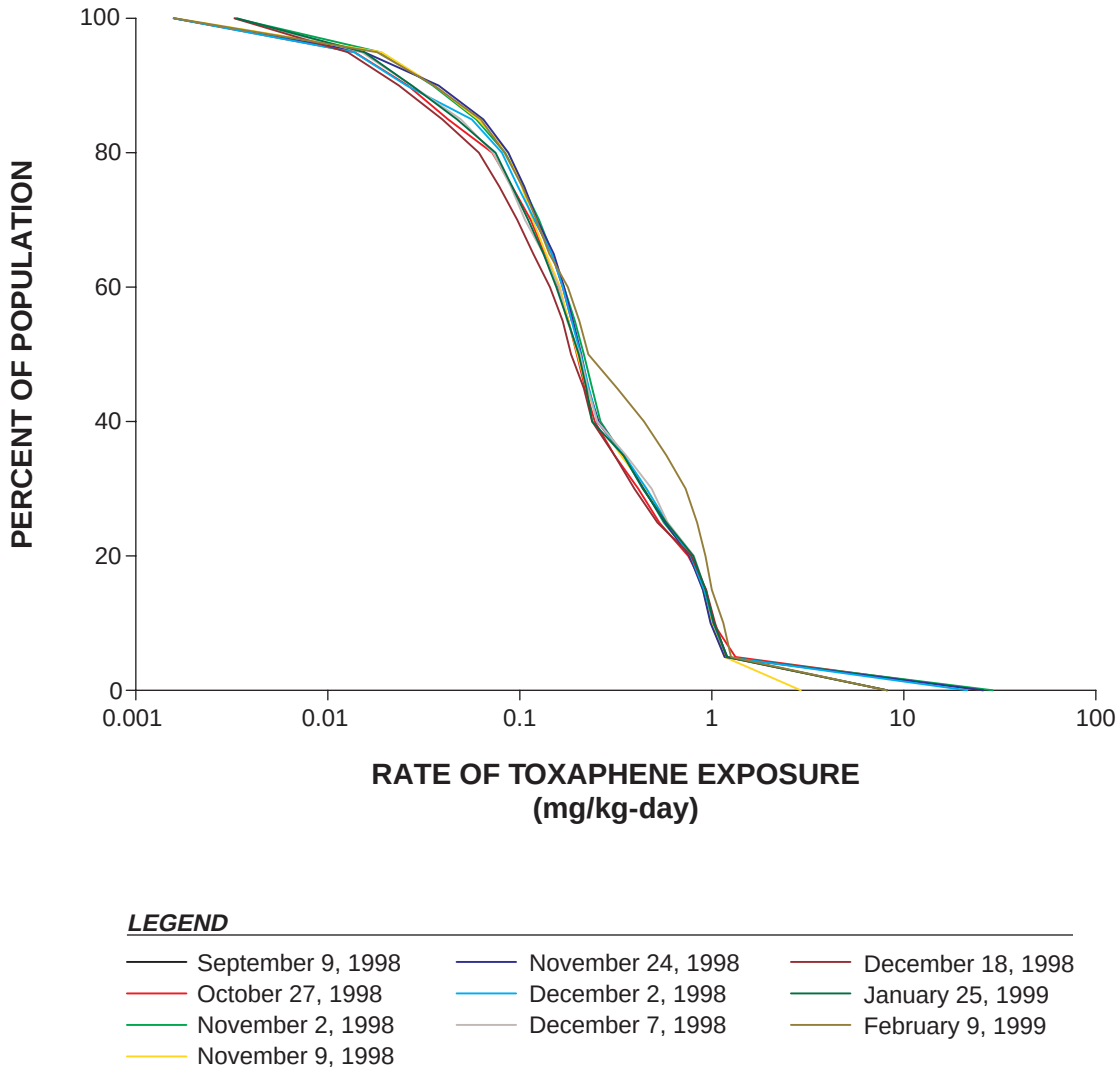


Figure 4-15. Variation in the proportion of the least sandpiper population at the North Shore Restoration Area exposed to toxaphene at different time periods (i.e., different inundation scenarios)

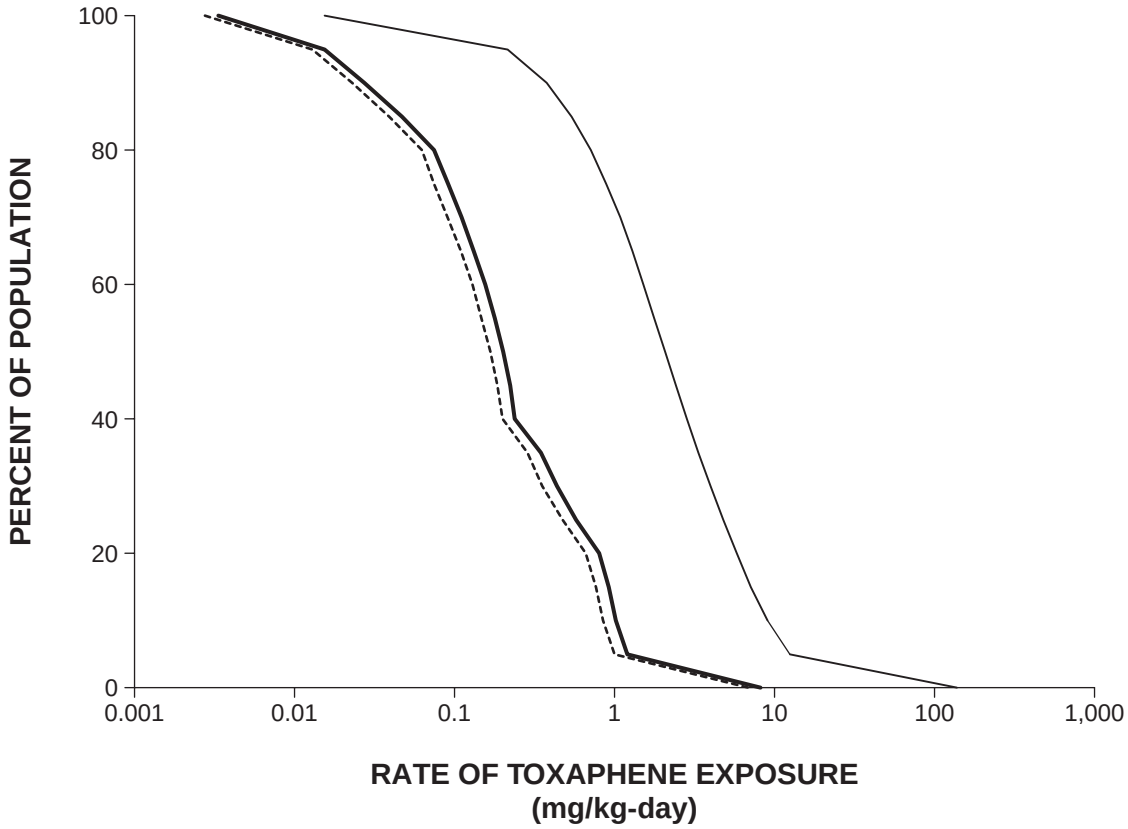


Figure 4-16. Comparison of modeled daily exposure distributions for toxaphene between the American white pelican, least sandpiper, and American coot at the North Shore Restoration Area

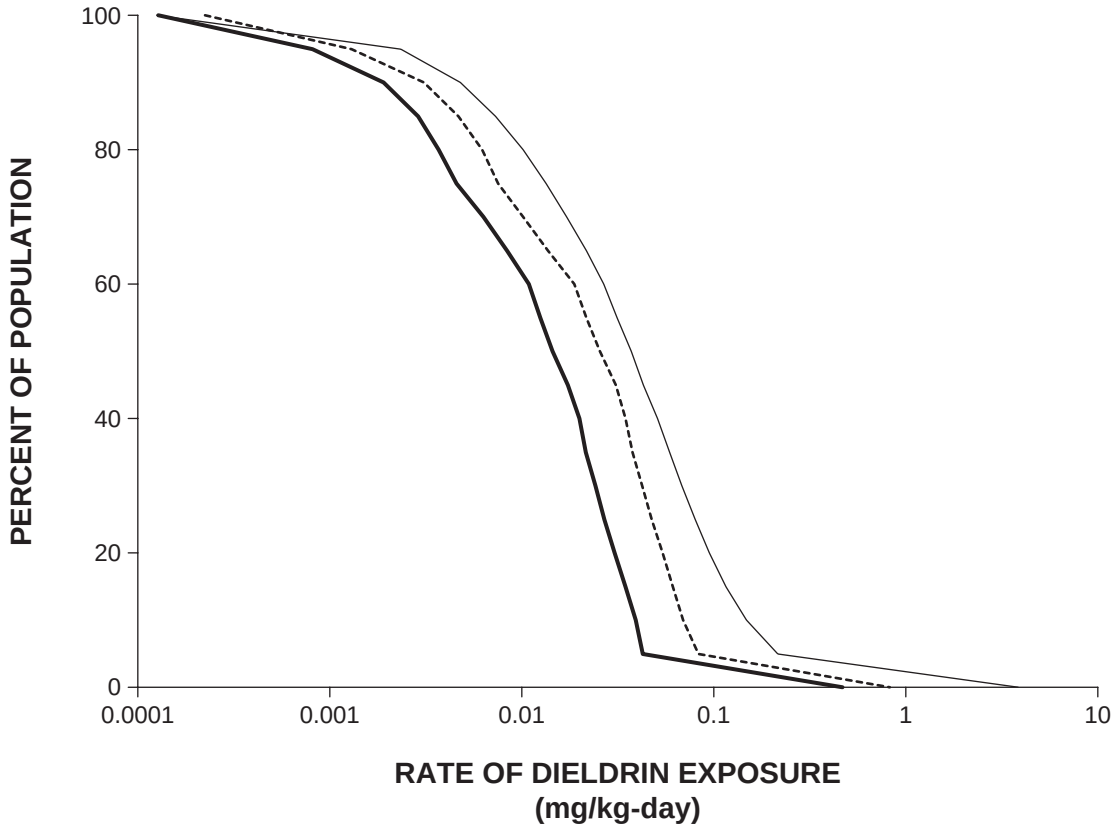


Figure 4-17. Comparison of modeled daily exposure distributions for dieldrin between the American white pelican, least sandpiper, and American coot at the North Shore Restoration Area

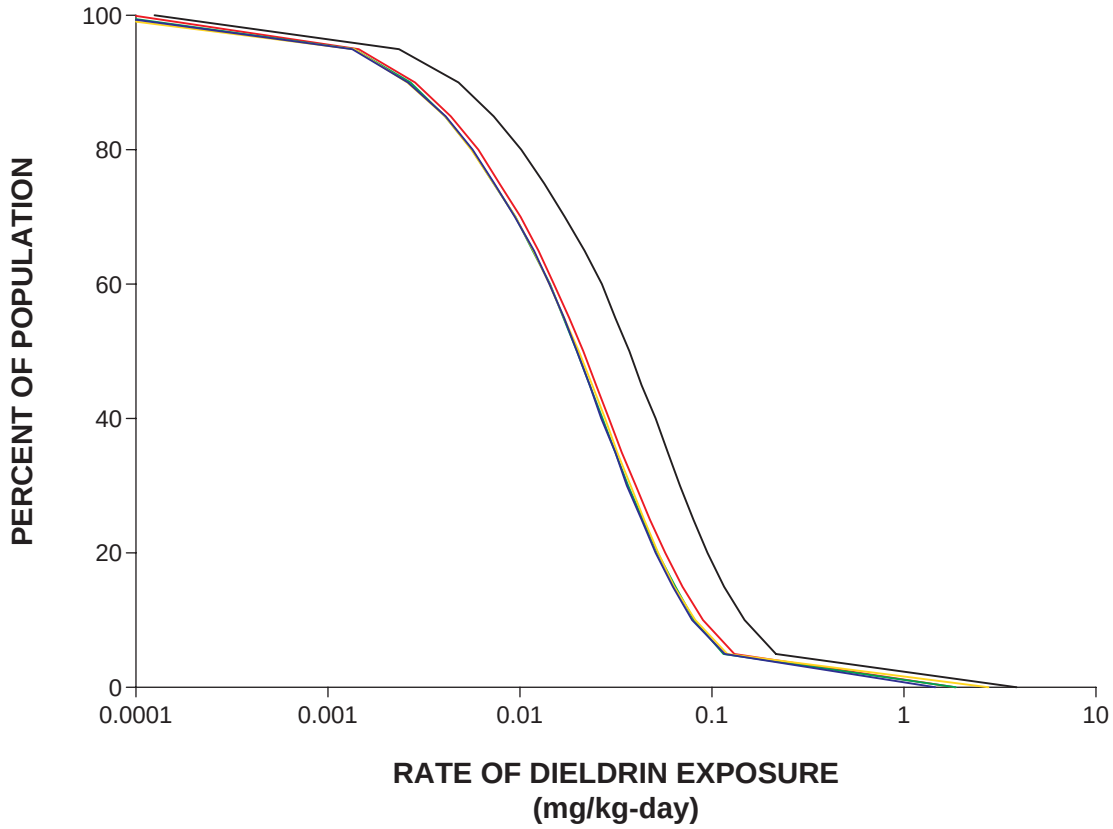


Figure 4-18. Comparison of modeled daily exposure distributions for dieldrin between the American white pelican and other piscivorous species at the North Shore Restoration Area

