EFFECTS OF ENVIRONMENTAL POLLUTANTS UPON ANIMALS OTHER THAN MAN

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All pollutants are waste products. They include both "natural" compounds that are present in undesirable concentrations in local ecosystems and chemical species foreign to the environment. What distinguishes them from other chemical wastes is, by definition, a potential capacity to inflict harm upon one or more species of an ecosystem. Many populations of wildlife are currently affected by pollutants that decrease the life span of adults or lower their reproductive capacity. Since these species breathe the same air as does man and consume some of the same food, they constitute an early warning system for the future health of man. In some cases it is not immediately evident which pollutants are producing the observed effects and an increasing amount of research is being devoted to these problems. The present paper will attempt to summarize our current knowledge in the field of pollutant ecology that relates to the effect of pollutants upon wildlife populations. Hopefully it will provide a useful background to the formulation of programs that will look for effects of these same pollutants upon human health.

1. Dimensions of the system

It is evident that the capacity of the earth to support life is finite. A convenient parameter with which to discuss the dimensions of the global ecosystem is the amount of organic carbon synthesized per year by the photosynthetic activity of plants. Production of organic carbon in the sea has been estimated to be in the order of 2×10^{16} grams per year [52]. Photosynthetic processes on land produce in the order of 6×10^{16} grams of organic carbon per year [38]. The sum of these numbers, 8×10^{16} grams, is therefore a useful number with which to compare such parameters as annual petroleum production, the annual U.S. production of organic chemicals, global mineral production, and the total amount of waste material formed by the sum total of global technology. The latter might be considered a measure of the current level of human activity.

2. Waste products as pollutants

Carbon monoxide is a waste product that has become a locally dangerous pollutant in urban areas. Worldwide emissions from major industrial sources,

excluding fuel consumption, amounted to 2.6×10^{13} grams in 1968 [38]. The atmospheric concentrations of carbon monoxide, however, do not appear to be increasing [16], [30] in spite of the considerable input. In sinks such as soil [29] carbon monoxide may be converted to either carbon dioxide or methane. The ecosystem appears therefore to be able to absorb the increased input.

In contrast, the atmospheric concentrations of carbon dioxide are increasing. The burning of fossil fuels currently releases in the order of 14 billion metric tons of carbon dioxide into the atmosphere per year or 1.4×10^{16} grams. This figure is close to the activity level of the natural ecosystem. Approximately one-half of this input remains in the atmosphere, resulting in an increase in CO_2 concentrations at the rate of 0.2 per cent per year [38], [34], [42].

Several of the heavy metals may become pollutants when environmental levels are increased significantly above background. Zinc, copper and iron are essential components of enzymes or other proteins and are not toxic at lower concentrations. Lead, mercury and cadmium, however, may be highly toxic to biological systems. All are natural components of the earth's crust and are transported to the sea upon weathering of the rocks by water or wind. Ultimately they are deposited in sediments which in turn are uplifted to form new mountains.

The present rate of input of lead into the oceans is approximately ten times greater than the rate of introduction by natural weathering [13]. Concentrations of lead in surface waters are higher than in deeper waters. Moreover, the isotope composition of the lead in surface waters and in recent precipitation is more similar to that of mined ore leads than to that in marine sediments [12]. There are almost no data, however, that would suggest that the higher concentrations of lead in surface seawater derived from the lead transported through the atmosphere have resulted in higher concentrations in marine wildlife. Lead poisoning is frequently encountered in waterfowl that have ingested lead shot from the bottom mud of marshes, but there are very few data that would indicate what should be "natural" levels in terrestrial and fresh water wildlife. Lead concentrations that have been measured in liver and bone of selected species of birds have been compiled by Bagley and Locke [5]. Annual global production of lead is in the order of 3×10^{12} grams [38].

Global production of cadmium ranged from 1.2 to 1.4×10^{10} grams per year between 1963 and 1968 [38] and in addition significant quantities are released into the environment as by-products of zinc mining operations. These quantities are sufficiently high to indicate that cadmium could be a significant pollutant in local areas. Extensive poisoning of human populations living downstream from a zinc mine in Japan has been documented [64]. Cadmium levels in tissues of the Ashy Petrel (*Oceanodroma homochroa*), an oceanic species resident in the coastal waters of California, were approximately twice as high as concentrations in tissues of two populations of the Wilson's Petrel (*Oceanites oceanicus*) that breed in the Antarctic but summer in the Atlantic and Australian regions respectively. Cadmium levels in tissues of the Snow Petrel (*Pagodroma nivea*), a species

that never leaves the Antarctic pack ice, were in the same order of magnitude as those in the Wilson's Petrel. Cadmium levels in eggs of the Common Tern (*Sterna hirundo*) from Long Island Sound were in the order of 0.2 ppm dry weight, not appreciably higher than those in the Antarctic Tern (*Sterna vittata*) from the Antarctic with levels in the order of 0.1 ppm. These limited data do not suggest that cadmium has become a significant marine pollutant [2].

