POLLUTANT CONCENTRATIONS IN ABNORMAL YOUNG TERNS FROM LONG ISLAND SOUND

HELEN HAYS AND ROBERT W. RISEBROUGH

THIS paper reports the results of preliminary analyses for chlorinated hydrocarbons and mercury in young terns with visible abnormalities found in a colony on Great Gull Island, 72° 07′ W, 41° 12′ N. Analyses are also reported on eight species of fish brought to the colony as food for the young or as part of behavioral displays.

Great Gull Island lies at the eastern end of Long Island Sound about 170 km east of New York City and almost 16 km east-northeast of the eastern tip of Long Island. In the late 19th century the island supported a large colony of Common Terns, *Sterna hirundo*, and Roseate Terns, *Sterna dougallii*, until a military fort built there in 1895 displaced them. When the U. S. Army abandoned the island in the late 1940s, many of the tern breeding grounds in Long Island Sound and along the Connecticut shore had either been destroyed by real estate encroachment or were permanently disturbed by improved transportation and the development of recreational facilities in the metropolitan area.

Aware of the island's history as the site of a large tern colony, the American Museum of Natural History and the Linnaean Society of New York cooperated to acquire the island hoping that terns might once again nest there. The museum received title to the island in 1949 and assigned to the Linnaean Society the task of returning it to a condition suitable for nesting terns. In 1949 and 1950 buildings were leveled, vegetation burned, and sand deposited in an attempt to create artificial beaches. These efforts were successful, and in 1955 a nucleus of 25 pairs of terns nested. By 1966 the number of breeding pairs exceeded 3,000 and the colony was considered sufficiently large and stable to permit detailed studies of the breeding biology of both species.

During the summers of 1969 and 1970, a team studying the productivity of both species of terns in the colony on Great Gull Island noted a number of abnormalities in the young terns. Most frequently observed were young birds that dropped their wing and tail feathers when they were 2 to 3 weeks old. Bill, eye, and foot deformities were also evident in several newly hatched young terns. Over 2,000 young Common Terns and more than 800 young Roseate Terns were handled each year. In 1970 the numbers of young terns of both species exhibiting some abnormality increased markedly.

 The Auk, 89: 19–35. January 1972

MATERIALS AND METHODS

In 1966 a grid was set up dividing Great Gull Island, which has an area of 17 acres, into quadrats 25 × 25 m square. For the production study undertaken in 1969 and 1970, all nests were located and recorded by quadrat; this helped finding the nests as they were rechecked. Both years daily checks were made from the time the first egg in the first nest was found until the last young in a sample of 1,500 nests had been ringed on a leg with a plastic band. Each nest was marked with a numbered wooden stick (commercial tongue depressor) placed in the ground beside it, and the nest number, location, and number of eggs were recorded. The eggs in each clutch were marked when found with a dri-mark pen to permit following the development of each egg.

Both Roseate and Common Terns lay small clutches varying in size from one to four eggs that hatch, as a rule, 20–28 days after they are laid. Eggs are normally laid at the rate of one per day, but as a day or two may be skipped between eggs occasionally, each clutch was checked for 7 days to insure that it was complete. In order to band the young terns as they hatched, a recheck date of 20 days after the laying of the first egg was assigned each nest. In both years over 1,500 nests were marked in this fashion before the young terns began hatching.

Upon hatching each young tern was given a numbered plastic band. Individuals of the same clutch were given the same number, but different colors. Birds from 1-egg clutches received purple bands, birds from 2-egg clutches yellow and green, and those from 3-egg clutches red, white, and blue respectively.

Intensive nest checks for newly hatched terns were carried out daily during both years from the time the first young hatched during the first 10 days in June until 4 July in 1969 and until 19 July in 1970 when more people were able to participate later in the season. Checks were continued at less regular intervals both years until the middle of August when the last young on the island hatched. During the final phase of the study, the numbered plastic bands were removed and replaced with a unique combination of three color bands and the aluminum U. S. Fish and Wildlife Service band, so that individual birds could be recognized and followed.

On all checks of the colony dead young were picked up and their numbers recorded. Abnormal young terns were either frozen or preserved in 10 percent formalin. In 1970 all fish found on the ground in the colony were collected and preserved in 10 percent formalin. Both the abnormal young and the fish samples were analyzed for total mercury, DDT, and PCB compounds using the following techniques:

For determination of total mercury, a portion of the left lobe of the liver was removed and sent to Gulf General Atomic, San Diego, California, for analysis by neutron activation, according to the following procedure as described by the company. "Weighed portions of each sample were sealed in vials and irradiated at a flux of 10^{12} thermal neutrons per cm^2-per second together with a mercury comparator standard. The irradiated samples were digested, in the presence of mercury carrier, in a mixture of HNO_3 and H_2SO_4 under reflux conditions. Multi-channel gamma ray spectrometry was used to identify and quantitate mercury."