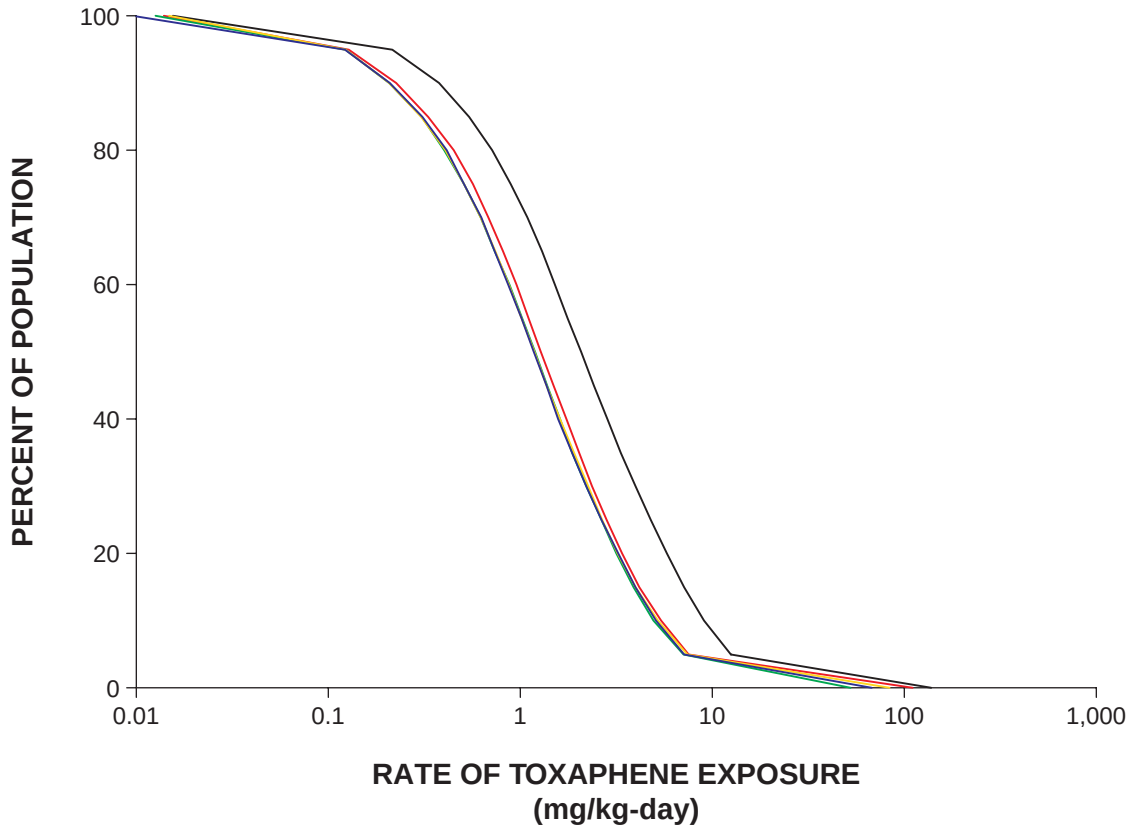


Figure 4-19. Comparison of modeled daily exposure distributions for toxaphene between the American white pelican and other piscivorous species at the North Shore Restoration Area

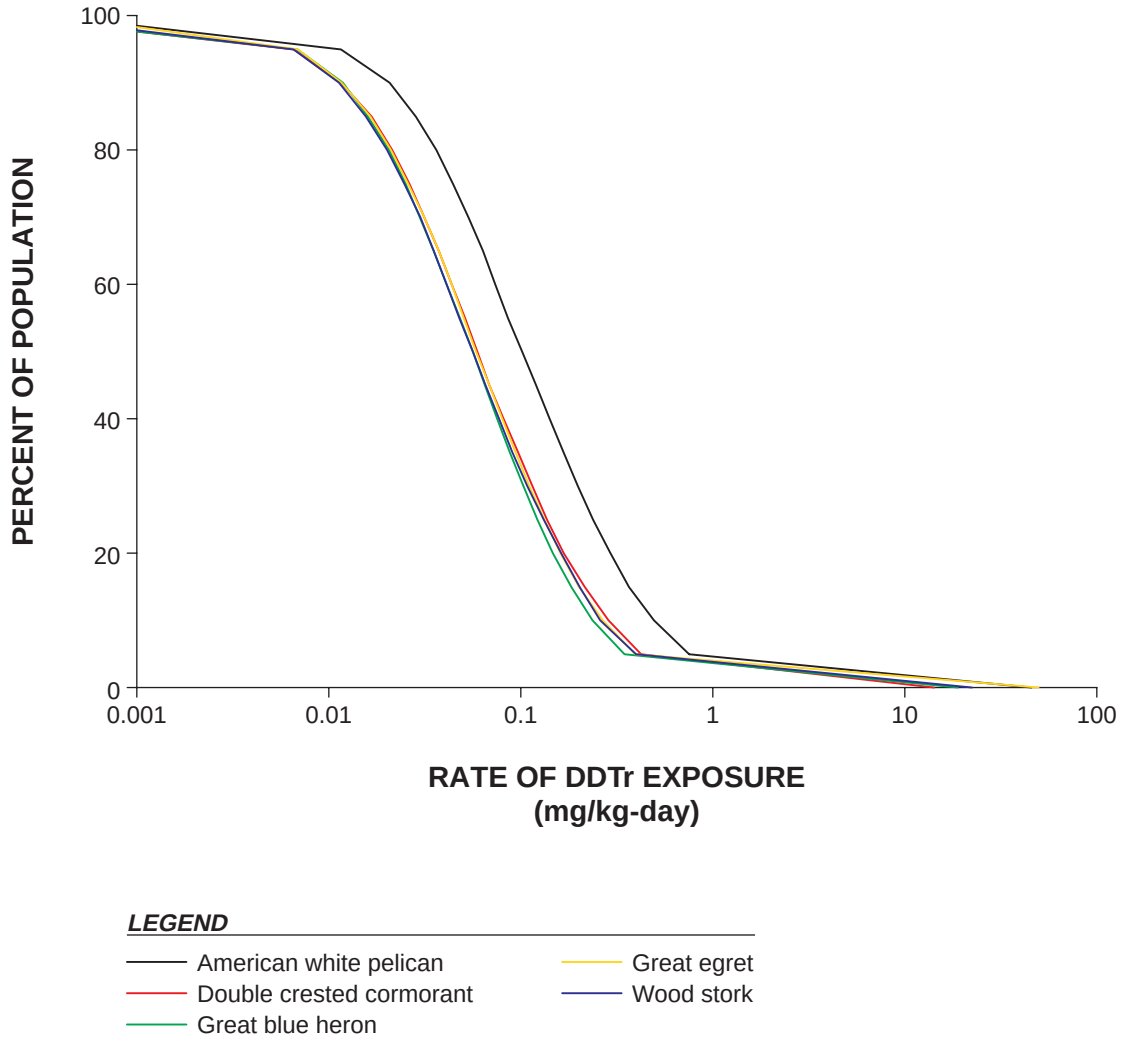


Figure 4-20. Comparison of modeled daily exposure distributions for DDTr between the American white pelican and other piscivorous species at the North Shore Restoration Area

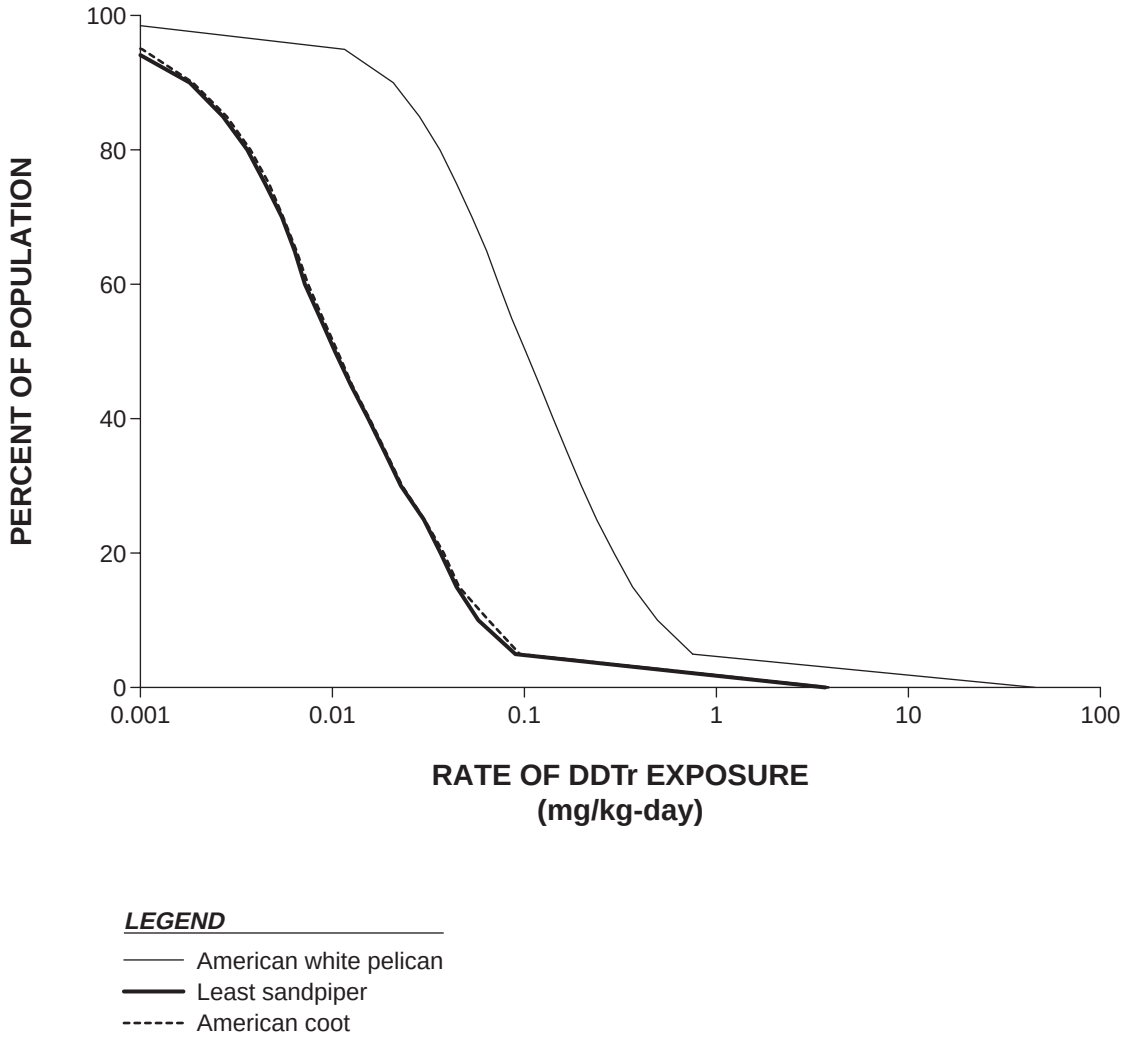


Figure 4-21. Comparison of modeled daily exposure distributions for DDTr between the American white pelican, least sandpiper, and American coot at the North Shore Restoration Area

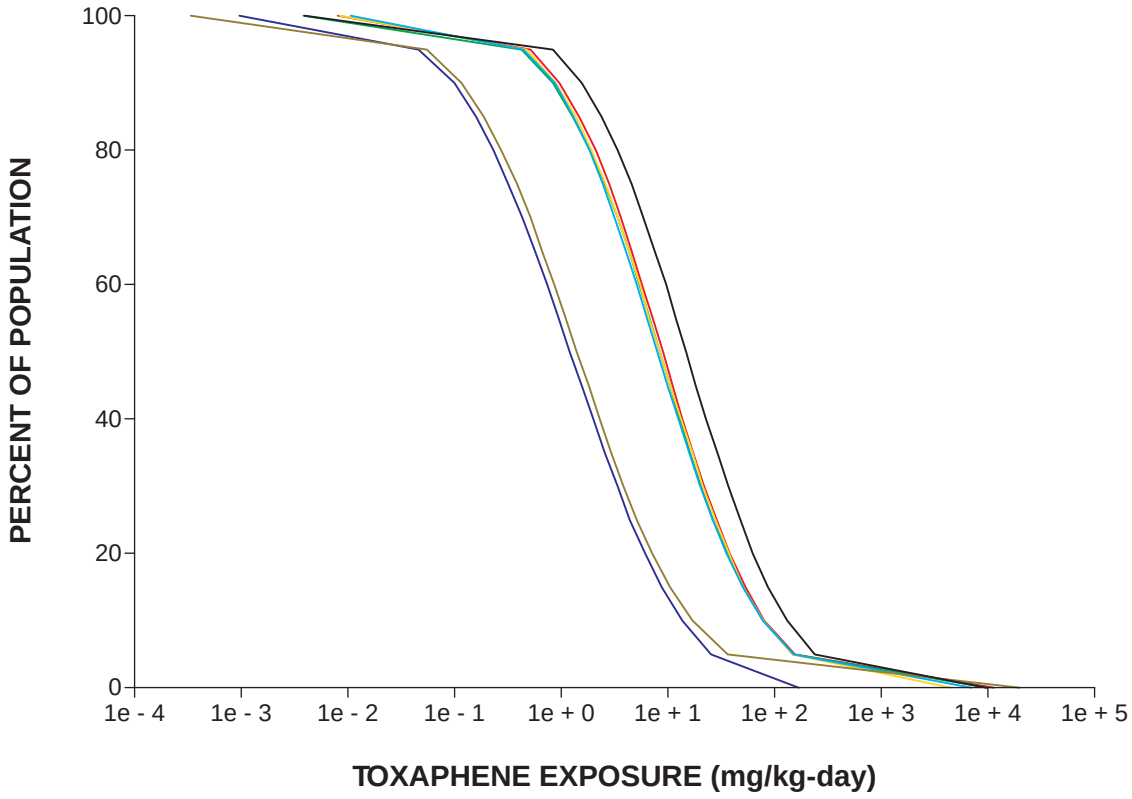


Figure 4-22. Comparison of relative exposure distributions of toxaphene among bird species resident exclusively in the vicinity of the airstrip