An estimated 5,000 tons of mercury, or 5×10^9 grams, are transferred per year from the continents to the oceans as a result of continental weathering [35]. Global production of mercury is currently about twice as high, in the order of 9×10^9 grams per year [23]. In addition, the burning of petroleum releases in the order of 1.6×10^9 grams into the atmosphere per year [9]. A conservative estimate of the amount of mercury released per year into the global environment from the burning of coal is in the order of 3×10^9 grams [33]. Thus the amount of mercury mobilized by man is considerably higher than the amounts released by natural weathering. Nevertheless the amount of mercury estimated to be in the oceans is in the order of 10^{14} grams, approximately three orders of magnitude higher than the amount of mercury consumed in the United States since 1900. Mercury in marine organisms is therefore most likely of natural origin. Thus, mercury concentrations in the tissues of the Ashy Petrel from the coastal waters of California, the site of most of the mercury mines in the United States, were in the same order of magnitude as the mercury concentrations in tissues of the Snow Petrel from Antarctica. Mercury concentrations in nine eggs of the Common Tern from Long Island Sound were only slightly higher than in nine eggs of the Antarctic Tern from Antarctica [2].

Environmental residues of mercury in Sweden, as measured by concentrations of mercury in feathers of several species of birds, rose dramatically in the years following 1940. The source was considered to be the alkyl-mercury compounds used as seed dressings [8]. The use of mercury compounds as seed dressings ha also resulted in higher environmental levels of mercury in Alberta [19]. Local pollution of lakes and rivers in North America has resulted from the discharge of the wastes from factories using mercury, notably chlorine-caustic soda plants.

Pollution of the environment by heavy metals might therefore be considered equivalent to an accelerated weathering process, resulting in local concentrations that are higher than background levels and in some cases higher than a threshold of damage to one or more species. Local pollution by other inorganic compounds such as salts are also instances of concentrations of naturally occurring compounds exceeding those normally encountered by wildlife.

In contrast, several of the organic compounds that have become pollutants are synthetic, unknown to exist in the environment before the development of chemical technology and represent a new evolutionary factor in ecosystems. Total U.S. production of synthetic organic chemicals in 1968 was 120,000 million pounds or approximately 5×10^{13} grams, a 15 per cent increase over 1967 [59]. Growth over the past decade has been comparable, and it can be anticipated that a comparable rate of growth will continue, not only in the

United States but in the remainder of the world. Among the groups of chemicals showing high rates of growth have been the plastics, pesticides, and resins. U.S. production of DDT in 1968 was 140 million pounds or about 6×10^{10} grams; production of the dioctyl phthalates, one of the principal groups of plasticizers, was 440 million pounds [59]. Production figures of the polychlorinated biphenyls (PCB), industrial compounds that like the DDT compounds have become widespread pollutants in the global environment [32], [50] are not available because of current U.S. practice that protects proprietary information.

Eventually all of the organic chemicals synthesized become waste products and their potential threat to the environment is evidently a function of their input, their chemical and biological stability, mobility, toxicity, and of the properties of breakdown products. Carcinogens and mutagens clearly pose a much more significant threat to human populations, which value each individual life, than to wildlife populations which can much more readily withstand the loss of individuals as long as the reproductive capacity is not impaired. Although wildlife populations might serve to monitor increases in environmental levels of chemical carcinogens or mutagens, damage to a species from such chemicals has not been documented. Environmental chemicals that lower the reproductive capacity of a population pose a much more serious threat to wildlife.

3. The thin eggshell phenomenon

In 1967 Ratcliffe [43] published a paper that showed for the first time that physiological changes in wildlife species were correlated with geographical and temporal patterns of environmental pollution. Eggs of the Peregrine Falcon (*Falco peregrinus*), Sparrowhawk (*Accipiter nisus*) and Golden Eagle (*Aquila chrysaetos*) laid in several regions of Great Britain after 1947 tended to have lower amounts of calcium carbonate in the shells than did eggs laid by the same species in the past. No changes were detected in the Central and Eastern Highlands of Scotland which are comparatively remote from pollution sources. Hickey and Anderson [28] subsequently showed that many populations of species of fish-eating and raptorial birds in North America were also suffering from the same syndrome and, as in Britain, the changes were first detectable in eggs laid in 1947. Ratcliffe [44] has documented the species for which shell thinning has been shown in Britain. In North America the phenomenon has now been documented in the following families of birds: the pelicans, *Pelecanidae* [4], [48]: cormorants, *Phalacrocoracidae* [4]; hawks, *Accipitridae*, [28]; falcons, *Falconidae* [28]; ospreys, *Pandionidae* [28]; herons, *Ardeidae* [18]; gulls, *Laridae* [28]; auks, *Alcidae* [22]; and petrels, *Procellariidae* [14].

Possible causes of the phenomenon were first discussed by Ratcliffe [43]. In North America the scientific considerations have frequently been obscured by political and economic factors [15]. Of the species affected, the Brown Pelican (*Pelecanus occidentalis*) is perhaps best known to the general public and has been

frequently mentioned in public hearings [15], [58]. The available pollutant data and relevant biological information on this species are discussed in detail below.

4. The Brown Pelican: a species endangered by pollution

The Brown Pelican is a coastal species and is not found on inland waters. It feeds primarily upon fish and is therefore exposed to pollutants by eating contaminated fish. In the United States breeding colonies have been found in Central and Southern California, along the Gulf States, and on the Atlantic coast north to North Carolina [1]. The present status of the Brown Pelicans in the United States has recently been reviewed by Schreiber and Risebrough [54]. Although it is the state bird of Louisiana, no wild birds have nested there since 1961. The reasons for their sudden disappearance and for the death of adult birds has not been documented, but factors other than a decrease in reproductive capacity were evidently responsible.