DDT and PCB compounds were measured by methods previously described (Risebrough et al., 1970). The composition of the PCB compounds in both the terns and the fish closely resembled that of Aroclor 1254 (Monsanto Chemical Company, St. Louis, Missouri). PCB was therefore quantified as Aroclor 1254 using the peak with retention time 1.33 relative to p,p'-DDE on a QF-1 column as a standard (Peak 8, Ahling and

TABLE 1

DDE, PCB, AND MERCURY RESIDUES IN ABNORMAL YOUNG TERNS FROM GREAT GULL ISLAND

Age	Collection No.	Abnormality	Percent lipid	DDE[1]	PCB[1]	Mercury[2]
COMMON TERN						
Embryo	6	Dead in egg at pipping, crossed bill (Fig. 2)	NM[3]	3.1	56	0.238
Embryo	7	Dead in egg at pipping, underdeveloped lower mandible (Fig. 3)	NM	4.5	14	0.774
1 day	2	Splayed legs	NM	0.92	4.9	1.030
1 day	3	Eyes very small (Fig. 6)	NM	8.1	92	1.160
1 day	4	Underdeveloped legs and feet, little body down (Fig. 5)	NM	9.0	140	0.838
3 days	1	Four legs (Fig. 4)	NM	0.84	16	0.708
28 days	8	Dropping primaries and secondaries (Fig. 7)	17.4	2.5	32	NM
28 days	9	Little body down (Fig. 7)	2.8	0.70	13	NM
36 days	12	Found dead; primaries, secondaries, tail feathers missing	1.4	1.3	25	NM
42 days	13	Primaries, secondaries, tail feathers missing	NM	0.49	8.8	0.853
Adult	10	None, found dead	4.9	4.6	10	NM
ROSEATE TERN						
Embryo	5	Unable to emerge from abnormal egg	NM	3.2	63	0.870
21 days	14	Crossed bill, abortive feather development on wings (Fig. 1)	22.1	2.1	37	0.364
41 days	15	Primaries, secondaries, tail feathers missing	1.7	0.47	7.8	NM

[1] Concentrations in parts per million, wet weight, of breast muscle.
[2] Concentrations in parts per million, wet weight, of liver.
[3] NM = not measured.

Jensen, 1970). This peak does not interfere with any of the common insecticide derivatives on this column (Risebrough et al., 1969).

RESULTS AND DISCUSSION

Of 971 young Roseate Terns banded at hatching in 1969, 2 or 0.2 percent were found on the island late in the summer unable to fly. Examination showed both were missing primaries in each wing, but new feathers appeared to be growing in.

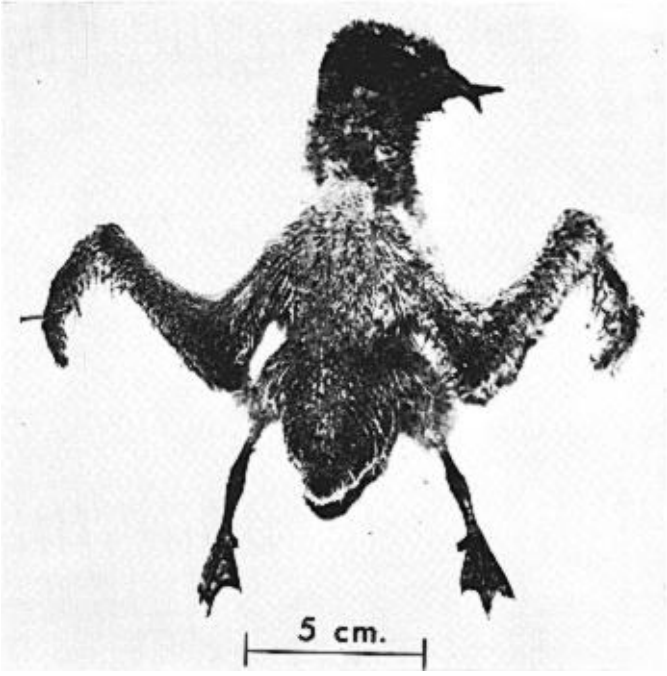

Figure 1. Young Roseate Tern with crossed bill and sparse down on abdomen.

In 1970 of 806 young Roseate Terns checked at hatching and again 2 weeks later, 5 or 0.6 percent showed some sort of abnormality. In one of these birds (no. 14 of Table 1, Figure 1), the upper mandible was crossed over the lower, the bird had little down on the abdomen, and little feather development on the wings. Four others appeared normal at hatching, but lost primaries, secondaries, and tail feathers when they were between 2 and 3 weeks of age. These feathers were not replaced. The Roseate Terns found with missing primaries in 1969 were probably the first to indicate the occurrence of this feather loss in the nesting population on Great Gull Island.

Of 2,089 young Common Terns banded at hatching in 1969, 1 or 0.04 percent was abnormal. This bird showed a crossed upper mandible similar to that of the Roseate Tern in Figure 1.

In 1970 of 2,316 young Common Terns examined at hatching, 35 or 1.5 percent were abnormal. In 9, or 26 percent of these, the abnormalities were evident at the time of hatching. Two (Nos. 6 and 7 of Table 1) had bill deformities and did not emerge from the shell. The upper mandible of