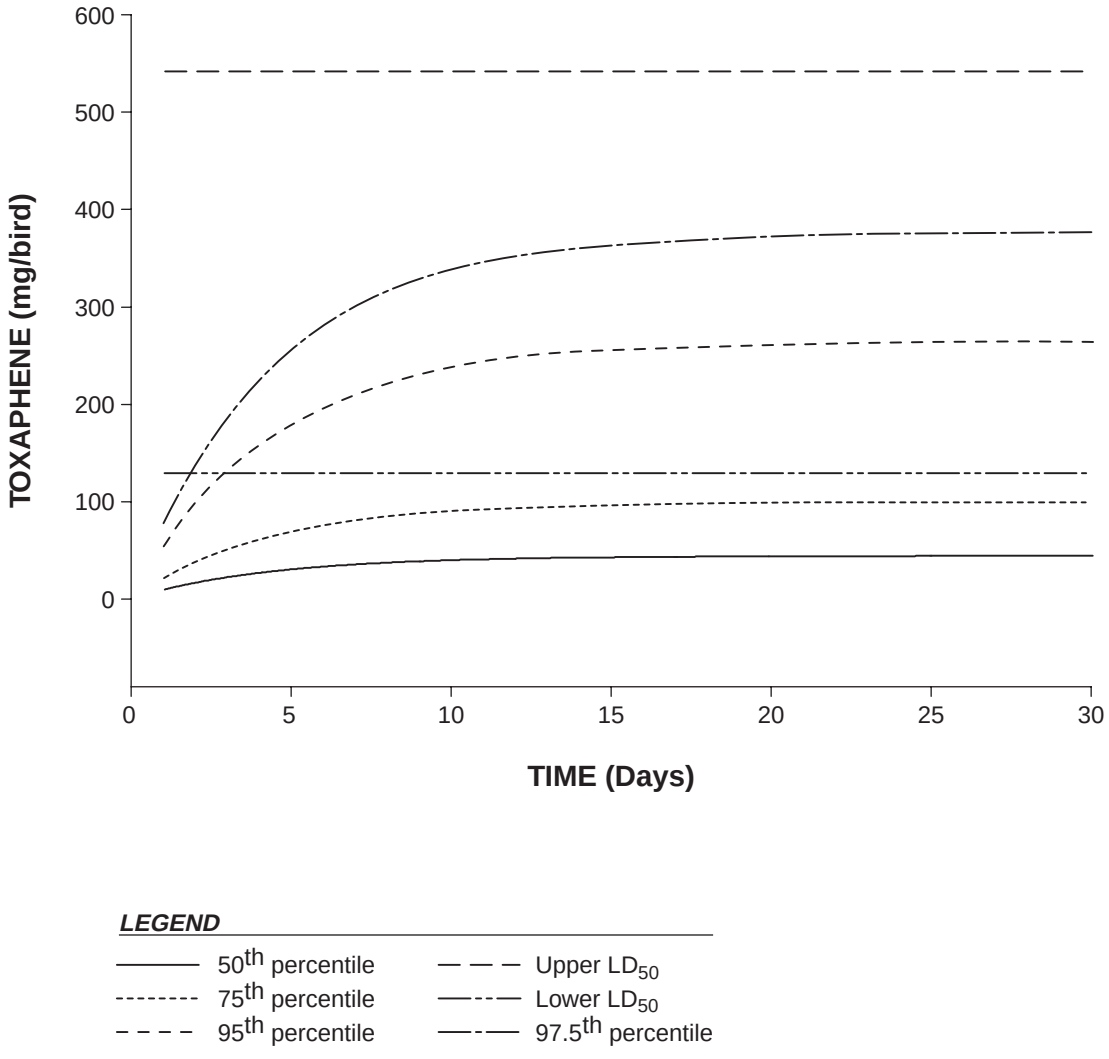


Figure 4-23. Projected body burden accumulation in the American white pelican from daily exposure to toxaphene associated with the North Shore Restoration Area

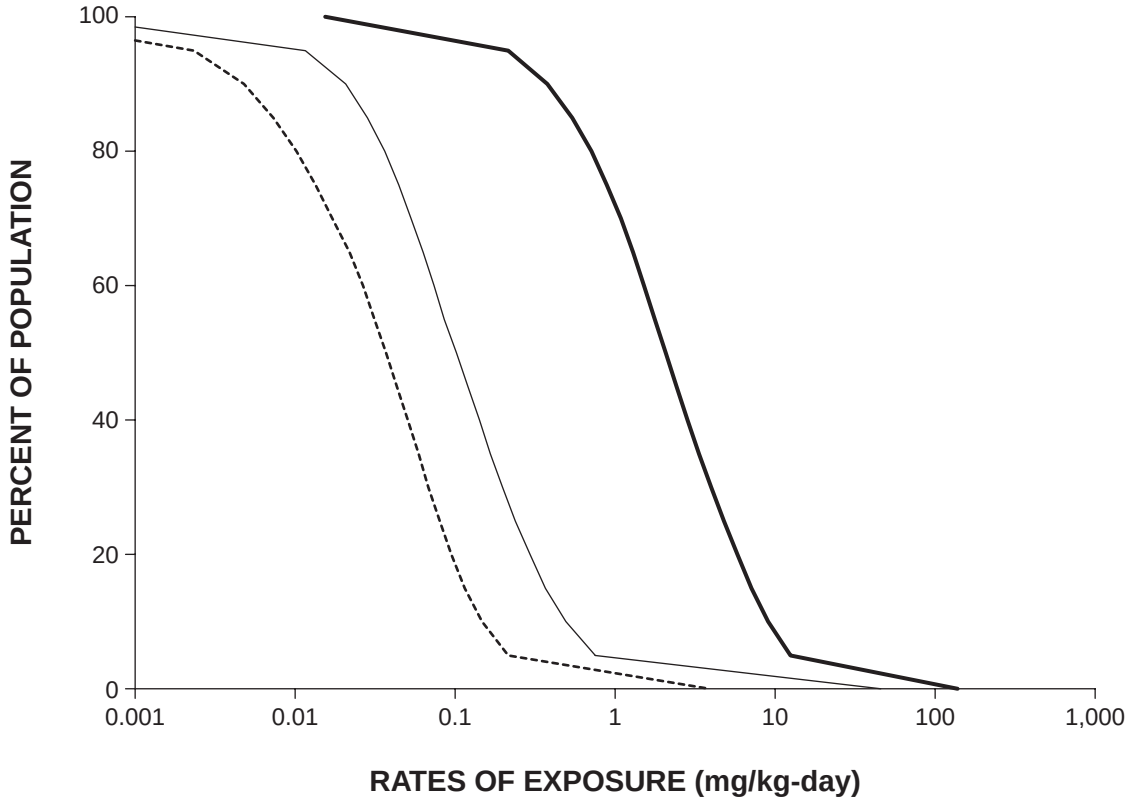


Figure 4-24. Relative distribution of exposure to organochlorine pesticides for the American white pelican resident in the North Shore Restoration Area

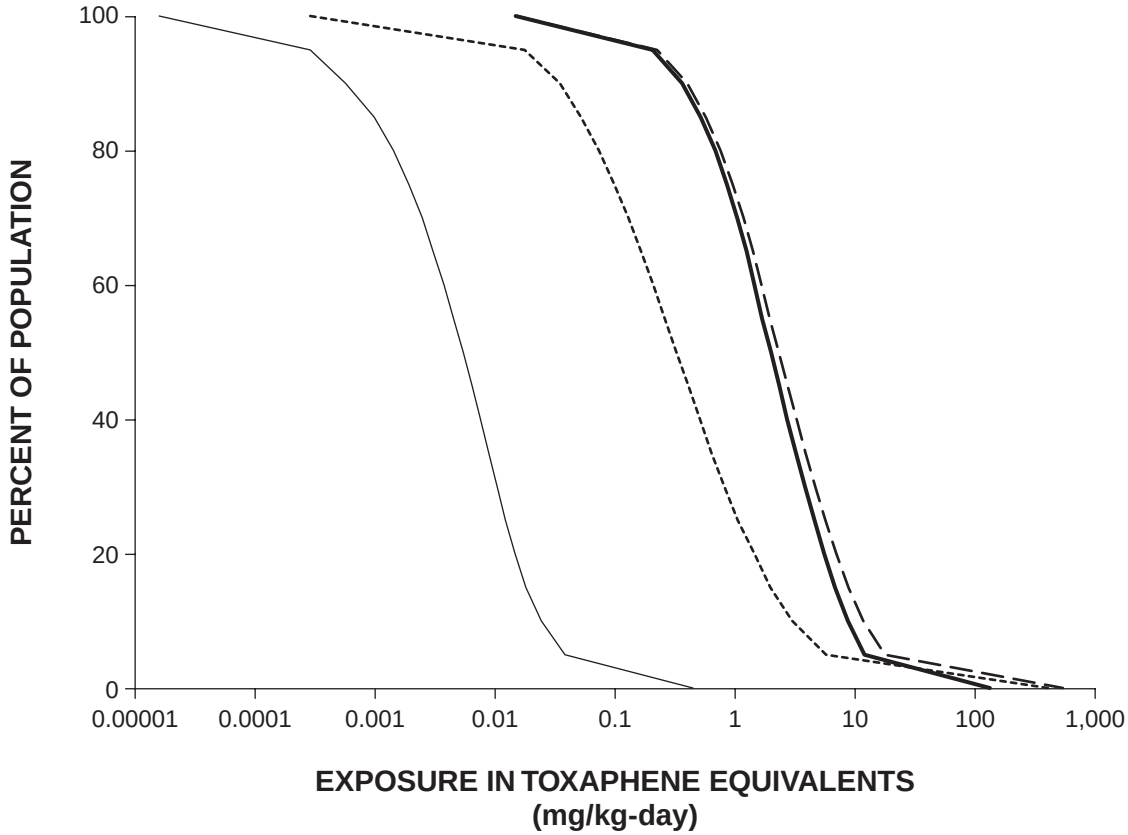
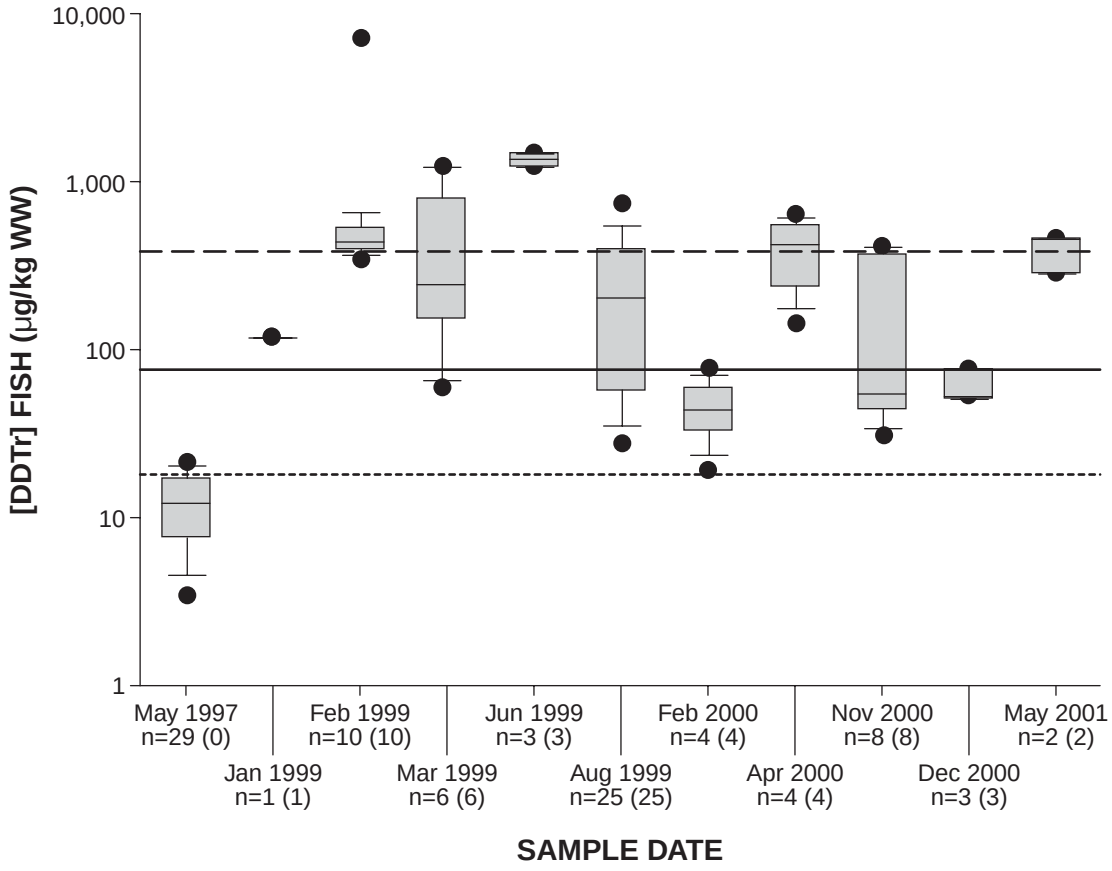
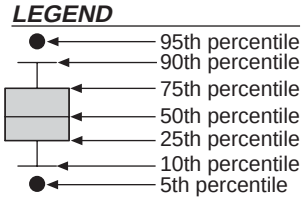


Figure 4-25. Comparative exposure of the American white pelican resident in the North Shore Restoration Area to organochlorine pesticides expressed as acute toxaphene equivalents



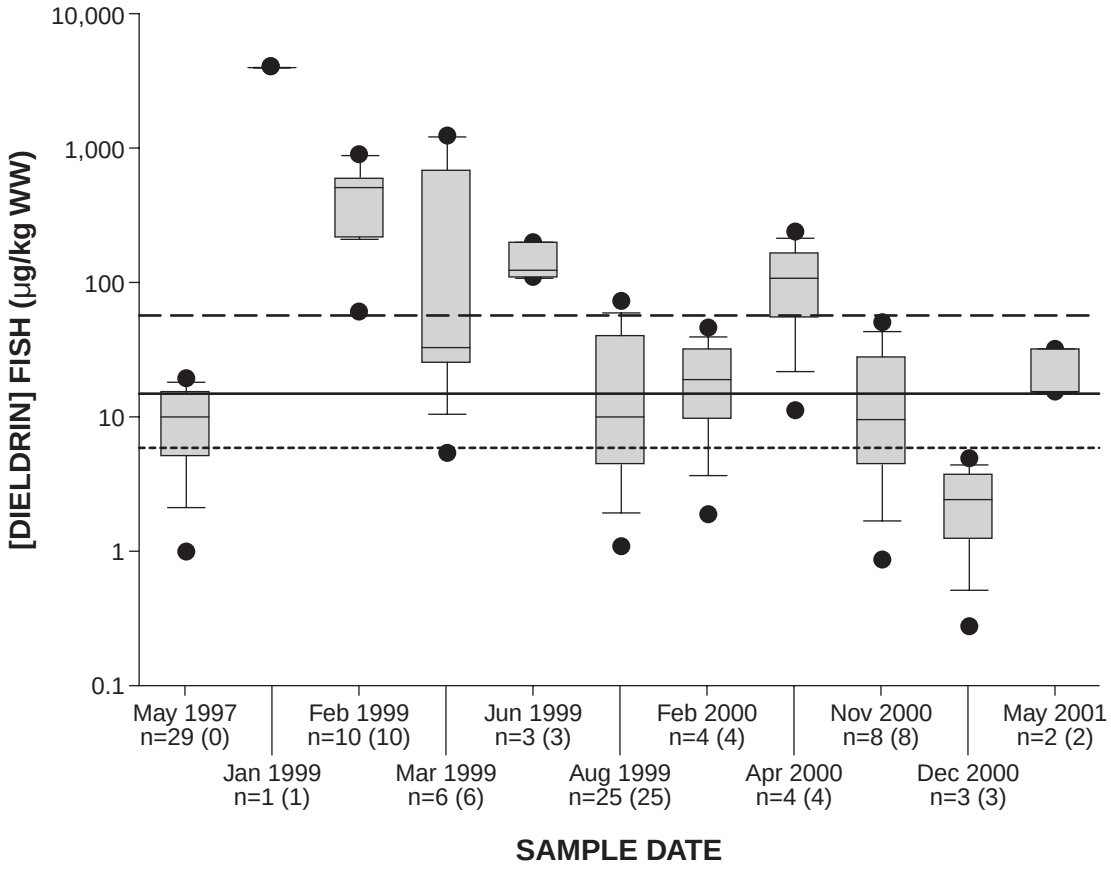


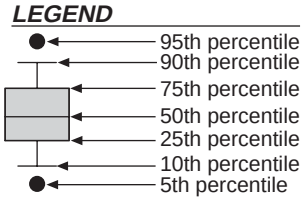
Notes: Horizontal lines represent the 25th, 50th, and 75th percentiles of the entire data set.

