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## **MARINE BIRD DEATHS AT LOS ANGELES ZOO**

During the spring of 1976, nearly the entire collections of Brandt's cormorants (*Phalacrocorax penicillatus*), guanay cormorants (*P. bougainvillii*), and California gulls (*Larus californicus*) in the Los Angeles County Zoo died after exhibiting signs of pesticide poisoning. The possibility that this occurrence was linked with DDT in municipal wastewater was suggested; therefore, the Project conducted a limited investigation to determine if this pollutant could have been the cause of death for these specimens of marine birds. The study revealed that levels of DDT residues in the specimens were within the range that had been found to be lethal to American kestrels in laboratory feeding studies. The zoo birds apparently were exposed to the DDT when they were fed fish collected in the general vicinity of Palos Verdes Peninsula.

DDT contamination of the Southern California Bight by Los Angeles County municipal wastewater has been well documented over the last decade (MacGregor 1974; Young et al. 1976). The dominant source appears to have been industrial wastes released by Montrose Chemical Company to Los Angeles County's Joint Water Pollution Control Plant (JWPCP) sewage system before 1970 and discharged some 3 km off the Palos Verdes Peninsula. Although this input to the sewer system stopped in 1970, highly contaminated sediments that remained in the system have slowly released more DDT, and the annual emissions of this substance have only gradually declined—from 22 metric tons in 1971 to about 1 metric ton in 1976. A 1972 study showed that approximately 200 metric tons of DDT residues remained in the upper 30 cm of bottom sediments in a 50 sq km area near the JWPCP discharge; this reservoir appears to have led to persistently high DDT levels in at least one fish species living on these sediments (Young et al. 1976).

The history of the bird deaths was reported to us by Zoo Veterinarian Dr. Gerald Ezra. Between 22 May and 24 June 1976, sixteen California gulls and five cormorants died at the zoo. These birds were all from the same exhibit within the aquatics section of the zoo; the deaths left only one surviving cormorant in that exhibit. The gulls had been brought to the zoo in 1969 to recover from oil damage. The cormorants had been in captivity since 1970 and had been shipped from the San Francisco Zoo to Los Angeles in 1971.

The first gull death was on 22 May and the other mortalities occurred relatively uniformly through 10 June. Four of the cormorants died between 12 and 15 June; the final death occurred on 24 June. Clinically, the birds first exhibited a loss of appetite, and within 1 or 2 days, began trembling mildly and died after a few minutes of muscle spasms. Atropine, antibiotics, and

supportive therapy did not alter the course of the disease. Necropsy findings revealed no common pathology except a low nutritional status and nearly empty digestive tracts. Histological examination by Dr. Erwin Howard, Los Angeles Department of Public Health Services, showed no significant pathology except vascular dilatation and collapse and congestion in many organs. There was no evidence of infectious disease.

All of the birds that died had been kept in the same exhibit since their arrival at the zoo, and all had been fed exclusively on a diet of queenfish (*Seriphus politus*). Another collection of marine birds at the zoo, brown pelicans (*Pelicanus occidentalis*), are fed surf smelt (*Hypomesus pretiosus*); there were no deaths in this collection characterized by the symptoms described above. Queenfish are caught commercially off Los Angeles, and the fish fed to the affected birds had been supplied to the zoo for several years by a fish company in San Pedro. In contrast, surf smelt are caught commercially only north of Point Conception.

When Dr. Howard's histological examination revealed no evidence of infection, the Project was asked to investigate the possibility of chlorinated hydrocarbon poisoning. Unfortunately, only a few samples remained in the frozen state; these were brain, liver and breast muscle from one cormorant and one gull, breast muscle from two other gulls, and a composite of liver tissue obtained from three other birds that had died in spasms. With the assistance of Robert Johnson of the Project, we analyzed all of the available samples in triplicate for identifiable chlorinated hydrocarbons, using electron-capture gas chromatography. Corresponding analyses were conducted on two wild pelicans (provided by Al Lohman, Radio Station KFI) that had died of fish line strangulation in northern Santa Monica Bay during summer 1976. We also analyzed 6 to 7 whole specimens from each of two different lots of frozen queenfish that had been fed to the affected birds during their captivity, as well as 4 whole surf smelt from the zoo's supply of food for the unaffected pelican collection. Because mercury poisoning has been implicated in mortality of birds from a number of countries in the Northern Hemi-sphere, Robert Eganhouse analyzed single composites of most of our samples for total mercury by cold-vapor atomic absorption spectrometry. Our analytical procedures have been reported in previous Project publications.