Anderson and Hickey [3], in comparing eggs of the Brown Pelican obtained after 1949 with those obtained before 1943, found that recent eggs from Florida, Texas, and California were thin-shelled. Field observations carried out in California and northern Baja California in 1968 showed that reproduction was abnormally low [53]. More extensive field studies in 1969 on Anacapa Island, the only breeding site in California, showed that the birds could no longer reproduce because virtually all of the eggs laid by the pelicans were so thin-shelled that they collapsed during incubation [51]. The eggs laid in 1970 and 1971 were also thin-shelled and very few young were hatched [48].

Following the discovery of the breeding failures on Anacapa Island in 1969 J. R. Jehl visited Mexican islands along the western shore of Baja California that had been traditional nesting sites of Brown Pelicans. On the Islas Coronados near San Diego, California, the pelicans were experiencing the same kind of reproductive failures that had been observed on Anacapa. Most of the eggs laid by the birds were crushed; the remains were scattered about the colony. To the south, on Isla San Martin, some shell thinning was evident, and discarded, broken eggs were found. Nevertheless, some survived to permit hatching of young birds. On the Islas San Benitos still further to the south, most of the eggs did not appear to be thin-shelled upon superficial examination [31].

Studies of the breeding biology of the Brown Pelicans in Florida had been begun in 1968 by R. W. Schreiber. In 1969 and 1970 a total of 87 eggs were obtained from four different colonies for thickness measurement and pollutant analysis. No reduction in population numbers of the Florida pelicans was evident and apparently normal numbers of young birds were being fledged [54].

The methodology of measuring concentrations of chlorinated hydrocarbons in the eggs has been described elsewhere [45], [47]. The eggshell thickness measurements were made with a micrometer at the girth of the egg, and represent the means of at least four measurements of each egg. The thickness value

includes the proteinaceous membrane attached to the shell. The mean thickness of this membrane was 0.11 mm and this value should therefore be subtracted from the measurements presented to obtain the thickness of the calcium carbonate layer.

TABLE I

DDT AND PCB RESIDUES IN YOLK LIPIDS OF BROWN PELICANS, DDE-PCB RELATIONSHIPS, AND ASSOCIATED EGGSHELL CHANGES

Concentrations in parts per million of the lipid weight. Total DDT is the sum of *p,p'*-DDE, *p,p'*-DDD and *p,p'*-DDT.

From Risebrough, Gress, Baptista, Anderson and Schreiber [48].

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

General breeding area Colony	Sample size	Residue means			PCB *versus* DDE	Mean eggshell thickness (mm)
		Total DDT	*p,p'*-DDE	PCB	*r* value	
Pacific Coast						
Anacapa, California	65	1,223	1,176	210	0.520***	0.32
Los Coronados	28	1,158	1,109	266	0.495**	0.34
San Martin	6	429	411	72	0.970**	0.45
San Benitos	10	128	121	39	0.973***	0.51
Gulf of California	4	13	11	4	0.952	0.56
Atlantic and Gulf Coasts						
Tampa Bay, Florida, 1969	14	56	37	120	0.837***	0.51
Tampa Bay, Florida, 1970	21	36	26	69	0.346	0.51
Cocoa Beach, Florida, 1970	22	32	28	64	0.837***	0.50
Hemp Key, Florida, 1970	20	24	18	45	0.830***	0.52
Vero Beach, Florida, 1970	10	26	21	77	0.523	0.50
All Florida	87	34	26	71	0.701***	0.51

A summary of results is presented in Table I. Because distribution of pollutant residues in these samples was not Gaussian [6], confidence limits of the arithmetic means of pollutant concentrations are not given.

Concentrations of the DDT compounds are evidently much higher in the west than in Florida. The arithmetic mean of the concentrations of total DDT residues in the yolk lipids of the 87 Florida eggs was 34 parts per million, whereas the arithmetic mean of the DDT concentrations of 65 broken eggs from Anacapa Island was 1,223 ppm. Furthermore, a north-south gradient is evident along the west coast, with concentrations highest in southern California. PCB concentrations are also higher in southern California than in Florida. In addition, many of the eggs were analyzed for dieldrin and endrin, two highly toxic organochlorine compounds used as insecticides that have also become marine pollutants. Five eggs obtained on Anacapa in 1971 were analyzed for mercury, lead, cadmium, chromium and several other heavy metals, but to date the metal analyses of eggs from other regions have not yet been finished.

Nevertheless, it is possible to determine the nature of the relationships between the magnitude of the thinning effect and the concentrations in the eggs of the DDT and PCB compounds, the two pollutant groups suspected to be major contributors to the shell thinning phenomenon. Table II lists for all eggs analyzed the shell thickness, the concentrations in the yolk lipids of the principal DDT compound, *p, p'*-DDE, the total concentrations of the three DDT compounds, *p, p'*-DDE, *p, p'*-DDD and *p, p'*-DDT, and the concentrations of PCB.

The thinning cannot be considered a direct consequence of the amount or kind of pollutant present in the egg, since pollutants are laid down with the yolk or albumin before the shell is formed. Rather, the degree of thinning must be a result of the physiological condition of the shell gland where the shell is formed. Since the shell gland is highly vascular, the sensitive sites are exposed to pollutants in the blood. It is therefore assumed that the thinning effect is a function of the pollutant levels in the blood. Since the blood carries both the yolk lipids and associated pollutants to the ovary where they are deposited in the forming yolk, it is further assumed that the pollutant concentration in the yolk lipid is also a function of its concentration in the blood. The relationships found between the thinning and the pollutant concentrations in the egg are therefore indirect.