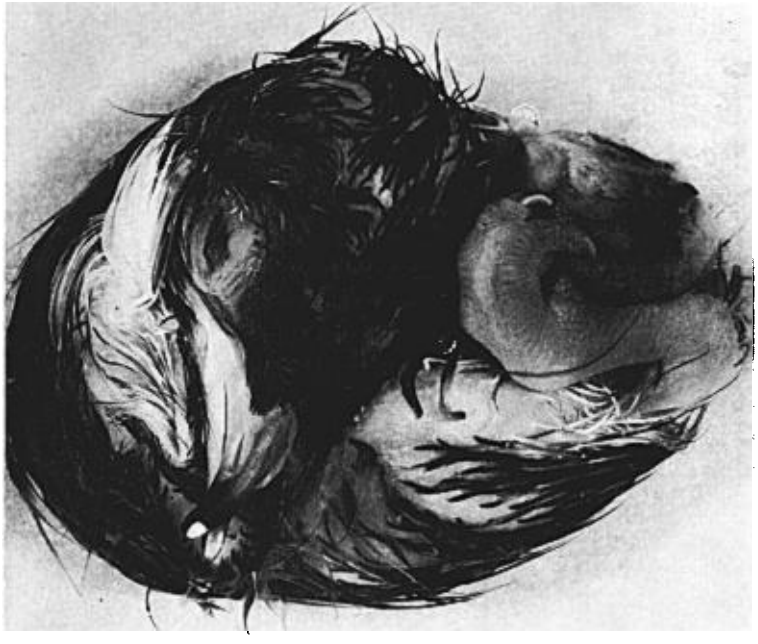

Figure 2. Young Common Tern with crossed mandible. It reached this stage of development in the egg and did not hatch.

Figure 3. Young Common Tern with underdeveloped lower mandible. It reached this stage of development in the egg and did not hatch.

Figure 4. Young Common Tern with four legs.

No. 6 was crossed over the lower (Figure 2) and No. 7 had an underdeveloped lower mandible (Figure 3). A third young Common Tern hatched with four legs lived only 3 days (No. 1 of Table 1, Figure 4); a fourth had underdeveloped legs and feet with little body down (No. 4, Figure 5); two

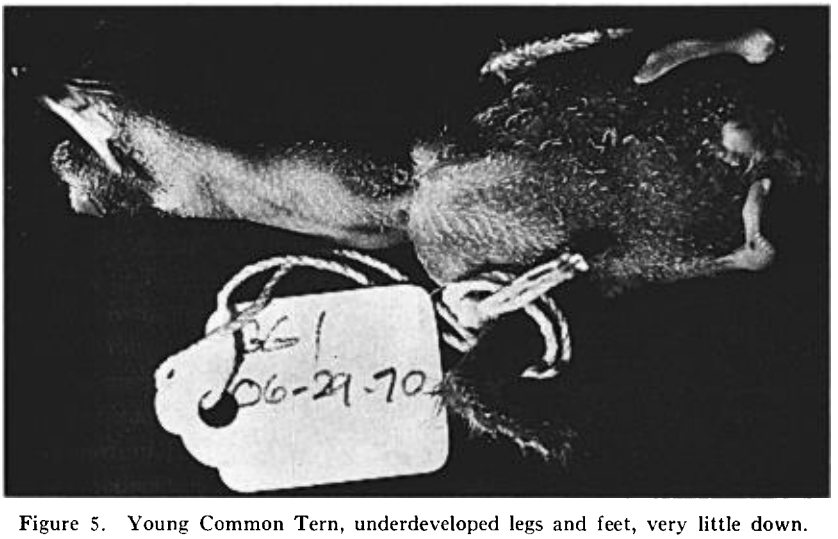

Figure 5. Young Common Tern, underdeveloped legs and feet, very little down.

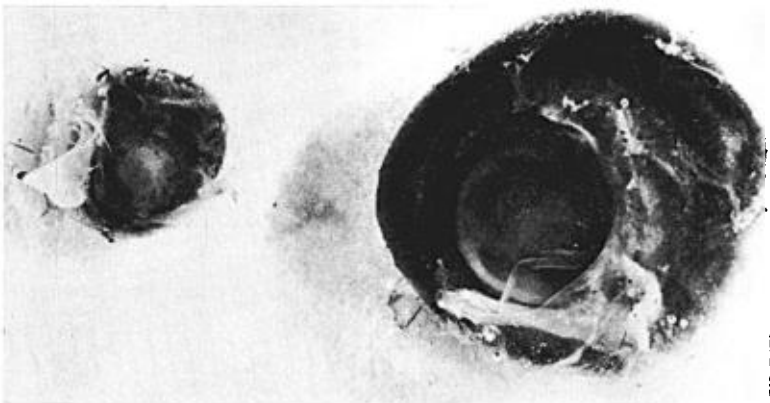

Figure 6. Eyes from two 1-day-old Common Terns; the one on the right was dissected from a bird that appeared normal but died, the other was dissected from a bird that appeared to have no eyes at the time of hatching.

had abnormally small eyes (one was collected, No. 3, Figure 6); two had splayed legs as though tendon development were abnormal (one of which was collected, No. 2); and the ninth had a swelling about the base of the bill.

Of the young Common Terns plastic-banded at hatching 26 or 1.1 percent appeared normal, but lost their primaries, secondaries, and tail feathers between 2 and 4 weeks of age. Two birds from the same nest that had lost feathers are shown in Figure 7, Nos. 8–9 of Table 1. These birds were energetic and vigorous. One of the birds that had been flightless at 28 days of age on Great Gull Island we saw later at Milford, Connecticut, about 125 km west of Gull Island, on 25 August when it was 57 days old; it was identified by its color combination. This sighting, as well as the observations of the young Roseate Terns examined on Great Gull Island in 1969, in which the primaries appeared to be growing in to replace those that were missing, suggests that the feather loss is not always permanent.

Of the 26 birds seen in a flightless condition on Great Gull Island in 1970, we were able to follow only a few. The rest disappeared and some were taken by gulls or hawks. One young Common Tern that had lost all wing feathers as well as tail feathers lived to be 60 days old, at which time it was collected. Four others of these flightless birds were found dead at 31, 35, 36, and 42 days of age respectively, and none had replaced the lost feathers.