The results of our study are presented in Tables 1 through 3. These data show that the brain and liver tissues of the zoo birds contained concentrations of total DDT that were two orders of magnitude above the mean values for the wild pelican specimens. On the average, p,p'-DDE constituted more than 90 percent of the total DDT values obtained in the various sample classes. Dr. Robert Risebrough (University of California at Berkeley) pointed out that the concentrations of p,p'-DDE in the brain samples from the gulls and cormorants ( $430 \pm 10$  and  $220 \pm 8$  ppm) were very similar to those measured by Porter and Wiemeyer (1972) in the brains of two kestrels (301 and 212 ppm) that had died in tremors after being maintained for 14 to 16 months on a diet containing p,p'-DDE. The p,p'-DDE levels in the gull and cormorant livers and in the liver composites sample ( $3,100 \pm 300$  ppm,  $750 \pm 65$  ppm, and 800

$\pm 200$  ppm) were considerably greater than those found in the kestrels (130 to 250 ppm). Further, the average concentrations of p,p'-DDE in the two batches of queenfish from the zoo lockers were  $3.1 \pm 0.7$  and  $4.2 \pm 1.1$  ppm (Table 2); the diet that killed the kestrels contained 2.8 ppm p,p'-DDE. In contrast, the samples of surf smelt used to feed the unaffected zoo pelicans averaged  $0.018 \pm 0.002$  ppm p,p'-DDE. These results strongly implicate the residue of DDT in the queenfish in the deaths of the captive gulls and cormorants.

An explanation for the sudden deaths of the zoo birds, after what was probably a gradual buildup of DDT residues from the continual ingestion of contaminated queenfish, can be inferred from the work of Porter and Wiemeyer (1972). These authors pointed out that seasonal weight losses and depletion of fat reserves caused by reproductive stresses can release lipid-associated toxicants, such as chlorinated hydrocarbons, into the bloodstream, where they are transferred to other sites, including the brain. When levels in this vital organ reach a critical peak—apparently in the range of 200 to 400 ppm—the resultant neurological damage is fatal.

The data in Table 1 show that another type of chlorinated hydrocarbon, the polychlorinated biphenyls, also had been accumulated by the zoo birds; concentrations of total PCB in the liver (140 to 500 ppm) and brain (49 to 66 ppm) tissues generally were two orders of magnitude above those found in the wild pelican specimens (0.4 to 3.0 and 0.2 to 1.6 ppm, respectively). However, these PCB levels were an order of magnitude below those of total DDT in the affected birds. Thus, although the PCB's may have contributed to the bird deaths, the evidence presented above suggests that the DDE was the primary cause. This conclusion is strengthened by the fact that there were no large differences between concentrations of mercury in the zoo bird and wild pelican tissues, although average concentrations of this trace metal in the former specimens generally were 2 to 3 times those measured in the latter.

Over the last 6 years, we have analyzed many hundreds of samples from the Southern California Bight for DDT and PCB residues. As a result, we have come to recognize the distinct pattern obtained on gas chromatograms of tissues of fish and other organisms collected off the Palos Verdes Peninsula. The fact that the queenfish samples all reflected this pattern suggested to us that they had been living in this region. A subsequent review of fish lot numbers by Larry Espinosa (California Department of Fish and Game) and Scott Winters (University of California at Irvine) indicated that these specimens very probably had been caught during 1975-76 within about 20 km of the JWPCP outfalls. These results are yet another indication that, despite termination of the dominant DDT input to the JWPCP municipal wastewater system in 1970, potentially toxic levels of DDT residues are still found in certain fishes occupying the discharge region off the Palos Verdes Peninsula.

## REFERENCES

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Porter, R.D., and S.N. Wiemeyer. 1972. DDE at low dietary levels kills captive American kestrels. *Bull. Environ. Contam. Toxicol.* 8:193.