It is also necessary to make an assumption about the comparability of the east coast and west coast eggs. The Florida Brown Pelicans belong to a different geographical race, *Pelecanus occidentalis carolinensis* than do the western birds, which are *Pelecanus occidentalis californicus*. The eastern birds are smaller, with slightly different body coloration [62]. As a result of their smaller body size, the Florida birds lay slightly smaller eggs than do the western pelicans. Thus, the mean thickness of 172 eggs obtained in Florida prior to 1943 and now preserved in museums was 0.557 ± 0.004 mm (95 per cent confidence limits) whereas the mean thickness of 111 eggs obtained on the west coast prior to 1943 was 0.571 mm [3]. The measured thickness of all Florida eggs obtained in 1969 and 1970 was therefore multiplied by the constant factor 1.03 to make the east coast eggs comparable to those from the west coast for the consideration of pollutant effects. Thus the thickness data for the Florida eggs of Table I represent the original values, whereas those of Table II have been multiplied by the correction factor.

From examination of Table I it is apparent that there is a marked difference between the distributions of the PCB and DDT compounds on the east and west coasts. In the east, PCB is more abundant than is DDE, whereas in the west DDE is more abundant than PCB. Moreover, within most colonies there is a highly significant linear relationship between the concentrations of DDE and PCB in the pelican eggs (Table I). Both compounds are very resistant to chemical and biological degradation, both are very insoluble in water and highly soluble in fats, and both move through ecosystems in comparable ways. Comparable relationships have been observed in other ecosystems [50]. Other pollutants with similar biological and chemical properties but that are undetected by the methodology that measures chlorinated hydrocarbons might therefore show a comparable correlation with the concentrations of DDE. Correlations between

TABLE II

DDE, TOTAL DDT, AND PCB CONCENTRATIONS IN BROWN PELICAN EGGS
PARTS PER MILLION OF THE YOLK LIPIDS

Locality	Sample no.	Thickness (mm)	DDE	Total DDT	PCB
Anacapa	15	0.14	2500	2571	452
	85	0.19	1337	1382	184
	14	0.20	1003	1055	115
	65	0.20	1780	1875	315
	64	0.21	1780	1862	139
	58	0.22	617	647	177
	59	0.22	1310	1376	214
	66	0.22	2145	2219	356
	101	0.23	1623	1660	166
	43	0.23	1638	1694	246
	1	0.23	1760	1764	177
	41	0.23	1925	2020	289
	84	0.24	1410	1454	175
	3	0.25	1458	1525	296
	19	0.25	1495	1586	205
	50	0.26	860	965	324
	72	0.26	1220	1296	260
	31	0.26	1290	1353	188
	27	0.26	1380	1425	208
	86	0.27	950	981	109
	55	0.28	600	648	204
	46	0.28	641	663	89
	52	0.28	1205	1280	320
	70	0.29	569	569	265
	2	0.29	862	898	138
	12	0.29	1170	1215	198
	70	0.29	1373	1442	191
	48	0.29	1940	1956	193
	32	0.29	2100	2192	316
	17	0.30	613	632	122
	102	0.30	800	850	305
	24	0.30	1060	1096	203
	93	0.30	1418	1478	396
	103	0.30	1430	1490	250
	124	0.30	2010	2031	230
	104	0.30	2379	2440	214
	105	0.31	301	305	46
	49	0.31	1560	1622	256
	63	0.32	1680	1758	204
	21	0.34	748	780	150
	42	0.34	1405	1473	218
	69	0.34	1435	1490	261
	16	0.35	969	999	143
	54	0.35	1080	1132	229
	37	0.36	949	975	173
	67	0.36	1275	1316	132
	22	0.36	1355	1418	175
	74	0.37	703	734	236
	30	0.37	1055	1091	220
	29	0.37	1140	1176	212

TABLE II (continued)

Locality	Sample no.	Thickness (mm)	DDE	Total DDT	PCB
Anacapa (continued)	90	0.39	390	403	119
	88	0.39	441	462	144
	79	0.39	882	928	147
	96	0.40	700	733	171
	91	0.41	895	943	216
	11	0.41	1430	1513	232
	94	0.42	745	783	216
	87	0.42	850	879	164
	92	0.42	890	921	185
	89	0.44	416	438	87
	98	0.46	625	645	216
	95	0.46	785	824	199
	97	0.47	755	768	236
	100	0.49	657	672	237
	99	0.49	686	708	206
Los Coronados	2137	0.23	2610	2620	464
	2136	0.25	1598	1686	590
	2140	0.25	1135	1185	162
	2288	0.25	448	472	202
	2128	0.27	1730	1807	206
	2130	0.27	1410	1499	171
	2133	0.27	1732	1823	230
	2139	0.28	2221	2299	302
	2135	0.29	1470	1475	270
	2131	0.31	1422	1527	178
	2129	0.32	1598	1710	338
	2078	0.33	1330	1393	412
	2074	0.34	1140	1206	213
	3138	0.34	622	650	85
	2072	0.34	720	770	218
	2289	0.37	691	730	204
	2132	0.37	1500	1579	167
	2286	0.38	685	712	481
	2134	0.39	1985	2050	530
	2076	0.39	335	351	130
	2287	0.40	246	258	102
	2285	0.40	321	334	110
	2075	0.42	1420	1493	1060
	2077	0.42	340	370	77
	2284	0.44	98	104	41
	2294	0.44	1035	1055	191
	2073	0.44	450	486	157
	2079	0.45	748	792	154
San Martin	2184	0.27	1710	1779	194
	2082	0.37	384	397	73
	2080	0.49	103	114	70
	2083	0.49	118	122	30
	2081	0.52	59	65	37
	2151	0.54	91	95	28
San Benito	2067	0.39	636	679	234
	2066	0.40	247	252	39
	2070	0.44	85	90	35
	2297	0.49	25	28	11