To test the hypothesis that the birds that had lost feathers might have contracted a disease, one young Roseate Tern and two young Common Terns that were dropping their wing and tail feathers were sent to Louis Leibovitz,

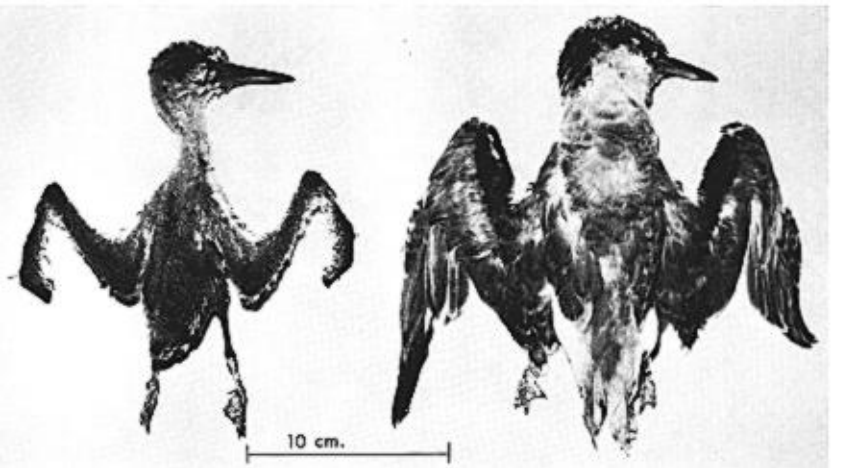

Figure 7. Two Common Terns from the same clutch, each 28 days old. The bird on the left never grew much down and few feathers came in. The bird on the right is losing primaries and secondaries.

an avian pathologist at Cornell University's Duck Research Laboratory at Eastport, Long Island. He reported that no significant bacterial or viral pathogens were recovered from these birds.

One young Common Tern that had lost primaries, secondaries, and tail feathers was sent to Malcolm Peckham, Department of Avian Diseases at Cornell University, who noted that the feathers had been abortive in development. Growth had been interrupted, resulting in weakening of the shaft and dropping of the feather. The search for the causes of the abnormalities, unlike the shell-thinning effect that can be related directly to concentrations of pollutants such as DDE (Anderson et al., 1969), is hampered by the nature of the relationships between environmental mutagens or teratogens and the incidence of observed effects. Exposure of a large population of organisms to comparable concentrations of a mutagen or teratogen could be expected to produce abnormalities in only a few individuals. Concentrations of the chemical or chemicals responsible could be as high in birds that appeared normal as in those few with defects. Increasing the level of exposure, however, would increase the probability and therefore the incidence of the effects. Within a given ecosystem, therefore, such as the marine ecosystem supporting the terns of Great Gull Island, it may not be possible to relate the abnormalities to concentrations of any one or a combination of pollutants in the birds. Three of the pollutants suspected to be causes of or contributors to the production of abnormalities are the DDT compound DDE, polychlorinated biphenyl (PCB), and mercury. The results of the

analyses for these chemicals in the young terns described above and of a young bird that had been unable to emerge from an unusually long egg, as well as an adult Common Tern found dead, are given in Table 1. These data might be considered as an indication of the DDE, PCB, and mercury contamination of the tern populations on Great Gull Island. Further studies relating the incidence of defects with the contamination levels of ecosystems would require measurements of pollutant concentrations in colonies where the incidence of abnormalities is lower.

Although the number of abnormalities observed in 1970 was considerably greater than the number found in 1969 among a comparable number of nestlings, the literature contains few data indicating the proportion of abnormalities that might be expected in a population uncontaminated by synthetic pollutants. Austin (1969) reported a young Sooty Tern, *Sterna fuscata*, found on Bush Key in the Dry Tortugas 11 June 1967 with extra toes projecting from the tarsometatarsus of both legs. He noted that this was the first abnormality observed in banding over 125,000 Sooty Tern young during the preceding eight seasons. Smith and Diem (1971) report four California gulls, *Larus californicus*, with crossed bills after handling 6,543 individuals during 12 years of banding in Wyoming.

The phenomenon of feather loss observed on Great Gull Island appears not to have been described previously in the literature. During the summer of 1970, birds that had dropped wing and tail feathers were reported from colonies on the south shore of Long Island. Michael Gochfeld (pers. comm.) reported 18 Common Terns with missing primaries and secondaries from the Jones Beach colony located on the south shore at the western end of Long Island and 8 flightless young Common Terns from Cedar Beach, located just east of Jones Beach. Erma J. Fisk (pers. comm.) reported one bird that she checked twice at Yarmouth Beach on Cape Cod, Massachusetts. When first examined, feathers were beginning to break through the sheath on the left wing, but no feathers at all were visible on the right. When examined 2 weeks later a few primaries on the right wing were beginning to come in, but the growth pattern was irregular. Ian C. T. Nisbet (pers. comm.) reported finding two young Common Terns both of which had lost feathers on Ram Island, Massachusetts on 26 July 1970.

Long Island Sound receives industrial, municipal, and agricultural wastes from one of the most densely populated areas of the United States and is very likely one of the most polluted areas of salt water in the world. Effects of pollutants in marine ecosystems might, therefore, be observed here first.