The n-values represent the number of observations with the number of detected observations in parentheses.

DDTr scored as detected if any metabolite was measured above quantification limits.

Figure 4-26. Time-course of DDTr concentrations in blue tilapia sampled in the vicinity of the North Shore Restoration Area

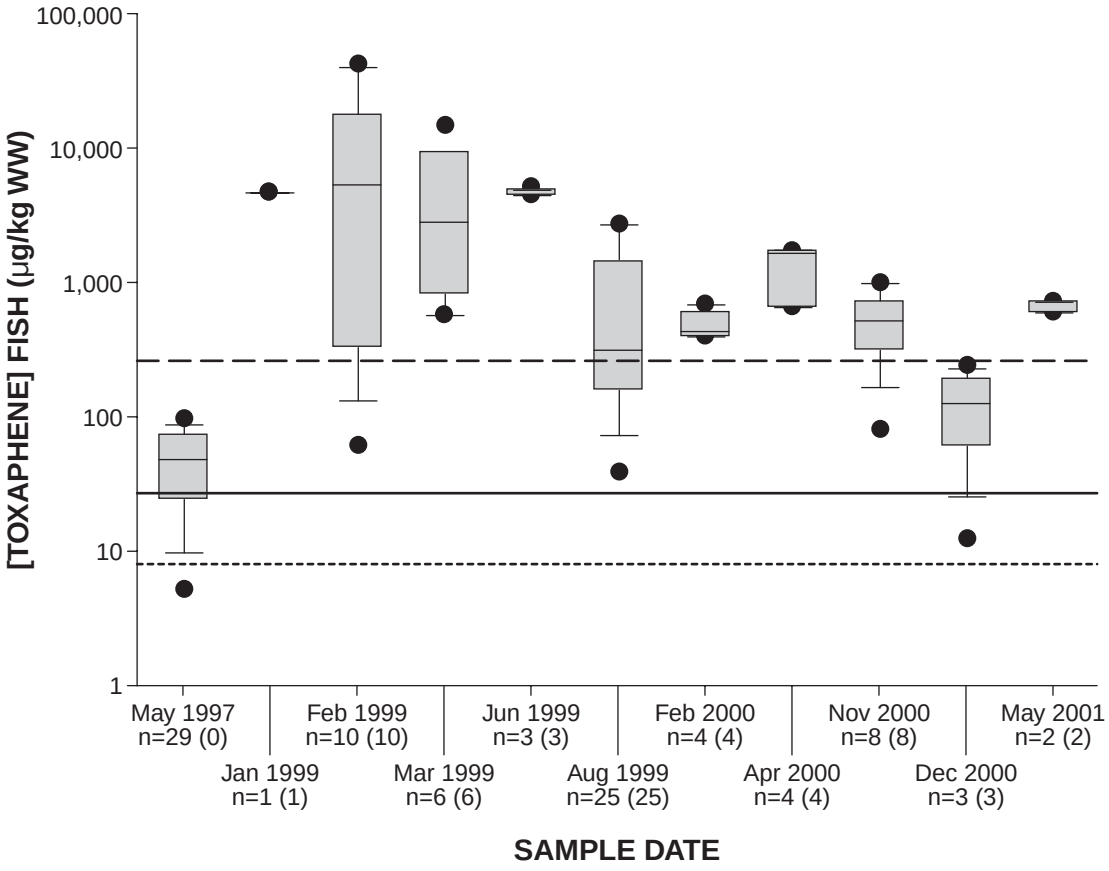


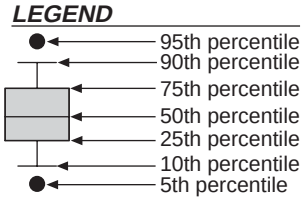


Notes: Horizontal lines represent the 25th, 50th, and 75th percentiles of the entire data set.

The n-values represent the number of observations with the number of detected observations in parentheses.

Figure 4-27. Time-course of dieldrin concentrations in blue tilapia sampled in the vicinity of the North Shore Restoration Area

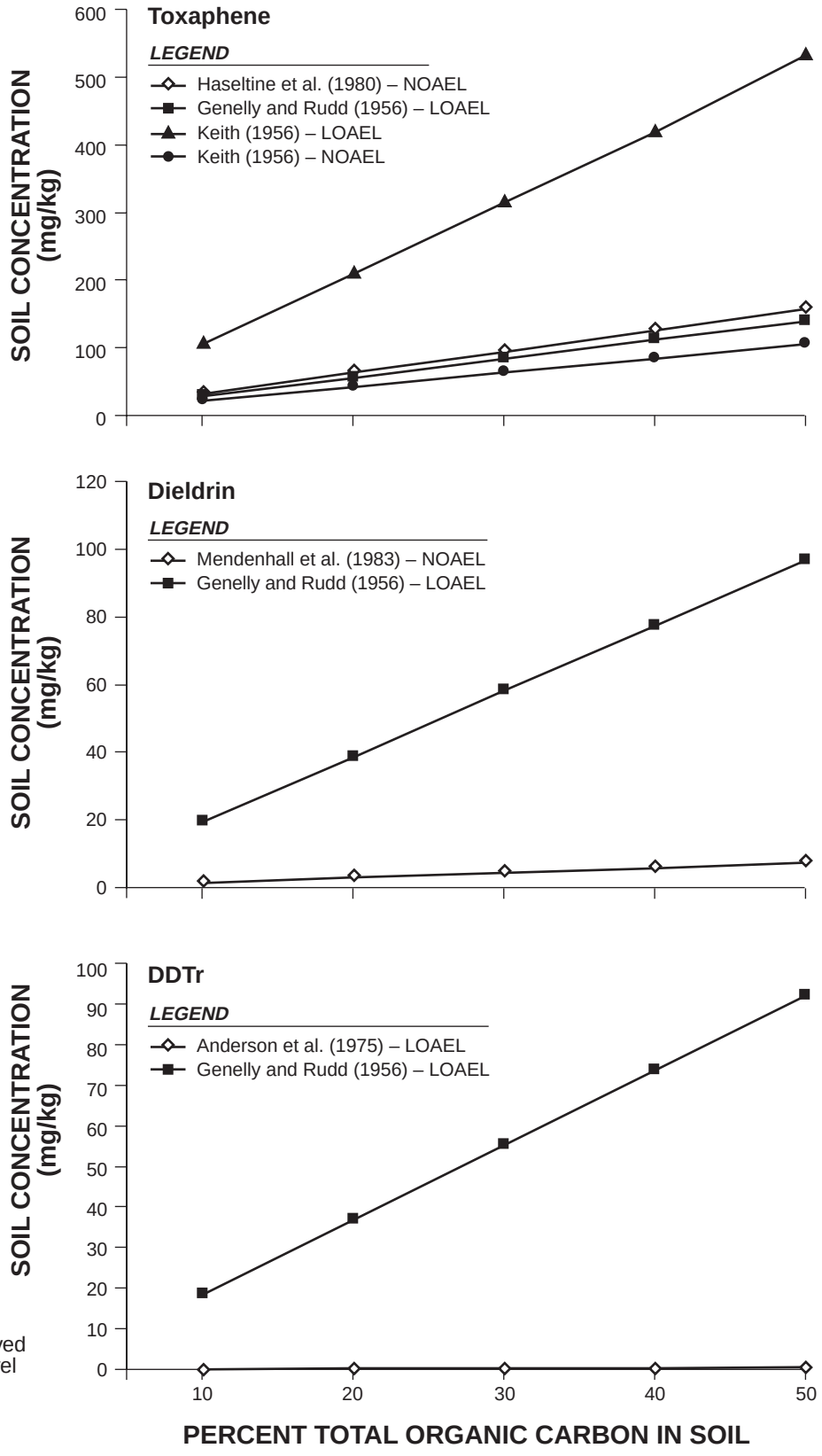




Notes: Horizontal lines represent the 25th, 50th, and 75th percentiles of the entire data set.

The n-values represent the number of observations with the number detected observations in parentheses.

Figure 4-28. Time-course of toxaphene concentrations in blue tilapia sampled in the vicinity of the North Shore Restoration Area



Note: NOAEL–no observed adverse effects level

LOAEL–lowest observed adverse effects level

Figure 8-1. Threshold soil concentrations for potential adverse effects for toxaphene, dieldrin, and DDTr at the North Shore Restoration Area under different total organic carbon levels

Tables

Table 2-1. Average abundances of bird species observed at the North Shore Restoration Area of Lake Apopka from August 15, 1998, to April 11, 1999

	Time Interval End Date								
Species	8/16/1998	9/13/1998	9/27/1998	10/11/1998	10/25/1998	11/8/1998	11/22/1998	12/6/1998	12/20/1998
Pied-billed grebe	20	108	200	133	420	441	621	546	295
American white pelican			216	96	123	130	661	1,005	377
Double-crested cormorant		3	2	9	98	321	159	171	126
Anhinga	4	6	12	6	19	23	24	23	25
Great blue heron	250	78	195	120	89	189	269	313	228
Great egret	750	358	670	398	853	1,245	1,280	976	488
Snowy egret	300	125	205	106	93	39	33	17	3
Little blue heron	70	15	6	6	4	7	5	3	9
Tricolored heron	60	41	35	6	3	5	7	8	12
Cattle egret	400	2,250	2,800	463	1,042	39	276	983	1,038
Green heron	10	9	11	8	2	5	4	3	5
Black-crowned night heron	1		12	8	37	5	18	4	4
White ibis	1,000	150	165	14	78	46	176	86	249
Glossy ibis	200	22	150	48	235	249	349	289	458
Wood stork	30	155	546	320	580	625	1,034	700	42
Green-winged teal					2	189	820	1,463	7,682
Mottled duck	20	53	104	30	41	21	51	13	39
Blue-winged teal	2	55	135	655	5,000	8,400	1,690	780	5,392
Northern shoveler		17	20	38	105	188	416	405	533
Ring-necked duck					4	226	474	7,221	2,660
Common moorhen	20	850	685	523	1,315	409	243	123	47
American coot		35	57	95	2,375	5,550	14,470	5,820	4,471
Black-bellied plover	35	34	4	10	19	133	167	306	183
Killdeer	90	23	12	6	52	16	81	276	222
Greater yellowlegs	10	60	70	50	233	341	176	88	205
Lesser yellowlegs	140	430	370	291	880	769	546	708	745
Least sandpiper	800	885	350	3	1,045	1,690	1,663	1,000	1,838
Short-billed dowitcher	350	385	315	84	303	579	180	30	
Long-billed dowitcher				18	18	195	628	946	1,095
Ring-billed gull				5	3	48	302	350	305
Forster's tern	90	323	47	20	4	58	268	230	202
Red-winged blackbird	60	1,600	2,200	545	113	181	724	443	603
Boat-tailed grackle	500	400	135	101	255	246	503	969	698

Table 2-1. (cont.)

	Time Interval End Date (cont.)							
Species	1/3/1999	1/17/1999	1/31/1999	2/14/1999	2/28/1999	3/14/1999	3/28/1999	4/11/1999
Pied-billed grebe	293	216	236	115	40			
American white pelican	1,938	1,990	2,920	2,186	2,190	5	18	6
Double-crested cormorant	44	20	21	23	39	35	38	69
Anhinga	22	18	13	9	15	6	3	9
Great blue heron	245	283	313	234	175	5	4	3
Great egret	429	578	702	510	210	1	1	1
Snowy egret	17	17	13	9	14			
Little blue heron	6	8	9	7	11			2
Tricolored heron	5	2	2	1				
Cattle egret	527	698	645	326	430	73	135	247
Green heron	5	12	10	8	8			
Black-crowned night heron	6	3	25	2	1			
White ibis	358	235	281	340	160	1	14	23
Glossy ibis	434	476	365	251	32		2	1
Wood stork	283	332	628	592	620	7	1	9
Green-winged teal	5,195	4,041	6,805	3,520	450	8		
Mottled duck	23	19	14	4			3	3
Blue-winged teal	388	1,122	3,873	1,543	50			
Northern shoveler	462	461	597	461	75			
Ring-necked duck	183	66	68	41	4			
Common moorhen	62	58	108	70	60			1
American coot	2,553	2,735	2,204	1,971	470			
Black-bellied plover	95	160	151	112	31	13	6	19
Killdeer	569	302	269	497	494	21	16	4
Greater yellowlegs	287	113	150	230	40			
Lesser yellowlegs	617	295	453	576	70	8		
Least sandpiper	1,862	1,070	1,350	1,128	90	30	85	36
Short-billed dowitcher								
Long-billed dowitcher	1,628	1,520	1,206	1,415	184			
Ring-billed gull	394	880	1,032	233	133	3	18	87
Forster's tern	71	51	58	48	11		3	1
Red-winged blackbird	977	502	509	383	265	183	415	225
Boat-tailed grackle	888	505	978	573	690	18	20	14

Table 2-2. Total number of dead and sick birds observed at the North Shore Restoration Area of Lake Apopka from December 12, 1998, through April 11, 1999, as recorded by SJRWMD and USFWS personnel

	SJRWMD		USFWS	
Species	Dead	Sick	Dead	Total
American white pelican	372	47	40	459
Great blue heron	7	3	39	49
Wood stork	21	2	16	39
Great egret	13	11	11 [a]	35
Black-crowned night heron	7	0	8 [b]	15
Ring-billed gull	9	2	6 [c]	11
Unknown species	0	0	8	8
Double-crested cormorant	5	0	1	6
White ibis	1	0	4	5
Blue-winged teal	0	0	3	3
Snowy egret	1	0	2	3
Yellow-crowned night heron	0	0	3	3
Boat-tailed grackle	2	0	0	2
Little blue heron	0	0	2	2
White egret	0	0	2	2
Eurasian curlew	0	0	1	1
Glossy ibis	0	0	1	1
Green heron	1	0	0	1
Mourning dove	0	0	1	1
Northern pintail	0	0	1	1
Osprey	0	0	1	1
Peregrine falcon	0	0	1	1
Sandpiper sp.	1	0	0	1
Short-billed dowitcher	0	0	1	1
White-faced ibis	0	0	1	1

Note: SJRWMD - St. Johns River Water Management District
USFWS - U.S. Fish and Wildlife Service

[a] Excludes 15 birds identified only as Egret *spp*.