TABLE II (continued)

Locality	Sample no.	Thickness (mm)	DDE	Total DDT	PCB
San Benito (continued)	2298	0.53	25	27	12
	2071	0.54	65	73	17
	2069	0.55	36	38	14
	2068	0.58	17	19	7
	2020	0.59	40	43	11
	2065	0.59	30	33	8
Gulf of California	9000	0.50	9.3	11.5	1.8
	2182	0.58	12.9	15.8	5.1
	9001	0.59	6.6	7.5	2.0
Florida	2346	0.403	84.7	124	61.6
	2123	0.433	63.0	105	340
	2328	0.433	26.5	39.7	92.5
	2110	0.464	51.2	65.5	200
	2330	0.464	45.8	53.5	70.5
	2338	0.474	23.2	31.4	95.5
	2122	0.484	55.4	82.5	214
	2340	0.494	24.6	36.6	48.3
	2341	0.495	15.9	18.8	48.8
	2117	0.505	45.5	65.1	163
	2121	0.505	58.0	91.5	118
	2329	0.505	16.9	28.2	42.3
	2336	0.515	15.8	21.7	51.3
	2337	0.515	36.0	54.1	99.5
	2111	0.525	39.6	50.2	58.0
	2124	0.525	44.0	74.5	153
	2109	0.536	38.8	55.2	96.0
	2114	0.536	31.4	53.5	100
	2343	0.536	43.9	56.1	163
	2112	0.546	21.4	30.1	75.0
	2125	0.546	28.8	45.2	59.0
	2326	0.547	23.6	33.1	79.2
	2331	0.547	28.0	41.1	121
	2333	0.547	15.5	20.5	36.3
	2334	0.547	18.5	22.0	70.2
	2113	0.556	23.2	35.7	48.3
	2335	0.556	29.3	38.2	59.3
	2339	0.556	17.8	22.4	50.0
	2119	0.567	10.2	14.0	29.9
	2120	0.567	10.7	15.8	27.5
	2327	0.567	19.0	29.8	64.8
	2332	0.567	15.3	20.6	38.0
	2344	0.567	14.6	17.9	34.8
	2342	0.577	19.8	22.4	83.0
	2345	0.598	19.7	27.6	36.2
	2348	0.474	26.6	31.6	60.2
	2349	0.474	35.7	43.5	114
	2357	0.474	18.4	23.2	78.2
	2352	0.485	32.7	38.3	88.4
	2360	0.485	16.6	19.2	26.8
	2354	0.495	38.6	45.5	96.8
	2356	0.495	40.1	45.6	89.6
	2366	0.495	39.7	43.6	68.8

TABLE II (continued)

Locality	Sample no.	Thickness (mm)	DDE	Total DDT	PCB
Florida (continued)	2350	0.505	22.8	30.1	56.8
	2363	0.505	22.0	24.7	34.7
	2368	0.505	65.1	74.3	134
	2358	0.526	22.0	25.1	45.7
	2362	0.526	22.7	24.9	35.3
	2364	0.526	19.9	22.8	49.5
	2367	0.526	23.1	27.7	66.2
	2351	0.536	41.0	44.6	69.8
	2353	0.536	31.2	34.7	62.5
	2355	0.547	29.6	33.9	66.3
	2361	0.547	13.3	15.0	21.7
	2365	0.547	21.7	23.9	50.7
	2359	0.556	19.9	22.3	72.9
	2347	0.577	8.4	10.6	15.1
	2372	0.443	42.3	59.9	87.6
	2380	0.443	41.0	47.9	180
	2371	0.474	22.6	28.1	40.7
	2379	0.485	30.1	39.2	122
	2382	0.485	13.8	17.6	52.8
	2376	0.495	10.1	12.9	32.7
	2387	0.505	33.0	42.2	77.7
	2386	0.515	14.2	18.5	50.3
	2369	0.536	10.5	12.3	13.3
	2374	0.547	16.0	20.4	10.5
	2375	0.547	20.6	30.2	43.2
	2378	0.547	13.2	19.0	20.9
	2373	0.567	9.9	13.5	17.2
	2377	0.567	4.4	5.3	15.2
	2381	0.567	12.4	15.1	31.3
	2384	0.567	20.2	24.1	19.1
	2385	0.567	18.1	29.5	38.0
	2370	0.577	5.0	6.1	3.7
	2383	0.597	19.0	25.0	19.4
	2388	0.629	9.5	10.8	16.8
	2389	0.474	20.8	24.1	57.0
	2398	0.485	22.9	29.9	129
	2390	0.495	18.1	22.0	64.8
	2392	0.505	19.6	24.1	43.1
	2397	0.526	11.1	11.7	32.7
	2394	0.526	37.1	45.9	105
	2391	0.536	22.1	25.2	154
	2393	0.536	20.9	30.2	71.2
	2396	0.536	24.2	31.6	64.3
	2395	0.547	9.2	11.8	53.3

concentrations of a pollutant and the environmental effect in only one locality must therefore be interpreted with caution.