Declining reproductive success of the Osprey, *Pandion haliaetus*, was first noted in nearby coastal Connecticut in the early 1960s by Ames (1966) and others. Reproductive failures of this species on Gardiner's Island, approximately 20 km distant from Great Gull Island, have been attributed

to shell thinning (P. Spitzer, MS), a phenomenon now believed to be caused principally by the DDT compound p,p'-DDE (Heath et al., 1969; Wiemeyer and Porter, 1970; Peakall, 1970). Effects of other pollutants, including no doubt several as yet undetected in the marine environment, might include abnormalities of the kind reported here. The tern colony, which permits a sample size of several thousand hatchlings to be examined yearly, therefore merits continued detailed study.

Table 1 gives the results of the analyses of the young terns and one adult Common Tern for DDT, PCB, and total mercury compounds. DDE was the only DDT compound detected in all but one of the samples. Concentrations ranged from 0.5 to 9 ppm on a wet weight basis. Concentrations of DDE of this order of magnitude are now present in populations of many species of birds and may be associated with significant shell thinning (Anderson et al., 1969). No evidence suggests that any of the DDT compounds cause embryonic abnormalities in birds within this range of concentration, although few other studies of pollutant effects upon natural populations have been based upon a yearly sample size as large as 2800 birds.

Concentrations of the polychlorinated biphenyls in the terns were considerably higher than those of DDE (Table 1).

Mercury concentrations ranged from 0.2 to 1.2 ppm in the livers on a fresh weight basis with arithmetic and geometric means of 0.76 ppm and 0.68 ppm respectively (Table 1). Our sample consisted of young birds only, whose mercury concentrations might be lower than those of adults. To date there are no data to provide expected background levels of mercury in this ecosystem.

A recent discussion of mercury in the environment (Hammond, 1971) points out that the total amount of mercury so far extracted from the earth's crust by man is between 100 and 1000 times lower than the total amount of mercury estimated to be in the oceans. Mercury concentrations in oceanic fish and birds might therefore be of natural rather than pollutant origin. Further investigation is necessary to determine whether mercury levels in coastal waters such as Long Island Sound are significantly higher than those in waters farther from the source of mercury contamination.

Several papers from Sweden (Berg et al., 1966; Borg et al., 1969; Johnels and Westermark, 1969) have reported on the environmental distribution of mercury compounds in that country. Forty-one Goshawks, *Accipiter gentilis*, shot or trapped in Sweden at a time when mercury compounds were still being used as seed dressings, contained a median concentration of 5.3 ppm of total mercury in their livers (range 0.2–53 ppm) and 29 buzzards, *Buteo buteo*, contained a median concentration of 2.1 ppm (0.2–6.5 ppm) (Borg et al., 1969).

A study of mercury levels in some birds of Alberta and Saskatchewan showed considerably higher levels in Alberta, where mercury compounds have been used as seed dressings, than in Saskatchewan where concentrations are assumed to be closer to natural levels. Mean concentration of total mercury in livers of 32 adult seed-eating birds from Saskatchewan was 0.37 ppm. Adult seed-eating birds from Alberta averaged 1.16 ppm of total mercury in their livers (Fimreite et al., 1970). Although it is not possible to make meaningful comparisons among different species in very different ecosystems, the concentrations of mercury in the livers of the young terns do not appear excessively high.

Mercury concentrations that will produce deleterious effects may not be very much higher than concentrations assumed to be natural. When pheasants were fed wheat treated with methyl mercury dicyandiamide, decreased hatchability of eggs was associated with mercury concentrations in the eggs ranging from 1.3 to 2.0 ppm of the egg contents (Borg et al., 1969). Injections of 0.5 mg of mercuric chloride into chick embryos, equivalent to a concentration in the order of 10 ppm, were sufficient to kill all embryos at the beginning of incubation (McLaughlin et al., 1963). Mercury compounds must therefore be considered among the potential causes of the deformities, along with other heavy metals such as lead and cadmium, which are also mobilized in vast quantities from the earth's crust for industrial use and released as waste products in regions of technological activity.

Table 2 lists the results of the analyses of the fish samples for the DDT and PCB compounds. DDT concentrations are considerably lower than those reported in fish from the marine ecosystem of southern California, where the major source of DDT pollution has been the effluent of a DDT manufacturing factory (Risebrough et al., 1971); or from the Baltic (Jensen et al., 1969).

Two small Atlantic herrings, *Clupea harengus*, from Long Island Sound (Table 2) contained only 0.049 ppm of total DDT on a fresh weight basis. Total DDT residues in the blueback herring were 0.41 ppm on a fresh weight basis, or 6.4 ppm on a lipid basis. Residues of total DDT in the fat of the eight species examined ranged from 4 to 14 ppm. In the Baltic, concentration of the DDT compounds in a sample of fish oil was 16 ppm, and Atlantic herring were found to contain 0.68 and 17 ppm on a fresh tissue and lipid basis (Jensen et al., 1969). In southern California residue concentrations may be 10 ppm or higher on a fresh weight basis (Risebrough et al., 1971).

Concentrations of PCB in fish from San Francisco Bay and from the marine waters of southern California may be as high as 1 ppm in the flesh (Risebrough, 1969), and in the Baltic PCB concentrations in fish lipids

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

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

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

are in the order of 3 to 11 ppm (Jensen et al., 1969). PCB concentrations in lipids of fish obtained from Great Gull Island ranged from 10 to 175 ppm, with a geometric mean of 50 ppm. Long Island Sound, therefore, appears to be more contaminated with PCB than the Baltic, but comparable to some California coastal waters.