[b] Excludes 2 birds identified only as Night-heron *spp*.

[c] Excludes 6 birds identified only as Gull *spp*.

Table 2-3. Biweekly estimates of abundance and infirmity rates for species most adversely affected by the mortality event at the North Shore Restoration Area of Lake Apopka

	American White Pelican		Wood Stork		Great Egret		Great Blue Heron	
Two-week interval ending	Census count	Dead or sick	Census count	Dead or sick	Census count	Dead or sick	Census count	Dead or sick
12/20/1998	377	43	42		488	1	228	
1/3/1999	1,938		283		429		245	
1/17/1999	1,990	58	332	6	578	2	283	2
1/31/1999	2,920	56	628	6	702	6	313	1
2/14/1999	2,186	220	592	6	510	8	234	2
2/28/1999	2,190	41	620	2	210	7	175	2
3/14/1999	5	26	7	14	1	3	5	15
03/28/99	18	9	1	3	1	8	4	20
04/11/99	6	6	9	2	1		3	7
Total dead or sick		459		39		35		49
Total dead or sick as a percentage of maximum census count		15.7%		6.2%		5.0%		15.7%

Table 4-1. Exposure parameters used in Lake Apopka food-web models

	White Pelican	Wood Stork	Double-Crested Cormorant	Great Blue Heron	Great Egret	American Coot	Sandpiper
Body weight (g)	5,400 [a]	2,375 [d]	1,675 [d]	2,300 [d,j]	1,000 [k]	642 [d]	25.9 [n]
IRf (kg-dw/kg-day)	0.08 [a]	0.044 [e]	0.048 [h]	0.044 [e]	0.045 [e]	0.069 [m]	0.154 [m]
IRs (% of IRf)	10 [b]	10 [b]	10 [b]	10 [b]	10 [b]	24 [b]	10 [b]
Prey size range (mm)	400 [c]	250 [f,g]	280 [h]	330 [j]	370 [l]	NA	NA

Note: NA - not applicable because fish are not part of the diet

[a] Evans and Knopf (1993).

[b] Beyer et al. (1987).

[c] Derby and Lovvorn (1997).

[d] Dunning (1993).

[e] Kushlan (1978).

[f] Kahl (1964).

[g] Ogden et al. (1976).

[h] Campo et al. (1993).

[i] Butler (1992).

[j] Alexander (1977).

[k] Palmer (1962).

[l] Willard (1977).

[m] Nagy (1987).

[n] Cooper (1994).

Table 4-2. Probability of maximum daily exposure to toxaphene by modeled bird populations (December 18, 1998)

Percentile	Pelican (mg/kg-day)	Sandpiper (mg/kg-day)	Coot (mg/kg-day)	Cormorant (mg/kg-day)	Stork (mg/kg-day)	Egret (mg/kg-day)	Great Blue Heron (mg/kg-day)
0	0.02	0.00	0.00	0.01	0.01	0.01	0.01
5	0.22	0.02	0.01	0.13	0.12	0.12	0.12
10	0.38	0.03	0.02	0.23	0.21	0.21	0.21
15	0.54	0.05	0.04	0.33	0.31	0.30	0.31
20	0.71	0.08	0.06	0.45	0.41	0.41	0.40
25	0.89	0.09	0.07	0.57	0.51	0.51	0.51
30	1.09	0.11	0.09	0.68	0.63	0.62	0.62
35	1.30	0.13	0.11	0.82	0.74	0.75	0.74
40	1.52	0.16	0.13	0.96	0.87	0.88	0.88
45	1.77	0.18	0.15	1.11	1.02	1.02	1.03
50	2.08	0.20	0.17	1.28	1.18	1.18	1.19
55	2.43	0.22	0.19	1.50	1.37	1.37	1.38
60	2.86	0.24	0.20	1.74	1.58	1.62	1.61
65	3.35	0.35	0.29	2.04	1.87	1.92	1.87
70	3.99	0.44	0.36	2.37	2.21	2.26	2.20
75	4.79	0.58	0.48	2.82	2.66	2.67	2.65
80	5.80	0.80	0.66	3.40	3.24	3.23	3.17
85	7.14	0.93	0.77	4.18	3.99	3.99	3.91
90	9.09	1.02	0.85	5.43	5.08	5.21	4.96
95	12.56	1.21	1.01	7.58	7.14	7.42	7.10
100	138.87	8.23	6.88	110.85	67.91	83.79	52.64

Table 4-3. Probability of maximum daily exposure to dieldrin by modeled bird populations (December 18, 1998)

Percentile	Pelican (mg/kg-day)	Sandpiper (mg/kg-day)	Coot (mg/kg-day)	Cormorant (mg/kg-day)	Egret (mg/kg-day)	Great Blue Heron (mg/kg-day)	Stork (mg/kg-day)
0	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.01	0.00	0.00	0.00	0.00	0.00	0.00
20	0.01	0.01	0.00	0.01	0.01	0.01	0.01
25	0.01	0.01	0.00	0.01	0.01	0.01	0.01
30	0.02	0.01	0.01	0.01	0.01	0.01	0.01
35	0.02	0.01	0.01	0.01	0.01	0.01	0.01
40	0.03	0.02	0.01	0.02	0.01	0.01	0.01
45	0.03	0.02	0.01	0.02	0.02	0.02	0.02
50	0.04	0.03	0.01	0.02	0.02	0.02	0.02
55	0.04	0.03	0.02	0.03	0.02	0.02	0.02
60	0.05	0.03	0.02	0.03	0.03	0.03	0.03
65	0.06	0.04	0.02	0.03	0.03	0.03	0.03
70	0.07	0.04	0.02	0.04	0.04	0.04	0.04
75	0.08	0.05	0.03	0.05	0.04	0.04	0.04
80	0.10	0.05	0.03	0.06	0.05	0.05	0.05
85	0.12	0.06	0.03	0.07	0.06	0.06	0.06
90	0.15	0.07	0.04	0.09	0.08	0.08	0.08
95	0.22	0.08	0.04	0.13	0.12	0.12	0.12
100	3.88	0.83	0.47	1.85	2.76	1.87	1.46

Table 4-4. Probability of maximum daily exposure to DDTr by modeled bird populations (December 18, 1998)

Percentile	Pelican (mg/kg-day)	Sandpiper (mg/kg-day)	Coot (mg/kg-day)	Cormorant (mg/kg-day)	Great Blue Heron (mg/kg-day)	Egret (mg/kg-day)	Stork (mg/kg-day)
0	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.01	0.00	0.00	0.01	0.01	0.01	0.01
10	0.02	0.00	0.00	0.01	0.01	0.01	0.01
15	0.03	0.00	0.00	0.02	0.02	0.02	0.02
20	0.04	0.00	0.00	0.02	0.02	0.02	0.02
25	0.04	0.00	0.00	0.03	0.03	0.03	0.02
30	0.05	0.01	0.01	0.03	0.03	0.03	0.03
35	0.06	0.01	0.01	0.04	0.04	0.04	0.04
40	0.07	0.01	0.01	0.04	0.04	0.04	0.04
45	0.09	0.01	0.01	0.05	0.05	0.05	0.05
50	0.10	0.01	0.01	0.06	0.06	0.06	0.06
55	0.12	0.01	0.01	0.07	0.07	0.07	0.07
60	0.14	0.02	0.02	0.08	0.08	0.08	0.08
65	0.17	0.02	0.02	0.10	0.09	0.09	0.09
70	0.20	0.02	0.02	0.12	0.10	0.11	0.11
75	0.24	0.03	0.03	0.14	0.12	0.13	0.13
80	0.29	0.04	0.04	0.17	0.15	0.16	0.16
85	0.37	0.05	0.04	0.22	0.18	0.20	0.20
90	0.49	0.07	0.06	0.29	0.24	0.27	0.26
95	0.75	0.09	0.09	0.43	0.35	0.40	0.40
100	45.98	3.85	3.69	14.29	18.89	49.83	22.49

Table 5-1. Summary of necropsy report findings for birds that died in association with the avian mortality event at the North Shore Restoration Area of Lake Apopka, 1998–1999

								Gross/Histopathology							
Case #	Species	DOD	Location	Pathologist	Condition	Diagnosis	GI Content	Cardiac	GI	Liver	Kidney	Spleen	CNS	Hematology	Other
226-98	AWPE	12/13/1998	Zellwood	Gaydos	Nut-good; Carc-poor	Suspect toxicosis	NR	NR	None detectable from post-mortem artifacts	None detectable from post-mortem artifacts	None detectable from post-mortem artifacts	NR	None detectable from post mortem artifacts	NR	None detectable from post-mortem artifacts
231-98	AWPE	12/17/1998	Zellwood	Gaydos	Nut-good; Carc-poor	Suspect toxicosis	NR	NR	None detectable from post-mortem artifacts	None detectable from post-mortem artifacts	Single granuloma found	NR	None detectable from post mortem artifacts	NR	Pulmonary congestion; muscle hemorrhages; surficial hemorrhage on thorax and abdomen
3-99	BAEG	12/27/1998	NR	Gaydos	Nut-good; Carc-poor	Undetermined	Fluid only; bird in captivity for 3 days	NR	None detectable from post-mortem artifacts	None detectable from post-mortem artifacts	None detectable from post-mortem artifacts	NR	None detectable from post mortem artifacts	NR	None detectable from post-mortem artifacts
16138-015	GREG	1/4/1999	28.38.516 x 81.34.275	Meteyer	Nut-poor; Carc-good	Dieldrin toxicosis	Unidentified dark green matter	Unremarkable/NR	Unremarkable/Mild inflammation	Unremarkable/ Unremarkable	Unremarkable/ Unremarkable	Unremarkable/mild atrophy; eosinophilic droplets in cytoplasm of sheathed arteries	Unremarkable/WM vacuolation of thalamus; no inflammation	No effect on plasma cholinesterase	Spinal fracture between T5 and T6 and pulmonary intercostal muscle hemorrhage; result of trauma; noted dehydration
1-99	AWPE	1/4/1999	Zellwood	Gaydos	Nut-good; Carc-good	Suspect toxicosis	NR	NR	Mild hemorrhage-intestinal serosa	Congestion-mod, lymphatic	Medulary mineralization-multifoci with lymph infiltration	NR	Numerous clusters of objects resembling amoebic trophozoites; no inflammation	NR	None
W99-201	AWPE	1/8/1999	Zellwood	Spalding	Nut-good; Carc-good	Suspected OC poisoning	NR	Hydropericardium-acute, sev; congestion	NR	Congestion	Nephrosis-acute, multifocal, mod-sev	Lymphoid depletion-sev	Pending	NR	Congestion-lungs, adrenals; peritonitis-chronic, mild, focal, granulomalous
W99-202	AWPE	1/8/1999	Zellwood	Spalding	Nut-good; Carc-good	Suspected OC poisoning	Fluid only	Hydropericardium-acute, sev; congestion	Esophagitis ulceratic-multifocal, mild, ascarid nematodes	Congestion	Nephrosis-acute, multifocal, mod-sev	Lymphoid depletion-sev	Pending	NR	Congestion-lungs, adrenals; peritonitis-chronic, mild, focal, granulomalous
Lab 1	BDEG	1/11/1999	NR	Stroud	Nut-fair; Carc-good; Convulsions; Clenched feet; Pellets present	OC Toxicosis	Stomach: dark granular/semi-liquic content; Colon: tarry feces	Normal/NR	Atrophied Sm. Intestine/ NR	Gall enlarged; liver rounded and swollen/ NR	Normal/NR	Congested and enlarged/NR	Cerebellum sinus congested or hemorrhagic/ NR	NR	Pulmonary: Normal/ NR
W99-203	AWPE	1/12/1999	Zellwood	Spalding	Nut-good; Carc-good	Suspected OC poisoning	NR	Congestion	NR	Congestion; hepatitis necrotic, acute, multifocal, mild	Nephrosis-acute, multifocal, mod-sev	Lymphoid depletion-sev	Pending	NR	Congestion-adrenals; peritonitis-chronic, mild, granulomalous
W99-204	WOST	1/13/1999	Zellwood	Spalding	Nut-poor; Carc-Moderate	Suspected OC poisoning	Muck	Hydropericardium-acute, sev; congestion	NR	NR	NR	Lymphoid depletion-sev	Pending	NR	Osteoarthritis-r hock; chronic, bacterial

Table 5-1. (cont.)

		Disease				
Case #	Species	Newcastle	Botulinum	Cholera	Parasites	Other
226-98	AWPE	NA	Positive bioassay	NA	Cecal worms-throughout GI, assumed post-mortem distribution	
231-98	AWPE	NA	Unable to test	NA	NR	
3-99	BAEG	NA	Unable to test		NR	None detectable from post-mortem artifacts
16138-015	GREG	NA	Negative	NR	NR	Liver: no bacterial growth
1-99	AWPE	NA	Negative	NA	Cecal worms throughout GI	Aeromonas sobria-liver, kidney, lung, intestine; Citrobacter freundii-liver, kidney, intestine; Shewanella putrefaciens heavy-lungs; Acinetobacter sp. mod-lung
W99-201	AWPE	Pos Titer	NR	NR	Nematodes-stomach; trematodes-small and large intestine; Schist eggs-duodenum	Liver-Aeromonas sobria
W99-202	AWPE	Neg Titer	Equivocal	NR	Nematodes-stomach; trematodes-small and large intestine	NR
Lab 1	BDEG	NA	NA	NA	NR	NR
W99-203	AWPE	Pos Titer	Equivocal	NR	Nematodes-stomach; trematodes-small and large intestine; cestodes-small and large intestine	NR
W99-204	WOST	Neg Titer	NR	NR	Nematodes-esophagus; trematodes-small and large intestine; cestodes-small and large intestine	NR

Table 5-1. (cont.)