Table III lists the simple correlation coefficients, r, found to exist between thickness and the pollutants for each locality [20]. In all cases there is a significant negative correlation between thickness and the concentrations of either DDE or total DDT. In two cases there was no significant correlation between

TABLE III

SIMPLE LINEAR CORRELATION (r) BETWEEN SHELL THICKNESS AND CONCENTRATIONS OF DDE, TOTAL DDT AND PCB IN BROWN PELICAN EGGS

From Risebrough, Gress, Baptista, Anderson and Schreiber [48].
$^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$.

Sample	N	DDE	Total DDT	PCB
All eggs	199	−0.8512***	−0.8534***	−0.6128***
All Florida	87	−0.5794***	−0.5509***	−0.4987***
Anacapa	65	−0.5605***	−0.5659***	−0.2527*
Los Coronados	28	−0.5994***	−0.6003***	−0.0987
All eggs, DDE < 100 ppm	101	−0.5059***	Not determined	−0.4746***
All eggs, DDE > 99 ppm	98	−0.5985***	−0.6016***	−0.1545
West Coast, DDE < 100	14	−0.5961*	−0.5946*	−0.6736**

thickness and PCB; DDE concentrations were high in each. In only one category, the west coast eggs with concentrations of DDE less than 100 ppm, was the correlation coefficient for PCB higher than for DDE. Because of the relationships between DDE and PCB (Table I) this approach does not permit any conclusion about the contribution of PCB to shell thinning, especially when DDE concentrations are low.

The plot of the curve between thickness and DDE [48] shows a very steep drop in thickness with small concentrations of DDE, with a decreasing slope as DDE concentrations increase, suggesting a logarithmic rather than a linear relationship. A logarithmic relationship is consistent with a plausible physiological model, consisting of a finite number of sensitive sites in the shell gland. The number of pollutant molecules required to block each site would then be a function of the number of sites already blocked.

The DDE and PCB concentrations of Table II were therefore transformed to their logarithms, as well as to their square roots, squares, exponentials, and cross products. Of these, including the original variables, the logarithm of DDE concentrations showed the highest linear correlation with thickness, ($r = -0.8906$, $N = 199$). The logarithm of DDE was therefore selected as the initial independent variable of a regression equation relating pollutant concentrations with thickness. In order to determine whether PCB might be contributing to the mathematical relationship between ln DDE and thickness, the data of Table II were analyzed with a program that determines stepwise regression (BMD 02R, Biomedical Computer Programs (edited by Dixon, 1970) on the Control Data Corporation 6400 computer of the University of California, Berkeley. The other potential independent variables consisted of DDE, ln PCB, PCB, ln (DDE × PCB) and (DDE × PCB). The program first selected the parameter that is most highly correlated with the dependent variable, in this case the logarithm of the DDE concentration, with $F = 754$, and degrees of freedom 1,197. The program then entered DDE with a partial F of 14.4155, degrees of freedom (1,197), ($p < 0.005$). The other variables did not contribute

significantly to the regression ($p > 0.10$). The program then added DDE to ln DDE, with $F = 409$, d.f. (1,196). The part al F of the cross product DDE × PCB was 4.54, $0.02 < p < 0.05$. In these and subsequent operations none of the other variables contributed significantly to the regression.

The mathematical relationship between thickness and the added linear DDE term is also consistent with the biological model, since increasing amounts of DDE in the lipo-protein membranes of the shell gland could adversely affect ion transport across them, in addition to the blocking of sensitive sites. It is more difficult to interpret the contribution of the cross-product DDE × PCB, especially when no significant contribution of this term appeared in the first operation of the program. Synergistic effects between PCB and DDT compounds have been observed in other biological systems [37], but the magnitude of the effect here is not sufficiently convincing to conclude that PCB is modifying the DDE effect on pelican eggs. This is clearly a borderline case in the attempts to determine causal relationships between pollutants and a pollutant effect. An experimental approach is clearly preferable.

Unfortunately an experimental approach is not always feasible in the evaluation of pollutant effects upon ecosystems, especially when the ecosystems are unique. Species such as pelicans are extremely difficult to keep in captivity, especially under conditions that would approximate those encountered in their natural habitat. Because different species of birds differ widely in their physiological responses as a result of different food habits, different habitat selection, the necessity of some species to eliminate excess salt, and of many other factors, extreme caution must be made in extrapolating conclusions about the real world without recourse to adequate experimentation. Thus, Jukes [15] has written ". . . without conducting controlled experiments, DDE has been blamed for the thin shells of eggs laid by pelicans on Anacapa Island."

The conclusions about the shell thinning effects of DDE and PCB on pelican eggs based upon field data might, however, be compared with the results of an experiment designed to test the combined effects of DDE and PCB upon the thinning of eggs of mallard ducks (*Anas platyrhynchos*), [46]. Fifty mallard hens were divided at random into eight groups of five birds and one group of ten. The latter served as spare birds in case of need, but were not used. Each group of five was placed in a cage with two drakes. Two groups served as control, two groups were fed DDE at a concentration of 40 ppm, dry weight, of their diet, two were fed 40 ppm PCB (Aroclor 1254) and the remaining two received a combination of 40 ppm DDE + 40 ppm PCB. Instead of thickness, a thickness index, derived by dividing the weight of the dried shell in grams by the product of the length and breadth in cm^2 was used [43]. The data are summarized in Table IV. The distribution of shell thickness indices in each group was approximately normal [46], permitting determination of the 95 per cent confidence limits of the mean by means of the t test [20].