The toxicity of a PCB mixture, Aroclor 1254, when fed to Bengalese finches, *Lonchura striata*, was found to be considerably less than that of p,p'-DDT (Prestt et al., 1970). In testing the toxic effects of three different PCB mixtures upon chickens Vos and Koeman (1970) found that feather loss accompanied the mortality induced by PCB. An earlier study of the effects of feeding chlorinated biphenyls to chicks had noted feather loss in one of the birds (Flick, O'Dell, and Childs, 1965). Although the three PCB preparations, manufactured respectively in France, Germany, and the U.S.A., each contained approximately 60 percent chlorine and were therefore virtually identical in gross composition, their toxicities differed significantly. The French PCB preparation showed the highest toxicity and the American preparation the least (Vos and Koeman, 1970).

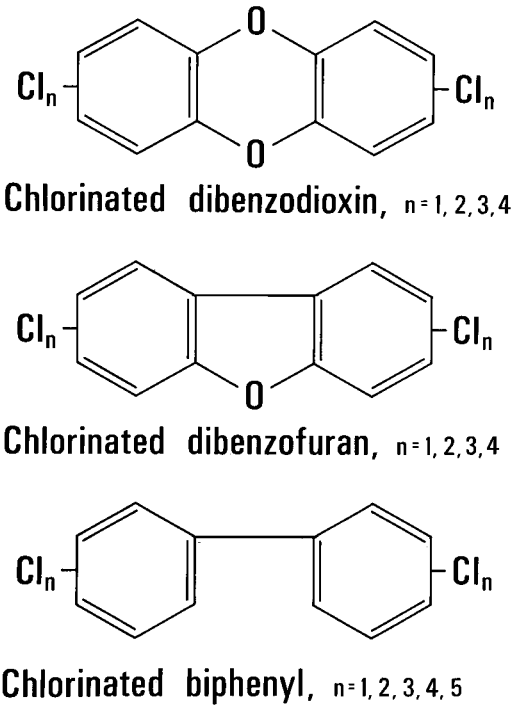

Figure 8. Structures of chlorinated biphenyl, chlorinated dibenzofuran, and chlorinated dibenzo-p-dioxin. Chlorine atoms may occupy any of the available positions.

Certain symptoms, such as atrophy of the spleen and a disturbance in prophyrin synthesis were elicited by all three preparations. In addition, the French and German products reproduced many of the effects characteristic of chick edema disease (Friedman et al., 1959; Douglass and Flick, 1961; Flick, Firestone and Marliac, 1965; Anonymous, 1968). These included abnormal abdominal and subcutaneous edema, and hydropericardium. Flick, O'Dell and Childs (1965) in their study of PCB toxicity also found that some of the symptoms produced were similar to those of the chick edema disease.

Chick edema disease is caused by one or more classes of chlorinated organic compounds (Firestone et al., 1963), some of which have been identified as chlorinated dibenzo-p-dioxins (Higginbotham et al., 1968; Cantrell et al., 1969). Semi-purified fractions of preparations that elicited the chick edema disease when fed to chicks were injected into fertile chicken eggs by Flick, Firestone and Marliac (1965). Embryos that failed to hatch showed developmental abnormalities similar to those observed in the tern chicks and included malformed beaks, eye defects, and leg deformities.

Chicks that hatched from the eggs injected with chick edema factor preparations "exhibited sparse and defective feathering (down)" (Flick, Firestone and Marliac, 1965).

The toxic factor in the polychlorinated biphenyls from France and Germany was traced to small amounts of contaminants, chlorinated dibenzofurans, present in the PCB in concentrations in the order of 5 and 20 ppm in the German and French preparations, respectively (Vos et al., 1970). These compounds are very similar in structure to the chlorinated dibenzo-p-dioxins (Figure 8). The toxic fraction, containing the chlorinated dibenzofurans isolated from PCB, when injected into fertile chicken eggs killed all the embryos during early incubation at an estimated dose of 0.2 micrograms of the pentachlorodibenzofuran, equivalent to a concentration in the embryo of several parts per billion (Vos et al., 1970). The chlorinated dibenzo-p-dioxins are among the most toxic compounds known, especially to developing embryos. Concentrations in the order of 20 parts per trillion may produce embryonic defects in chick embryos. The symptoms found in chicks that developed in eggs injected with purified 2,3,6,7,-tetrachloro-p-dioxin included eye and beak defects and short twisted feet (Verrett, 1970), strikingly similar to those observed in tern chicks on Great Gull Island. When applied to mammalian skin, both groups of compounds produce symptoms of chloracne (Bauer et al., 1961). One of the chicks developing in eggs injected with the polychlorinated biphenyl mixture, Aroclor 1242, showed a bill deformity (McLaughlin et al., 1963).

The abnormalities shown by the young terns are therefore very similar to those produced by the chlorinated dioxins under experimental conditions, and by PCB preparations that may contain the closely related chlorinated dibenzofurans. To date these compounds have not been found in environmental samples. The chlorinated dioxins are present as contaminants in preparations of the herbicide 2,4,5,-T and of other chemicals prepared from chlorinated phenols (Verrett, 1970). Many populations of birds now contain concentrations of PCB in the order of 100 parts per million of the body tissue. If the chlorinated dibenzofurans were associated with PCB in the environment in a ratio of one to ten parts of dibenzofuran per million parts of PCB, the concentrations of the benzofurans in the birds might be in the order of 100 to 1000 parts per trillion. Concentrations of this order of magnitude may be sufficient to produce embryonic effects (Vos et al., 1970).