Case #	Species	DOD	Location	Pathologist	Condition	Diagnosis	GI Content	Gross/Histopathology							
								Cardiac	GI	Liver	Kidney	Spleen	CNS	Hematology	Other
Lab 2	WAPE	1/22/1999			Nut-poor; Carc-good; Convulsions; feathers bile stained;	OC Toxicosis	Empty	Normal/NR	Atrophied Sm. intestine; fish spine abcess	Congested	Congested	Pale and atrophied	Autolyzed	NR	Enlarged adrenal glands
99-90	AWPE	1/23/1999	Ft. Myers	Cornish	Nut-good; Carc-good	Suspect toxicosis	NR	NR	Stomach-lining thickening	Congested; portal hepatitis	Congestion; multifocal necrotic lesions	Lymphoid depletion-mild	Cerebellar white matter-large aggregate vacuoles-(intramylegenic edema), neuropil-specific, dilated axonal, up to 20μm; intracytoplasmic vacuoles-grey matter of brain stem, neuronal degeneration and necrosis	NR	Congestion-lungs, necrotizing pneumonia; shrunken thymus
99-97	AWPE	1/23/1999	Sanibel Island	Cornish	Nut-good; Carc-good	Suspect toxicosis	NR	NR	Stomach-lining thickening	Congested; portal hepatitis	Congestion	Lymphoid depletion-mild	Cerebellar white matter-large aggregate vacuoles up to 20 μm, neuropil-specific, dilated axonal sheaths; intracytoplasmic vacuoles-grey matter of brain stem, neuronal degeneration and necrosis	NR	Congestion-lungs; shrunken thymus
Lab-5	AWPE	2/2/1999	Zellwood	Stroud	Nut-Fair; Carc-Euth; feathers contained brown/black oily substance	Suspect OC toxicosis	None	Myocardium mottled	Perforation of duodenum by fish spine; Duodenal obstructive enteritis (< 2mm)	Normal	Pale in color	Pale and atrophied with petechial hemorrhage	Brain normal	NR	Enlarged adrenal glands with yellowish color
W99-208	AWPE	2/2/1999	Silver Springs Shore	Spalding; initial exam by Rosenberg	Nut-Unknown; Carc-Formalin Fixed	Suspected OC poisoning	NR	NR	NR	Hepatitis-chronic, periportal, multifocal, mild atrophy	NR	Lymphoid depletion-mod	NR	NR	
W99-206	AWPE	2/3/1999	Silver Springs Shore	Spalding; initial exam by Rosenberg	Nut-Unknown; Carc-Formalin Fixed	Suspected OC poisoning	NR	NR	NR	NR	NR	Lymphoid depletion-sev	NR	NR	Sarcocystiasis-pectoral, mild
W99-209	AWPE	2/4/1999	Melborne Beach	Spalding	Nut-good; Carc-good	Suspected OC poisoning	NR	Petechial epicardium-hemorrhage, mild	Gastric ulcer-chronic, sev; intralesional bacteria	Hepatitis periportal/ multifocal hepatocellular necrosis; congestion	Nephrosis-acute, multifocal-sev	Lymphoid depletion-sev	NR	NR	
W99-210	AWPE	2/8/1999	Zellwood	Spalding	Nut-poor; Carc-good	Suspected OC poisoning	Emaciated; sev dehydration	NR	Gastric ulcer-sev; colitis-hemorrhagic	Hepatic atrophy-sev	Nephrosis-acute, multifocal, mild-mod	Lymphoid depletion-very sev	NR	NR	

Table 5-1. (cont.)

Case #	Species	Disease				
		Newcastle	Botulinum	Cholera	Parasites	Other
Lab 2	WAPE	NR	NR	NR	nematodes: stomach	
99-90	AWPE	NA	NR	NR	Nematodes-esophagus, stomach; flukes-kidney and GI	Lungs-mulitfocal inflammation with protozoal infiltration (toxoplasmosis); Clostridium perfringens: intestine, lung, liver
99-97	AWPE	NA	NR	NR	Nematodes-esophagus, stomach, flukes, kidney, and GI	Clostridium perfringens: intestine
Lab-5	AWPE	NR	NR	NR	nematodes: stomach	
W99-208	AWPE	NA	NR	NR	Schist eggs-liver	
W99-206	AWPE	NA	NR	NR	Trematodes-large intestine	NR
W99-209	AWPE	Pos Titer	NR	NR	Ascarid nematodes-esophagus and stomach; trematodes-kidney, large intestine, ceca/cloaca; Schist eggs-small and large intestine	Liver cultures-Clos. Difficile, Clos. sp., Fusobacterium necrophorum
W99-210	AWPE	Pos Titer	NR	NR	GI-enteritis, granulomatosis, chronic, cystic; trematodes-small intestine, necrotic debis; Schist eggs-lamina propria, cellulitis; cestode-subcutis, peritoneum, intestine; nematodes-stomach	

Table 5-1. (cont.)

								Gross/Histopathology							
Case #	Species	DOD	Location	Pathologist	Condition	Diagnosis	GI Content	Cardiac	GI	Liver	Kidney	Spleen	CNS	Hematology	Other
CP99-1239	AWPE	2/9/1999	Miami	Bossart	Nut-good; Carc-good	Undetermined	NR	NR	NR	Mottled and tan; vacuolar degeneration; cholestasis; cholangitis	Nephrosis; multifocal; pyelitis-chronic, multifocal, trematodiasis	Lymphoid depletion-mild	NR	NR	Lung-congestion and hemorrhage-mild; adrenal-lipid depletion; bursa-inflamed, necrotizing, acute, multifocal
W99-213	AWPE	2/11/1999	Zellwood	Spalding	Nut-good; Carc-30 min post mort	Suspected OC poisoning	Empty; mild dehydration	NR	Enteritis; hemorrhage, mild		Nephrosis-acute, multifocal, mod-sev	Lymphoid depletion and lymphopenia-sev	NR	Cell vol.-50.5%; lymphopenia-650μL; uric acid-31.6 mg/dL; K-5.86 meq/L; Cl-121 meq/L; Na-164 meq/L; albumin 1.9 g/L; CO2-11.5 meq/L; glucose-107 mg/dL; P-9.0 mg/dL	Adrenal-hyperplasia
Lab-6	AWPE	2/18/1999	NR	Stroud	Nut-Good; Carc-Good; feathers stained with bile; progressed muscle degeneration	Undetermined	None	Normal	Bile-stained mucosa in stomach	Normal	Congested	Pale and atrophied	Moderate congestion in brain	NR	Pulmonary normal; enlarged adrenal glands with yellowish color
16138-009	WOST	2/22/1999	28.38.722 x 81.34.277	Meteyer	Nut-good; Carc-euth; Seizures, tremors, extended feet and wings; dilated pupils; yellow watery droppings	Dieldrin toxicosis	Green granular material; chemical smell	Unremarkable/mild lymphocyte infiltration	Unremarkable/NR	NR/Subacute periportal inflammation; bile accumulation	Unremarkable/ Multifocal interstitial inflammation	NR/Tunica media of medium sized arteries (sic)	NR/Vacuolation in optic lobes; no inflammation	No effect on plasma cholinesterase	Pulmonary unremarkable
16138-010	WF Ibis	2/22/1999	NR	Meteyer	Nut-poor; Carc-euth; Seizures (head seizures), disoriented, blind; tremors, extended feet and wings; dilated pupils; yellow watery droppings	Dieldrin toxicosis	No ingesta	Unremarkable/ Unremarkable	Unremarkable/ Unremarkable	Unremarkable/ Unremarkable	Unremarkable/NR	Unremarkable/ Lymphocyte depletion	NR/Vacuolation in optic lobes; no inflammation	No effect on plasma cholinesterase	Pulmonary: moderate pneumoconiosis; single granuloma; eosinophilic droplets in walls of muscular arteries
Lab-10	AWPE	2/24/1999	Zellwood	Stroud	Nut-Good; Carc-Good	Undetermined	Empty	Normal	Gastric perforation by fish spine into peritoneum	Normal	Congested	Normal	Normal	NR	enlarged adrenal glands with yellowish color
Lab-11	WOST	2/24/1999	Zellwood	Stroud	Nut-Poor; Carc-Poor; Feathers caked with black sediment	Suspect OC toxicosis	Plant matter	Autolyzed	Autolyzed	Autolyzed	Autolyzed	Autolyzed	Autolyzed	NR	Pulmonary Autolyzed
Lab-12	AWPE	2/24/1999	Zellwood	Stroud	Nut-Fair; Carc-Good; feathers contained brown/black oily substance	Suspect OC toxicosis	Empty	Zonal myonecrosis	Bile-stained mucosa in stomach	Normal; Gall bladder distended	Congested	Pale in color; atrophied	Normal	NR	Pulmonary normal; enlarged adrenal glands with yellowish color
Lab-13	GREG	2/24/1999	Zellwood	Stroud	Nut-Good; Carc-Good; feathers contained brown/black oily substance; dehydrated	Suspect OC toxicosis	Empty	Normal	Bile-stained mucosa; atrophied intestine	Normal; Gall bladder distended	Pale in color	Pale in color; atrophied	Normal	NR	Pulmonary normal; enlarged adrenal glands with yellowish color

Table 5-1. (cont.)

		Disease				
Case #	Species	Newcastle	Botulinum	Cholera	Parasites	Other
CP99-1239	AWPE	NA	NR	NR	NR	
W99-213	AWPE	Pos Titer	NR	NR	Ascarid nematodes-stomach, mod; trematodes and nematodes-small intestine	
Lab-6	AWPE	NR	NR	NR	nematodes: stomach	
16138-009	WOST	NR	NR	NR	NR	Liver: no bacterial growth
16138-010	WF Ibis	NA	NA	NR	NR	Liver: no bacterial growth
Lab-10	AWPE	NR	NR	NR	nematodes: stomach	
Lab-11	WOST	NR	NR	NR	NR	
Lab-12	AWPE	NR	NR	NR	nematodes: stomach	
Lab-13	GREG	NR	NR	NR	NR	

Table 5-1. (cont.)

Case #	Species	DOD	Location	Pathologist	Condition	Diagnosis	GI Content	Gross/Histopathology							
								Cardiac	GI	Liver	Kidney	Spleen	CNS	Hematology	Other
64-99A	AWPE	2/25/1999	Zellwood	Gaydos	Nut-good; Carc-good	Suspect toxicosis	Sparse	NR	NR	NR	NR	NR	Numerous clusters of objects resembling protozan trophozoites; no inflammation	NR	Lung-granulomous inflammation
16138-014	GREG	3/1/1999	23.38.478 x 81.34.275	Meteyer	Nut-poor; Carc-fair	Dieldrin toxicosis	Small residues; no characterization	Unremarkable/NR	Unremarkable/ Unremarkable	Mildly congested/ Unremarkable	Unremarkable/ Unremarkable	Unremarkable/mild atrophy; eosinophilic droplets in cytoplasm of sheathed arteries	Unremarkable/ Unremarkable	NR	Pulmonary, thyroid unremarkable
16138-019	YCNH	3/3/1999	28.38.516 x 81.33.527	Meteyer	Nut-poor; Carc-euth; Sediment in mouth, feet, and hallux	Dieldrin toxicosis	Small amount of sediment	Unremarkable/NR	Unremarkable/NR	Unremarkable/ distended gall bladder/NR	Unremarkable/NR	Unremarkable/Mild atrophy	Unremarkable/WM vacuolation D brain stem; no inflammation	No effect on plasma cholinesterase	Pulmonary unremarkable
Lab 7	AWPE	3/3/1999	Zellwood	Stroud	Nut-Fair; Carc-Good; feathers stained with yellow pigment; progressed muscle degeneration	Undetermined	None	Hydropericardium	Bile-stained mucosa in stomach	Normal	Normal	Normal	Normal	NR	Pulmonary: fluid congestion around air sacs; enlarged adrenal glands with yellowish color
Lab-8	COEG	3/3/1999	NR	Stroud	Nut-Fair; Carc-Euth; feathers stained with yellow pigment; progressed muscle degeneration	Undetermined	Small amount of plant material	Normal	Bile-stained mucosa in stomach; intestinal atrophy	Normal	Normal	Normal	Normal	NR	Pulmonary normal
Lab-9	GBH	3/3/1999	Zellwood	Stroud	Nut-Poor; Carc-Good; progressed muscle degeneration	Undetermined	Unkown brown fluid; no food; mucous congestion in intestines	Hydropericardium	Gastric perforation by fish spine; Intestinal abcess at point of contact	Normal	Pale in color	NR	Normal	NR	Pulmonary: fluid congestion; enlarged adrenal glands with yellowish color
64-99B	GREG	3/4/1999	Zellwood	Gaydos	Nut-fair; Carc-fair	Suspect toxicosis	Sparse	None	NR	NR	NR	NR	NR	NR	No gross signs in lung or trachea
16188-001	BCNH	3/8/1999	NR	Meteyer	Nut-Poor; Carc-fair; Missing distal lower beak and intermandibular mucosa	Dieldrin toxicosis	Brown fluid	Pale in color/ Unremarkable	Unremarkable/ Granulomas in gizzard (parasital)	Irregular tan foci; Congested; mildly rounded; distended gall bladder	Unremarkable/ Unremarkable	Unremarkable/ Lymphocyte depletion	Unremarkable/ Vacuolated brain stem	No effect on plasma cholinesterase	Moderate pulmonary congestion; moderate colloid depletion in thyroid
16188-002	WOST	3/8/1999	Lake Prevatt	Meteyer	Nut-poor; Carc-good	Dieldrin toxicosis	Digested vegetation	Unremarkable/ Unremarkable	Unremarkable/ Perforated mucosa	Congested/ Periportal inflammation	Raised above margins; renal tubules prominent/ mild inflammation	Unremarkable/ Lymphocyte depletion	NR/ Unremarkable	No effect on plasma cholinesterase	Pulmonary: NR/ Unremarkable
16188-005	Eur Curlew	3/8/1999	28.39.696 x 81.35.566	Meteyer	Nut-poor; Carc-good	Dieldrin toxicosis	Sediment in mouth; undefined intestina contents	Unremarkable/ Unremarkable	Unremarkable/ Multifocal inflammation in lamina propria	Unremarkable/ Congested	Unremarkable/ Congested	Unremarkable/ Congested	Unremarkable/ Unremarkable	No effect on plasma cholinesterase	Pulmonary unremakable/ Multifocal inflammation adjacent to vessels
16188-006	BCNH	3/8/1999	Zellwood	Meteyer	Nut-poor; Carc-good; Pectoral muscle atrophy	Dieldrin toxicosis	NR	Unremarkable/NR	Unremarkable/ Unremarkable	Unremarkable/ Sinusoids poorly defined; granulomas and inflammation	Unremarkable/ Unremarkable	Unremarkable/ Unremarkable	NR/Unremarkable	No effect on plasma cholinesterase	Pulmonary: Unremarkable/ Hemorrhage in caudal pectoral muscle
W99-220	GBHE	3/25/1999	Zellwood	Spalding	Nut-good; Carc-good	Avian cholera	NR	Hemorrhagic epicarditis-acute, mild	NR	Necrosis-acute, mild	Nephrosis and glomerulonepritis-acute, mod	NR	NR	NR	Verminous peritonitis; bite wound-puncture thorax and abdomen

Table 5-1. (cont.)