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**Table 1. Mean concentrations (mg/wet kg,  $\pm$  standard error) of chlorinated hydrocarbons and total mercury in gulls and cormorants that died in convulsions at the Los Angeles zoo and in wild pelicans that died accidentally in Santa Monica Bay. Values, which are blank and recovery-corrected, are based on analysis of three subsamples of each tissue.**

	p,p'-DDE	Total DDT	1242 PCB	1254 PCB	Total Mercury*
<b>ZOO BIRDS</b>					
Gull 1					
Breast	230 $\pm$ 6	240 $\pm$ 6	4.4 $\pm$ 0.5	25 $\pm$ 3	0.829
Liver	3,100 $\pm$ 300	3,200 $\pm$ 400	61 $\pm$ 15	440 $\pm$ 70	1.08
Brain	430 $\pm$ 10	440 $\pm$ 15	12 $\pm$ 1	54 $\pm$ 7	0.404
Gull 2					
Breast	210 $\pm$ 9	220 $\pm$ 10	3.9 $\pm$ 0.3	30 $\pm$ 5	—
Gull 3					
Breast	85 $\pm$ 20	90 $\pm$ 20	1.6 $\pm$ 0.3	14 $\pm$ 3	—
Cormorant					
Breast	490 $\pm$ 60	500 $\pm$ 60	15 $\pm$ 2	77 $\pm$ 9	0.986
Liver	750 $\pm$ 65	810 $\pm$ 65	26 $\pm$ 2	110 $\pm$ 10	2.94
Brain	220 $\pm$ 8	230 $\pm$ 8	10 $\pm$ 2	39 $\pm$ 4	0.649
Liver Composite	800 $\pm$ 200	840 $\pm$ 190	29 $\pm$ 1	100 $\pm$ 30	3.07
<b>WILD BIRDS</b>					
Pelican 1					
Breast	9 $\pm$ 1	11 $\pm$ 1	0.7 $\pm$ 0.1	1.8 $\pm$ 0.1	0.150
Liver	1.6 $\pm$ 0.1	1.8 $\pm$ 0.1	0.11 $\pm$ 0.01	0.35 $\pm$ 0.03	0.583
Brain	1.0 $\pm$ 0.1	1.1 $\pm$ 0.1	0.07 $\pm$ 0.01	0.20 $\pm$ 0.01	0.084
Pelican 2					
Breast	110 $\pm$ 8	121 $\pm$ 9	0.6 $\pm$ 0.1	22 $\pm$ 2	0.545
Liver	12.7 $\pm$ 0.4	13.8 $\pm$ 0.4	0.11 $\pm$ 0.01	3.0 $\pm$ 0.2	1.03
Brain	7.8 $\pm$ 0.5	8.0 $\pm$ 0.5	0.08 $\pm$ 0.01	1.6 $\pm$ 0.2	0.322

\*Analysis of a single sample.

**Table 2. Concentrations (mg/wet kg) of chlorinated hydrocarbons in whole fish from the stock of food for marine birds at the Los Angeles Zoo.**

Sample	Mean Concentration ± Standard Error
Queenfish	
Lot 1 (n = 7)	
p,p'-DDE	3.1 ± 0.7
Total DDT	3.8 ± 0.8
1242 PCB	0.30 ± 0.06
1254 PCB	0.9 ± 0.2
Lot 2 (n = 6)	
p,p'-DDE	4.2 ± 1.1
Total DDT	5.0 ± 1.3
1242 PCB	0.38 ± 0.08
1254 PCB	0.9 ± 0.2
Surf smelt (n = 4)	
p,p'-DDE	0.018 ± 0.002
Total DDT	0.023 ± 0.004
1242 PCB	0.012 ± 0.001
1254 PCB	0.007 ± 0.002

**Table 3. Concentrations (mg/wet kg) of total mercury in tissues of fish from the stock of food for marine birds at the Los Angeles zoo.**

Sample	Concentration	
	Muscle	Liver
Queenfish		
Lot 1	0.107	0.095
Lot 2	0.066	0.077
Surf smelt	0.078	0.147