Thus PCB increased slightly the shell thickness index and DDE caused a significant reduction. Shell thickness index of birds receiving the combination

TABLE IV

SHELL THICKNESS INDEX CHANGES OF MALLARD DUCK EGGS

From Risebrough and Anderson [46].

Diet group	N	Thickness index $\pm$ 95% C.L.
Control	500	0.2098 ± 0.0014
DDE, 40 ppm	473	0.1745 ± 0.0017
DDE, 40 ppm and PCB, 40 ppm	264	0.1700 ± 0.0019
PCB, 40 ppm	388	0.2143 ± 0.0020

was slightly lower than that of birds receiving DDE alone. One of the birds on the DDE diet, however, began to lay thick-shelled eggs toward the end of the experiment [46]. If these eggs, representing the top eight values, are omitted from the calculations, the index becomes 0.1729 ± 0.0013, not significantly different from combination birds. The situation is again borderline, but the combination of DDE and PCB had other effects upon reproduction [46].

Other experiments have also shown that PCB alone has no effect upon the shell thickness of eggs of mallard ducks [26] or of ring doves, *Streptopelia risoria* [40].

More difficult is the evaluation of possible contributions by other pollutants, alone or in combination with DDE, which were not measured by the techniques used. The relationship between thickness and the logarithm of DDE fitted well the data from all colony sites, including both the Atlantic and Gulf coasts of Florida, California and western Mexico, except that at higher concentrations of DDE, thickness decreased more rapidly than predicted. These eggs were all from southern California and adjacent areas of Mexico. As indicated above, addition of a linear DDE term significantly improved the fit, and was furthermore consistent with a biological model. The possibility can not be eliminated however, that other pollutants are contributing slightly to the DDE effect. No correlation was found between thickness and concentrations of either dieldrin, which shows a slight shell-thinning effect in mallard eggs [36], or endrin [48]. The mean arithmetic concentrations of total mercury in the wet weight contents of the five eggs obtained in 1971 was 0.083 ppm (standard deviation 0.036 ppm) [2], lower than the mercury concentrations found in 85 eggs of 13 species of fish-eating birds from the Great Lakes region, all of which showed lesser amounts of thinning than the Brown Pelicans [17]. Mercury residues in a fish-eating species from Antarctica were only slightly lower [2]. Cadmium levels in these eggs were in the order of 0.1 ppm, dry weight, in the same range as the Antarctic species [2]. Lead levels were not above background (maximum concentration 0.1 ppm dry weight).

DDE appears therefore to be the major cause of the thinning of Brown Pelican eggshells. The amount of data presently available is not sufficient to show whether pollutants other than the DDT and PCB compounds are contributing to a minor extent.

5. Pollutant effects on other wildlife species

Among species of fish-eating and raptorial birds, patterns of regional decline and reproductive failures have almost always been associated with thinning of eggshells [28], [43], [44], [48]. The available evidence indicates that DDE is the pollutant primarily responsible for this phenomenon. Other potential contributors such as dieldrin [36] or metallic mercury [56] are usually much less abundant in environmental samples than is DDE.

A statistically significant inverse correlation between shell thickness and the concentrations of DDE in the egg have been recorded for: Herring Gull (*Larus argentatus*) [28]; Double-crested Cormorant (*Phalacrocorax auritus*) [4]; White Pelican (*Pelecanus erythrorhynchos*) [4]; and the Peregrine Falcon [11]. Low concentrations of DDE in the diet comparable to those found in fish have produced shell thinning under controlled experimental conditions in Mallard Ducks [25], [46], American Kestrel (*Falco sparverius*) [63] and Japanese Quail (*Coturnix*) [55]. Shell thinning has also been induced experimentally with DDE in Ring Doves [41].

Like the DDT compounds, dieldrin may be dispersed through the atmosphere [49], [57]. The greatest hazard of dieldrin exists to fish-eating birds such as the Bald Eagle (*Haliaeetus leucocephalus*) [39] and Common Egret (*Casmerodius albus*) [18] and to the Peregrine Falcon [44] which may accumulate lethal levels from fish or birds which are not themselves harmed.

Long term effects of PCB upon wildlife and on the environment in general are not known. PCB may increase susceptibility to infectious agents such as virus diseases [21]. Like other chlorinated hydrocarbons PCB increases the activity of liver enzymes that degrade sex hormones [50]. Highly toxic byproducts may also be associated with PCB [60], [61].

Illness of zoo animals in New York City caused by lead poisoning, presumably by breathing lead contaminated air, is an indication of the hazards of lead additives in gasoline to both wildlife and man [7].

The use of alkyl-mercury compounds as seed dressings in Sweden caused the death of numbers of seed-eating birds [10]. In Finland mercury may have contributed to the decline of the White-tailed Sea Eagle (*Haliaeetus albicilla*) in regions where the species feeds upon marine fish and marine birds [27].

In all cases the compounds suspected to cause damage have been the heavy metals or relatively persistent chlorinated hydrocarbons. No damage to a population has been linked to date with a compound acting primarily as a mutagen or teratogen.

6. Monitoring environmental levels of mutagens and teratogens

The number of individuals in wildlife populations that would be affected by environmental mutagens or teratogens is inevitably small. Without careful monitoring they would not be observed in most species. Because of high rates of natural mortality it is unlikely that deaths of comparatively few individuals

would affect population numbers. If a wildlife population were to be used, however, to monitor the levels of environmental chemicals that cause birth defects, the following criteria might apply:

(1) a species that occupies a relatively high position in the food web, such as a fish-eating bird, can be expected to accumulate greater amounts and varieties of pollutants that might cause birth defects than would a seed-eating species;

(2) the population studied should occupy a relatively polluted habitat, but other populations of the species should occupy relatively clean environments;

(3) it should be possible to examine large numbers of young at the time of birth.