Case #	Species	Disease				
		Newcastle	Botulinum	Cholera	Parasites	Other
64-99A	AWPE	NA	NR	NR	NR	
16138-014	GREG	NA	Negative	NR	Tapeworm-like parasite in RC pectoral muscle	Liver: no bacterial growth
16138-019	YCNH	NR	Positive	NR	Roundworm in stomach	Liver: no bacterial growth
Lab 7	AWPE	NR	NR	NR	nematodes: stomach	
Lab-8	COEG	NR	NR	NR	NR	
Lab-9	GBH	NR	NR	NR	nematodes and unidentified flukes: stomach	
64-99B	GREG	NA	NR	NR	Nematodes and trematodes-small intestine	
16188-001	BCNH	NR	NR	NR	NR	Liver negative for salmonella; positive fo Aeromonas hydrophilia (Gram-)
16188-002	WOST	NR	NR	NR	Mild acanthocephalan and schistosomes in intestines	Liver: no bacterial growth
16188-005	Eur Curlew	NR	NR	NR	light feather mites	Liver +ve for B. cereus
16188-006	BCNH	NR	NR	NR	NR	Liver: no bacterial growth
W99-220	GBHE	NA	NR	Pasteurella-septicemia and thrombosis, multifoci	Clinostomum-oral cavity	

Table 5-1. (cont.)

Case #	Species	DOD	Location	Pathologist	Condition	Diagnosis	GI Content	Gross/Histopathology							
								Cardiac	GI	Liver	Kidney	Spleen	CNS	Hematology	Other
W99-227	WOST	4/16/1999	Emerald Island	Spalding	Nut-good; Carc-mod.	Unknown	NR	NR	Peritonitis granulomalous-chronic, mild, noted plant material	NR	Undetermined	Lymphoid depletion-sev	NR	NR	
W99-105	AWPE	10/18/1999	Zellwood	Spalding	Nut-fair; Carc-mod	Unknown	Nematodes	NR	NR	NR	NR	NR	NR	NR	Hemorrhage on neck
W99-106	AWPE	10/18/1999	Zellwood	Spalding	Nut-fair; Carc-mod	Unknown	Nematodes	Hydropericardium	NR	NR	NR	NR	NR	NR	
W99-107	AWPE	NR	Zellwood	Spalding	Nut-good	Unknown	Nematodes	NR	Granulamatous enteritis	NR	NR	NR	NR	NR	Trauma at bill base
W99-108	AWPE	NR	Zellwood	Spalding	Nut-fair	Unknown	NR	Hydropericardium	NR	NR	NR	NR	NR	NR	
W99-296	AWPE	NR	FL Keys	Spalding	Nut-fair; Carc-fair	Unknown	Nematodes	NR	NR	NR	NR	NR	NR	NR	Abrasions, bilateral, carpi
W99-297	AWPE	NR	Lake City	Spalding	Nut-excellent; Carc-fair	Unknown	Fish	NR	NR	NR	NR	NR	NR	NR	Pododermatitis, arthritis, chronic, mild, phalanges, feet

Table 5-1. (cont.)

Case #	Species	Disease				
		Newcastle	Botulinum	Cholera	Parasites	Other
W99-227	WOST	NA	NR	NR	Schist enteritis-chronic, multifocal, mod, small intestine and mesentary; nematodes, trematodes, acanthocephlans-intestine	
W99-105	AWPE	NR	NR	NR	Ascarids-mild, stomach and intestine; trematodes, intestine; ectoparasites- mild, pouch lice	
W99-106	AWPE	NR	NR	NR	Ascarids-mild, stomach and intestine; trematodes, sm. intestine and kidney; ectoparasites-mild, pouch lice, mites	
W99-107	AWPE	NR	NR	NR	Acarids-mod, stomach and esophagus; schistosome eggs-presumptive, small intestine; trematodes-intestine; ectoparasites-mild, pouch lice, mice	
W99-108	AWPE	NR	NR	NR	Ascarids-mild; ectoparasites-pouch lice, chewing lice, mites	
W99-296	AWPE	NR	NR	NR	Ascarids, nematodes, cestodes, trematodes; ectoparasites-pouch lice, mites	
W99-297	AWPE	NR	NR	NR	Subcutis, mites, numerous; ascarids esophagus, stomach; cestodes, trematodes-intestine; ectoparasites-pouch lice, mites	

Note:

AWPE	- American white pelican	GI	- gastrointestinal
BCHNH	- black-crowned night heron	GREG	- great egret
BAEG	- bald eagle	NA	- not assessed
Carc	- carcass	NR	- not reported
CNS	- central nervous system	Nut	- nutritional
DOD	- date of death	OC	- organochlorine
Eur Curlew	- Eurasian curlew	WF Ibis	- white-faced ibis
euth	- euthanized	WOST	- wood stork
GBHE	- great blue heron	YNCH	- yellow-crowned night heron

Table 6-1. Evaluation of epidemiological criteria to determine if organochlorine pesticides were the cause of avian mortalities at the North Shore Restoration Area

Criterion	Evaluation
Probability	Indeterminate based on available data
Time order	Partially consistent with known events
Strength of association	Intermediate
Specificity of association	
Of effect	Intermediate
Of cause	Low
Consistency of association	Intermediate
Predictive performance	Indeterminate based on available data
Coherence	
Theoretical	Plausible
Biological	Plausible
Factual	Plausible, but not consistent with all known events

Table 8-1. Lipid-normalized concentrations of chemicals in prey fish corresponding to toxicity reference values derived for birds

Chemical	Avian TRV (mg/kg BW-day)	TRV Type	Reference for TRV	Avian Food Ingestion Rate (kg ww/kg BW-day)	Concentration in Prey (mg/kg ww)	Percent Lipid of Prey	Lipid-Normalized Concentration in Prey (mg/kg lipid)
Toxaphene	5.0	Unbounded NOAEL	Haseltine et al. (1980)	0.33	15.2	3.6	422
	3.3	NOAEL	Keith (1956)	0.33	10.0	3.6	278
	4.4	Unbounded LOAEL	Genelly and Rudd (1956)	0.33	13.3	3.6	369
	16.6	LOAEL	Keith (1956)	0.33	50.3	3.6	1,397
Dieldrin	0.078	Unbounded NOAEL	Mendenhall et al. (1983)	0.33	0.24	3.6	6.7
	1.03	Unbounded LOAEL	Genelly and Rudd (1956)	0.33	3.12	3.6	87
DDT	0.027	Unbounded LOAEL	Anderson et al. (1975)	0.33	0.08	3.6	2.2
	5.40	Unbounded LOAEL	Genelly and Rudd (1956)	0.33	16.4	3.6	456

Note: LOAEL - lowest-observed-adverse-effect level
NOAEL - no-observed-adverse-effect level
TRV - toxicity reference value

Table 8-2. Organochlorine pesticide concentrations in soils at four parts of the North Shore Restoration Area expressed on a dry-weight basis and as normalized to total organic carbon content of soils

Farm Region	Sample ID	4,4'-DDD (µg/kg)	4,4'-DDE (µg/kg)	4,4'-DDT (µg/kg)	4,4'-DDTr (µg/kg)	Dieldrin (µg/kg)	Toxaphene (µg/kg)	TOC (mg/kg)
Dry Weight Concentrations								
Duda West	DWFS199908171245-1	530	1,800	990	3,320	110	1,400	96,000
Duda West	DWFS199908171245-2	430	1,400	330	2,160	100	1,250	120,000
Duda West	DWFS199908171245-3	1,400	2,100	40,000	43,500	130	1,900	230,000
Duda West	DWFS199908171245-4	130	650	1,700	2,480	46	230	360,000
Duda West	DWFS199908171245-5	2,600	7,000	27,000	36,600	390	800	360,000
Duda West Average		1,018	2,590	14,004	17,612	155	1,116	233,200
Flow-way Cell G	FGFS199908249910301	1,500	2,500	170	4,170	350	9,200	50,000
Flow-way Cell G	FGFS199908249910301	1,600	2,700	275	4,575	530		25,000
Flow-way Cell G	FGFS199908249910302	1,500	1,900	220	3,620	220	7,800	240,000
Flow-way Cell G	FGFS199908249910303	1,500	2,000	270	3,770	120	7,400	220,000
Flow-way Cell G	FGFS199908249910304	1,300	1,700	130	3,130	270	8,700	270,000
Flow-way Cell G	FGFS199908249910305	870	1,900	110	2,880	280	6,100	310,000
Flow-way Cell G Average		1,378	2,117	196	3,691	295	7,840	185,833
Zellwood East	ZEFS199908171030-1	310	1,000	60	1,370	60	500	110,000
Zellwood East	ZEFS199908171030-2	150	500	29	679	29	360	110,000
Zellwood East	ZEFS199908171030-3	440	1,400	85	1,925	85	600	150,000
Zellwood East	ZEFS199908171030-4	670	2,300	165	3,135	165	850	93,000
Zellwood East	ZEFS199908171030-5	760	2,500	165	3,425	165	1,700	100,000
Zellwood East Average		466	1,540	101	2,107	101	802	112,600
Zellwood South	ZSFS199908170900-1	220	1,200	65	1,485	65	1,050	19,000
Zellwood South	ZSFS199908170900-2	220	1,300	65	1,585	65	1,550	210,000
Zellwood South	ZSFS199908170900-3	180	1,000	70	1,250	70	1,700	250,000
Zellwood South	ZSFS199908170900-4	190	950	80	1,220	80	1,000	230,000
Zellwood South	ZSFS199908170900-5	60	940	60	1,060	60	750	230,000
Zellwood South Average		174	1,078	68	1,320	68	1,210	187,800
TOC-Normalized Concentrations								
Duda West	DWFS199908171245-1	5,521	18,750	10,313	34,583	1,146	14,583	
Duda West	DWFS199908171245-2	3,583	11,667	2,750	18,000	833	10,417	
Duda West	DWFS199908171245-3	6,087	9,130	173,913	189,130	565	8,261	
Duda West	DWFS199908171245-4	361	1,806	4,722	6,889	128	639	
Duda West	DWFS199908171245-5	7,222	19,444	75,000	101,667	1,083	2,222	
Duda West Average		4,555	12,159	53,340	70,054	751	7,224	

Table 8-2. (cont.)

Farm Region	Sample ID	4,4'-DDD (µg/kg)	4,4'-DDE (µg/kg)	4,4'-DDT (µg/kg)	4,4'-DDTr (µg/kg)	Dieldrin (µg/kg)	Toxaphene (µg/kg)	TOC (mg/kg)
Flow-way Cell G	FGFS199908249910301	30,000	50,000	3,400	83,400	7,000	184,000	
Flow-way Cell G	FGFS199908249910301	64,000	108,000	11,000	183,000	21,200		
Flow-way Cell G	FGFS199908249910302	6,250	7,917	917	15,083	917	32,500	
Flow-way Cell G	FGFS199908249910303	6,818	9,091	1,227	17,136	545	33,636	
Flow-way Cell G	FGFS199908249910304	4,815	6,296	481	11,593	1,000	32,222	
Flow-way Cell G	FGFS199908249910305	2,806	6,129	355	9,290	903	19,677	
Flow-way Cell G Average		19,115	31,239	2,897	53,250	5,261	60,407	
Zellwood East	ZEFS199908171030-1	2,818	9,091	545	12,455	545	4,545	
Zellwood East	ZEFS199908171030-2	1,364	4,545	264	6,173	264	3,273	
Zellwood East	ZEFS199908171030-3	2,933	9,333	567	12,833	567	4,000	
Zellwood East	ZEFS199908171030-4	7,204	24,731	1,774	33,710	1,774	9,140	
Zellwood East	ZEFS199908171030-5	7,600	25,000	1,650	34,250	1,650	17,000	
Zellwood East Average		4,384	14,540	960	19,884	960	7,592	
Zellwood South	ZSFS199908170900-1	11,579	63,158	3,421	78,158	3,421	55,263	
Zellwood South	ZSFS199908170900-2	1,048	6,190	310	7,548	310	7,381	
Zellwood South	ZSFS199908170900-3	720	4,000	280	5,000	280	6,800	
Zellwood South	ZSFS199908170900-4	826	4,130	348	5,304	348	4,348	
Zellwood South	ZSFS199908170900-5	261	4,087	261	4,609	261	3,261	
Zellwood South Average		2,887	16,313	924	20,124	924	15,411	

Note: TOC - total organic carbon

Table 8-3. Organochlorine pesticide concentrations in fish collected at four parts of the North Shore Restoration Area expressed on a wet-weight basis