Preparations for continuing studies in 1971 are now under way. These should determine whether the abnormalities found in 1970, and to a lesser extent in 1969, were a singular occurrence or, like the broken eggs observed in eyries of the Peregrine Falcon, *Falco peregrinus*, in the 1950s (Ratcliffe, 1958), a portent of the future.

ACKNOWLEDGMENTS

Analytical work was supported by NSF grant GB 11649 to the Institute of Marine Resources, University of California, H. S. Olcott, principal investigator. We thank Patricia K. Schmidt for assistance in the laboratory.

Work on Great Gull Island was supported by The American Museum of Natural History, The Linnaean Society of New York, the Chapman Fund, the Mae P. Smith Fund, and the Anne S. Richardson Fund.

Field work was carried out by Grace Donaldson, Jacqueline Greenberg, Helen Lapham, Mary LeCroy, Kenneth Parkes, and Catherine Pessino who marked nests and plastic banded the young at hatching. David Duffy, Sara LeCroy, Roger Pasquier, and Alan Poole did the major part of adult color banding of the young terns. We would like to take this opportunity to thank Donald Rosen and Gareth J. Nelson for identifying the fish, Louis Liebovitz and Malcolm Peckham for examining tissues of some of the abnormal terns, William C. Dilger for examining and referring specimens, and Robert E. Logan and Arthur Singer for taking and printing the pictures of the abnormal young terns.

SUMMARY

The incidence of abnormal chicks of Common and Roseate Terns in the colony on Great Gull Island, New York, at the eastern end of Long Island Sound, was recorded during a productivity study in 1969 and 1970. Over 2,000 young Common Terns and more than 800 young Roseate Terns were handled each year. In 1969, 0.1 percent of the young terns showed an abnormality and in 1970, the incidence of visible defects was 1.3 percent. Abnormalities included feather loss in juvenile terns, as well as eye, bill, and foot deformities in several newly hatched young terns.

Abnormal young collected in 1970 and fish brought to the colony by adults were analyzed for the DDT and PCB compounds and in some cases for total mercury. Concentrations of mercury in the livers of the young terns ranged from 0.2 to 1.2 ppm on a fresh weight basis with arithmetic and geometric means of 0.76 and 0.68 ppm respectively. Median concentrations of DDE and PCB in the breast muscle of the young terns were 2.1 and 25 ppm wet weight, respectively. Median concentrations of the sum total of the three principal DDT compounds p,p′-DDE, p,p′-DDD, and p,p′-DDT and of PCB in collections of eight species of fish brought to the colony by adult terns were 0.19 and 1.0 ppm wet weight, respectively. On a lipid basis, DDT residues ranged from 4.2 to 13.9 ppm and PCB ranged from 10 to 180 ppm in six of the fish species.

Long Island Sound is much less contaminated with DDT compounds than are are the coastal waters of California.

The abnormalities recorded resemble those produced in young chickens under experimental conditions by the chlorinated dibenzo-p-dioxins and some preparations of the polychlorinated biphenyls.

LITERATURE CITED

AHLING, B., AND S. JENSEN. 1970. Reversed liquid-liquid partition in determination of polychlorinated biphenyl (PCB) and chlorinated pesticides in water. Anal. Chem., 42: 1483–1486.

ANDERSON, D. W., J. J. HICKEY, R. W. RISEBROUGH, D. F. HUGHES, AND R. E. CHRISTENSEN. 1969. Significance of chlorinated hydrocarbon residues to breeding pelicans and cormorants. Canadian Field-Naturalist, 83: 91–112.

AMES, P. L. 1966. DDT residues in the eggs of the Osprey in the north-eastern United States and their relation to nesting success. J. Appl. Ecol., 3: Suppl., 87–97.

ANONYMOUS. 1968. The chick edema factor. Nutrition Rev., 26: 28–30.

AUSTIN, O. L., JR. 1969. Extra toes on a Sooty Tern chick. Auk, 86: 352.

BAUER, H., K. H. SCHULZ, AND U. SPIEGELBERG. 1961. Berufliche Vergiftigung bei der Herstellung von Chlorphenol-Verbindungen. Arch. Gewerbepathol. Gewerbehvg., 18: 538–555.

BERG, W., A. JOHNELS, B. SJOSTRAND, AND T. WESTERMARK. 1966. Mercury content in feathers of Swedish birds from the past 100 years. Oikos, 17: 71–83.

BORG, K., H. WANNTORP, K. ERNE, AND E. HANKO. 1969. Alkyl mercury poisoning in terrestrial Swedish wildlife. Viltrevy, 6: 301–379.

CANTRELL, J. S., N. C. WEBB, AND A. J. MABIS. 1969. The identification and crystal structure of a hydropericardium-producing factor: 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin. Acta Cryst., B25: 150–154.

DOUGLASS, C. D., AND D. F. FLICK. 1961. Collaborative bioassay for chick edema factor. J. Assoc. Off. Anal. Chem., 44: 449–456.

FIMREITE, N., R. W. FYFE, AND J. A. KEITH. 1970. Mercury contamination of Canadian prairie seed eaters and their avian predators. Canadian Field-Naturalist, 84: 269–276.

FIRESTONE, D., W. IBRAHIM, AND W. HOROWITZ. 1963. Chick edema factor. III, Application of microcoulometric gas chromatography to detection of check edema factor in fats or fatty acids. J. Assoc. Off. Anal. Chem., 47: 384–396.

FLICK, D. F., D. FIRESTONE, AND J. P. MARLIAC. 1965. Studies of the chick edema disease. 2, Preparation and biological effects of a crystalline chick edema factor concentrate. Poultry Sci., 44: 1214–1222.

FLICK, D. F., R. G. O'DELL, AND V. A. CHILDS. 1965. Studies of the chick edema disease. 3, Similarity of symptoms produced by feeding chlorinated biphenyl. Poultry Sci., 44: 1460–1465.

FRIEDMAN, L., D. FIRESTONE, W. HOROWITZ, D. BANES, M. ANSTEAD, AND G. SHUE. 1959. Studies of the chicken edema disease factor. J. Assoc. Off. Anal. Chem., 42: 129–140.

HAMMOND, A. L. 1971. Mercury in the environment: Natural and human factors. Science, 171: 788–789.

HEATH, R. G., J. W. SPANN, AND J. F. KREITZER. 1969. Marked DDE impairment of Mallard reproduction in controlled studies. Nature, 224: 47–48.

HIGGINBOTHAM, G. R., A. HUANG, D. FIRESTONE, J. VERRETT, J. RESS, AND A. D. CAMPBELL. 1968. Chemical and toxicological evaluations of isolated and synthetic chloro derivatives of dibenzo-p-dioxin. Nature, 220: 702–703.

JENSEN, S., A. G. JOHNELS, M. OLSSON, AND G. OTTERLIND. 1969. DDT and PCB in marine animals from Swedish waters. Nature, 224: 247–250.

JOHNELS, A. G., AND T. WESTERMARK. 1969. Mercury contamination of the environment in Sweden. Pp. 221–241 *in* Chemical fallout (M. W. Miller and G. G. Berg, Eds.). Springfield, Illinois, Charles C Thomas.

MCLAUGHLIN, J., JR., J. P. MARLIAC, M. J. VERRETT, M. K. MUTCHLER, AND O. G. FITZHUGH. 1963. The injection of chemicals into the yolk sac of fertile eggs prior to incubation as a toxicity test. Toxic. Appl. Pharmacol., 5: 760–771.

PEAKALL, D. B. 1970. p,p'-DDT: Effect on calcium metabolism and concentration of estradiol in the blood. Science, 168: 592–594.

PRESTT, I., D. J. JEFFERIES, AND N. W. MOORE. 1970. Polychlorinated biphenyls in wild birds in Britain and their avian toxicity. Environ. Pollut., 1: 3–26.

RATCLIFFE, D. A. 1958. Broken eggs in Peregrine eyries. Brit. Birds, 51: 23–26.

RISEBROUGH, R. W. 1969. Chlorinated hydrocarbons in marine ecosystems. Pp. 5–23 *in* Chemical fallout (M. W. Miller and G. G. Berg, Eds.). Springfield, Illinois, Charles C Thomas.

RISEBROUGH, R. W., G. L. FLORANT, AND D. D. BERGER. 1970. Organochlorine pollutants in Peregrines and Merlins migrating through Wisconsin. Canadian Field-Naturalist, 84: 247–253.

RISEBROUGH, R. W., D. B. MENXEL, D. J. MARTIN, H. S. OLCOTT, F. GRESS, T. SCHMIDT, AND P. K. SCHMIDT. 1971. DDT residues in Pacific marine fish. Pesticides Monitoring J., in press.

RISEBROUGH, R. W., P. REICHE, AND H. S. OLCOTT. 1969. Current progress in the determination of the polychlorinated biphenyls. Bull. Environ. Contam. Toxicol., 4: 192–201.

SMITH, J., AND K. L. DIEM. 1971. Incidence of deformed bills in gulls (*Larus californicus*). Auk, 88: 435.

VERRETT, J. 1970. Statement before the Subcommittee on Energy, Natural Resources, and the Environment of the Committee on Commerce, United States Senate, Ninety-first Congress. Second Session on Effects of 2,4,5-T on Man and the Environment. Serial 91–60, U. S. Government Printing Office, pp. 190–360.

VOS, J. G., AND J. H. KOEMAN. 1970. Comparative toxicologic study with polychlorinated biphenyls in chickens with special reference to porphyria, edema formation, liver necrosis and tissue residues. Toxicol. Appl. Pharmacol., 17: 656–668.

VOS, J. G., J. H. KOEMAN, H. L. VAN DER MASS, M. C. TEN NOEVER DE BRAUW, AND R. H. DEVOS. 1970. Identification and toxicological evaluation of chlorinated dibenzofuran and chlorinated naphthalene in two commercial polychlorinated biphenyls. Fd. Cosmet. Toxicol., 8: 625–633.

WIEMEYER, S. N., AND R. D. PORTER. 1970. DDE thins eggshells of captive American Kestrels. Nature, 227: 737–738.

HELEN HAYS, *Department of Ornithology, American Museum of Natural History, Central Park West at 79th Street, New York, New York 10024,* and ROBERT W. RISEBROUGH, *Institute of Marine Resources, Department of Nutritional Sciences, University of California, Berkeley, California 94720. Present address: Bodega Marine Laboratory, University of California, P. O. Box 247, Bodega Bay, California 94923.* Accepted 11 October 1971.