Farm Region	Species	Sample ID	4,4'-DDD (µg/kg)	4,4'-DDE (µg/kg)	4,4'-DDT (µg/kg)	4,4'-DDTr (µg/kg)	Dieldrin (µg/kg)	Toxaphene (µg/kg)	Lipid (percent)	Solids (percent)
Duda West	Tilapia	DWFF01 TILAPIA	420	2,200	38	2,658	5	360	4.1	25
Duda West	Tilapia	DWFF02 TILAPIA	470	2,400	33	2,903	5	340	4	24
Duda West	Tilapia	DWFF03 TILAPIA	280	1,700	27	2,007	5	250	3.4	26
Duda West	Tilapia	DWFF04 TILAPIA	150	890	13	1,053	5	125	1.4	22
Duda West	Tilapia	DWFF05 TILAPIA	160	1,000	5	1,165	5	125	1.8	26
Duda West Tilapia Average			296	1,638	23	1,957	5	240	3	25
Flow-way Cell G	Tilapia	FGFF01TILAPIA	1,700	3,100	5	4,805	60	2,700	5.7	25.6
Flow-way Cell G	Tilapia	FGFF02TILAPIA	1,600	3,600	5	5,205	29	2,500	7	25.7
Flow-way Cell G	Tilapia	FGFF03TILAPIA	1,500	3,400	5	4,905	39	2,700	5.9	25.4
Flow-way Cell G	Tilapia	FGFF04TILAPIA	2,200	6,700	5	8,905	99	3,400	7	27.5
Flow-way Cell G	Tilapia	FGFF05TILAPIA	1,400	3,000	12.5	4,413	59	2,600	5.9	25.6
Flow-way Cell G	Tilapia	FGFF06TILAPIA	930	2,000	12.5	2,943	50	2,100	4	24.5
Flow-way Cell G Tilapia Average			1,555	3,633	8	5,196	56	2,667	6	26
Zellwood East	Tilapia	ZEFF01 TILAPIA	1,400	6,400	25	7,825	180	1,400	1.7	25
Zellwood East	Tilapia	ZEFF02 TILAPIA	240	2,900	25	3,165	25	310	0.62	24
Zellwood East	Tilapia	ZEFF03 TILAPIA	370	2,400	25	2,795	25	480	0.91	23
Zellwood East	Tilapia	ZEFF04 TILAPIA	280	2,400	25	2,705	25	320	0.71	24
Zellwood East	Tilapia	ZEFF05 TILAPIA	530	3,900	25	4,455	25	500	0.8	24
Zellwood East Tilapia Average			564	3,600	25	4,189	56	602	1	24
Zellwood South	Tilapia	ZSFF01TILAPIA	54	540	5	599	5	125	1.2	23
Zellwood South	Tilapia	ZSFF02 TILAPIA	120	1,300	13	1,433	12	125	2.5	24
Zellwood South	Tilapia	ZSFF03 TILAPIA	61	870	5	936	5	125	1.6	23
Zellwood South	Tilapia	ZSFF04 TILAPIA	71	1,000	11	1,082	5	125	1.8	22
Zellwood South	Tilapia	ZSFF05 TILAPIA	33	370	5	408	5	125	1.1	21
Zellwood South Tilapia Average			68	816	8	892	6	125	2	23
Duda West	Bullhead	DWFF01 BULLHEAD	580	6,400	24	7,004	5	530	2.5	23
Duda West	Bullhead	DWFF02 BULLHEAD	1,400	8,200	25	9,625	25	1,400	6.5	28
Duda West	Bullhead	DWFF03 BULLHEAD	450	5,400	25	5,875	25	270	1.5	22
Duda West	Bullhead	DWFF04 BULLHEAD	340	2,300	25	2,665	25	290	1.9	23
Duda West	Bullhead	DWFF05 BULLHEAD	620	5,900	25	6,545	25	460	3	23
Duda West Bullhead Average			678	5,640	25	6,343	21	590	3	24
Zellwood South	Bullhead	ZSFF06 BULLHEAD	240	5,900	11	6,151	23	780	3.8	26
Duda West	Crappie	DWFF01 CRAPPIE	540	6,500	30	7,070	5	440	3.2	30
Duda West	Crappie	DWFF02 CRAPPIE	680	7,100	44	7,824	5	490	3.8	30
Duda West	Crappie	DWFF03 CRAPPIE	300	9,200	53	9,553	5	530	3.3	27
Duda West	Crappie	DWFF04 CRAPPIE	310	9,000	57	9,367	5	520	3.7	28

Table 8-3. (cont.)

Farm Region	Species	Sample ID	4,4'-DDD (µg/kg)	4,4'-DDE (µg/kg)	4,4'-DDT (µg/kg)	4,4'-DDT-r (µg/kg)	Dieldrin (µg/kg)	Toxaphene (µg/kg)	Lipid (percent)	Solids (percent)
Duda West	Crappie	DWFF05 CRAPPIE	180	3,800	18	3,998	5	125	3.4	30
Duda West	Crappie	DWFF06 CRAPPIE	30	2,600	5	2,635	5	125	0.55	30
Duda West Crappie Average			340	6,367	35	6,741	5	372	3	29
Duda West	Shad	DWFF01 SHAD	780	4,300	47	5,127	5	750	4.7	26
Duda West	Shad	DWFF02 SHAD	1,100	5,700	40	6,840	5	1,000	7.2	28
Duda West Shad Average			940	5,000	44	5,984	5	875	6	27
Zellwood East	Gambusia	ZEFF01 GAMBUSIA	650	5,300	25	5,975	92	630	2.8	16

Table 8-4. Organochlorine pesticide concentrations in fish collected at four parts of the North Shore Restoration Area expressed on a lipid-normalized basis

Farm Region	Species	Sample ID	4,4'-DDD (µg/kg lipid)	4,4'-DDE (µg/kg lipid)	4,4'-DDT (µg/kg lipid)	4,4'-DDD-r (µg/kg lipid)	Dieldrin (µg/kg lipid)	Toxaphene (µg/kg lipid)
Duda West	Tilapia	DWFF01 TILAPIA	10,244	53,659	927	64,829	122	8,780
Duda West	Tilapia	DWFF02 TILAPIA	11,750	60,000	825	72,575	125	8,500
Duda West	Tilapia	DWFF03 TILAPIA	8,235	50,000	794	59,029	147	7,353
Duda West	Tilapia	DWFF04 TILAPIA	10,714	63,571	929	75,214	357	8,929
Duda West	Tilapia	DWFF05 TILAPIA	8,889	55,556	278	64,722	278	6,944
Duda West Tilapia Average			9,966	56,557	750	67,274	206	8,101
Flow-way Cell G	Tilapia	FGFF01TILAPIA	29,825	54,386	88	84,298	1,053	47,368
Flow-way Cell G	Tilapia	FGFF02TILAPIA	22,857	51,429	71	74,357	414	35,714
Flow-way Cell G	Tilapia	FGFF03TILAPIA	25,424	57,627	85	83,136	661	45,763
Flow-way Cell G	Tilapia	FGFF04TILAPIA	31,429	95,714	71	127,214	1,414	48,571
Flow-way Cell G	Tilapia	FGFF05TILAPIA	23,729	50,847	212	74,788	1,000	44,068
Flow-way Cell G	Tilapia	FGFF06TILAPIA	23,250	50,000	313	73,563	1,250	52,500
Flow-way Cell G Tilapia Average			26,085	60,001	140	86,226	965	45,664
Zellwood East	Tilapia	ZEFF01 TILAPIA	82,353	376,471	1,471	460,294	10,588	82,353
Zellwood East	Tilapia	ZEFF02 TILAPIA	38,710	467,742	4,032	510,484	4,032	50,000
Zellwood East	Tilapia	ZEFF03 TILAPIA	40,659	263,736	2,747	307,143	2,747	52,747
Zellwood East	Tilapia	ZEFF04 TILAPIA	39,437	338,028	3,521	380,986	3,521	45,070
Zellwood East	Tilapia	ZEFF05 TILAPIA	66,250	487,500	3,125	556,875	3,125	62,500
Zellwood East Tilapia Average			53,482	386,695	2,979	443,156	4,803	58,534
Zellwood South	Tilapia	ZSFF01TILAPIA	4,500	45,000	417	49,917	417	10,417
Zellwood South	Tilapia	ZSFF02 TILAPIA	4,800	52,000	520	57,320	480	5,000
Zellwood South	Tilapia	ZSFF03 TILAPIA	3,813	54,375	313	58,500	313	7,813
Zellwood South	Tilapia	ZSFF04 TILAPIA	3,944	55,556	611	60,111	278	6,944
Zellwood South	Tilapia	ZSFF05 TILAPIA	3,000	33,636	455	37,091	455	11,364
Zellwood South Tilapia Average			4,011	48,113	463	52,588	388	8,307
Duda West	Bullhead	DWFF01 BULLHEAD	23,200	256,000	960	280,160	200	21,200
Duda West	Bullhead	DWFF02 BULLHEAD	21,538	126,154	385	148,077	385	21,538
Duda West	Bullhead	DWFF03 BULLHEAD	30,000	360,000	1,667	391,667	1,667	18,000
Duda West	Bullhead	DWFF04 BULLHEAD	17,895	121,053	1,316	140,263	1,316	15,263
Duda West	Bullhead	DWFF05 BULLHEAD	20,667	196,667	833	218,167	833	15,333
Duda West Bullhead Average			22,660	211,975	1,032	235,667	880	18,267

Table 8-4. (cont.)

Farm Region	Species	Sample ID	4,4'-DDD (µg/kg lipid)	4,4'-DDE (µg/kg lipid)	4,4'-DDT (µg/kg lipid)	4,4'-DDD-r (µg/kg lipid)	Dieldrin (µg/kg lipid)	Toxaphene (µg/kg lipid)
Zellwood South	Bullhead	ZSFF06 BULLHEAD	6,316	155,263	289	161,868	605	20,526
Duda West	Crappie	DWFF01 CRAPPIE	16,875	203,125	938	220,938	156	13,750
Duda West	Crappie	DWFF02 CRAPPIE	17,895	186,842	1,158	205,895	132	12,895
Duda West	Crappie	DWFF03 CRAPPIE	9,091	278,788	1,606	289,485	152	16,061
Duda West	Crappie	DWFF04 CRAPPIE	8,378	243,243	1,541	253,162	135	14,054
Duda West	Crappie	DWFF05 CRAPPIE	5,294	111,765	529	117,588	147	3,676
Duda West	Crappie	DWFF06 CRAPPIE	5,455	472,727	909	479,091	909	22,727
Duda West Crappie Average			10,498	249,415	1,113	261,026	272	13,861
Duda West	Shad	DWFF01 SHAD	16,596	91,489	1,000	109,085	106	15,957
Duda West	Shad	DWFF02 SHAD	15,278	79,167	556	95,000	69	13,889
Duda West Shad Average			15,937	85,328	778	102,043	88	14,923
Zellwood East	Gambusia	ZEFF01 GAMBUSIA	23,214	189,286	893	213,393	3,286	22,500

Table 8-5. Biota-sediment accumulation factors for fish collected at four parts of the North Shore Restoration Area

Farm Region	Species	Sample ID	4,4'-DDD	4,4'-DDE	4,4'-DDT	4,4'-DDT-r	Dieldrin	Toxaphene
Duda West	Tilapia	DWFF01 TILAPIA	2.2	4.4	0.02	0.9	0.16	1.2
Duda West	Tilapia	DWFF02 TILAPIA	2.6	4.9	0.02	1.0	0.17	1.2
Duda West	Tilapia	DWFF03 TILAPIA	1.8	4.1	0.01	0.8	0.20	1.0
Duda West	Tilapia	DWFF04 TILAPIA	2.4	5.2	0.02	1.1	0.5	1.2
Duda West	Tilapia	DWFF05 TILAPIA	2.0	4.6	0.01	0.9	0.4	1.0
Duda West Tilapia Average			2.2	4.7	0.01	1.0	0.3	1.1
Flow-way Cell G	Tilapia	FGFF01TILAPIA	1.6	1.7	0.03	1.6	0.20	0.8
Flow-way Cell G	Tilapia	FGFF02TILAPIA	1.2	1.6	0.02	1.4	0.08	0.6
Flow-way Cell G	Tilapia	FGFF03TILAPIA	1.3	1.8	0.03	1.6	0.13	0.8
Flow-way Cell G	Tilapia	FGFF04TILAPIA	1.6	3.1	0.02	2.4	0.27	0.8
Flow-way Cell G	Tilapia	FGFF05TILAPIA	1.2	1.6	0.07	1.4	0.19	0.7
Flow-way Cell G	Tilapia	FGFF06TILAPIA	1.2	1.6	0.11	1.4	0.24	0.9
Flow-way Cell G Tilapia Average			1.4	1.9	0.05	1.6	0.18	0.8
Zellwood East	Tilapia	ZEFF01 TILAPIA	18.8	25.9	1.5	23.1	11.0	10.8
Zellwood East	Tilapia	ZEFF02 TILAPIA	8.8	32.2	4.2	25.7	4.2	6.6
Zellwood East	Tilapia	ZEFF03 TILAPIA	9.3	18.1	2.9	15.4	2.9	6.9
Zellwood East	Tilapia	ZEFF04 TILAPIA	9.0	23.2	3.7	19.2	3.7	5.9
Zellwood East	Tilapia	ZEFF05 TILAPIA	15.1	33.5	3.3	28.0	3.3	8.2
Zellwood East Tilapia Average			12.2	26.6	3.1	22.3	5.0	7.7
Zellwood South	Tilapia	ZSFF01TILAPIA	1.6	2.8	0.5	2.5	0.5	0.7
Zellwood South	Tilapia	ZSFF02 TILAPIA	1.7	3.2	0.6	2.8	0.5	0.3
Zellwood South	Tilapia	ZSFF03 TILAPIA	1.3	3.3	0.3	2.9	0.3	0.5
Zellwood South	Tilapia	ZSFF04 TILAPIA	1.4	3.4	0.7	3.0	0.3	0.5
Zellwood South	Tilapia	ZSFF05 TILAPIA	1.0	2.1	0.5	1.8	0.5	0.7
Zellwood South Tilapia Average			1.4	2.9	0.5	2.6	0.4	0.5
Duda West	Bullhead	DWFF01 BULLHEAD	5.1	21.1	0.02	4.0	0.3	2.9
Duda West	Bullhead	DWFF02 BULLHEAD	4.7	10.4	0.01	2.1	0.5	3.0
Duda West	Bullhead	DWFF03 BULLHEAD	6.6	29.6	0.03	5.6	2.2	2.5
Duda West	Bullhead	DWFF04 BULLHEAD	3.9	10.0	0.02	2.0	1.8	2.1
Duda West	Bullhead	DWFF05 BULLHEAD	4.5	16.2	0.02	3.1	1.1	2.1
Duda West Bullhead Average			5.0	17.4	0.02	3.4	1.2	2.5

Table 8-5. (cont.)

Farm Region	Species	Sample ID	4,4'-DDD	4,4'-DDE	4,4'-DDT	4,4'-DDT-r	Dieldrin	Toxaphene
Zellwood South	Bullhead	ZSFF06 BULLHEAD	2.2	9.5	0.3	8.0	0.7	1.3
Duda West	Crappie	DWFF01 CRAPPIE	3.7	16.7	0.02	3.2	0.21	1.9
Duda West	Crappie	DWFF02 CRAPPIE	3.9	15.4	0.02	2.9	0.18	1.8
Duda West	Crappie	DWFF03 CRAPPIE	2.0	22.9	0.03	4.1	0.20	2.2
Duda West	Crappie	DWFF04 CRAPPIE	1.8	20.0	0.03	3.6	0.18	1.9
Duda West	Crappie	DWFF05 CRAPPIE	1.2	9.2	0.01	1.7	0.20	0.5
Duda West	Crappie	DWFF06 CRAPPIE	1.2	38.9	0.02	6.8	1.2	3.1
Duda West Crappie Average			2.3	20.5	0.02	3.7	0.4	1.9
Duda West	Shad	DWFF01 SHAD	3.6	7.5	0.02	1.6	0.14	2.2
Duda West	Shad	DWFF02 SHAD	3.4	6.5	0.01	1.4	0.09	1.9
Duda West Shad Average			3.5	7.0	0.01	1.5	0.12	2.1
Zellwood East	Gambusia	ZEFF01 GAMBUSIA	5.3	13.0	0.9	10.7	3.4	3.0
Average All Fish (excluding Zellwood East)			**2.5**	**9.4**	**0.1**	**2.6**	**0.4**	**1.4**