These conditions are met in a colony of Common Terns on Great Gull Island in Long Island Sound, which has been studied intensively by ornithologists from the American Museum of Natural History. The terns prey upon small fish, and Long Island Sound is one of the areas of the sea that are heavily polluted with such industrial chemicals as PCB [24]. Nests are marked when the eggs are first laid and examined at the time of hatching. The young terns are marked with a unique combination of bands, some of which are colored. In 1970, 33 or 1.5 per cent of the 2,316 young Common Terns possessed a gross abnormality. Most frequently observed was a loss of feathers when the birds were two to three weeks old. This phenomenon appears to be pollutant-induced [24]. Other abnormalities included beak, eye and foot deformities. At the present time data from control colonies are not sufficient to permit conclusions that the incidence of abnormalities is significantly above the expected. If levels of a chemical that causes birth defects are increasing, however, in the environment, the effects might first be noted in a polluted area adjacent to a highly industrialized, densely populated region such as Long Island Sound. Increasing levels of deformities in the tern colony on Great Gull Island would serve as an "early warning" to the human populations which share the environment with the terns.

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Discussion

Question: E. B. Hook, Birth Defects Institute, Albany Medical College

Regarding the observation of congenital defects in the terns in Long Island Sound:

(1) Over how long a period of time have the birds been systematically examined?

(2) What is the observed incidence rate of these defects?

(3) Where else do these birds breed and/or feed?

(4) Have these defects been observed in other species of birds and/or have any other birds been systematically examined?

(5) Are there internal defects found on autopsy of those birds with external limb defects in higher frequency than in birds without external defects?

Reply: R. Risebrough

(1) Detailed studies were begun in 1969 and have continued through 1971.

(2) In 1969 only three of 3,160 young terns showed abnormalities. Both

Roseate Terns and Common Terns were studied. In 1970, 3,122 young terns of both species were examined; 38 showed abnormalities. Data for 1971 have not yet been compiled, but several abnormalities have been noted to date.

(3) At least some of the birds winter in the Caribbean, as shown from the banding returns. The Common Tern also breeds in many localities along the east coast and in eastern and middle North America.

(4) To my knowledge they have not been looked for in other species.

(5) These were not looked for, but the specimens have been preserved for future studies, as required.

Question: E. Tompkins, Human Studies Branch, Environmental Protection Agency

What is your explanation for the sharp dichotomy between the thickness of the egg shells in museums in England between 1944 and 1945? It seems to be a sharp drop to associate with the gradual buildup of an environmental pollutant.

Reply: R. W. Risebrough

The sharpest drop occurred between 1946 and 1947. The year 1947 was also the year when thin shelled eggs of the Peregrine were first observed in Massachusetts and California. The curve relating shell thickness to DDE concentrations in the Brown Pelican eggs, and in eggs of other species as well, shows a very sharp initial drop with low concentrations of DDE. In a given ecosystem, the Peregrine always has the highest concentrations of DDE. The relatively sharp drop in thickness is, therefore, compatible with other observations.

Question: Alexander Grendon, Donner Laboratory, University of California, Berkeley

I understood you to say that the eggs with low levels of DDE were museum specimens. Since these would be empty shells, how did you determine their DDE content?

Reply: R. W. Risebrough

No, the eggs with low levels of DDE were from Florida or from the Gulf of California. Chlorinated hydrocarbons were, therefore, measured in the egg contents. I am not aware of any attempts to measure DDE in egg shells and I doubt that they would be worthwhile.

Question: Burton E. Vaughan, Ecosystems Department, Battelle Memorial Institute, Richland, Washington

Would you say something about DDT levels in open ocean as compared to estuarine and other coastal water samplings? Are open ocean levels high in relation to the levels necessary for egg shell thinning?

Reply: R. W. Risebrough

California coastal waters are a special case, since they have received huge amounts of DDT compounds from the effluent of a factory in Los Angeles. The Peregrine Falcons of Amchitka Island in the North Pacific show slight but significant amounts of shell thinning and DDE levels in the fat are in the order of 100–300 ppm. On the east coast, the gannets which feed on fish in the coastal

waters show significant thinning of shells. Of the true sea birds, the petrels, shearwaters, and albatrosses, shell thinning has been looked for and found only in the Ashy Petrel of California. But levels in some of the other species appear sufficiently high to cause a slight amount of thinning.

Question: E. J. Sternglass, School of Medicine, University of Pittsburgh

Have you examined the possible effect of radioactive fallout on animal reproduction, for instance as observed by Dr. Norman B. French at U.C.L.A. for a natural population of desert animals exposed to very low levels of daily doses?

Reply: R. W. Risebrough

Ratcliffe's initial studies of shell thinning showed that the geographical patterns of thinning corresponded to pollution patterns. Remote areas in Scotland, which presumably received as much fallout as southern England, showed relatively slight thinning of the birds' eggs. In North America, the pattern of shell thinning has corresponded closely to pollution patterns rather than to one shown by fallout. The species first affected have invariably been those highest in the food webs. Related species inhabiting the same areas have either not shown thinning or have shown it several years later than the most sensitive species. The few available data on radioactivity in birds show that body burdens are higher in mountain areas than along the coast. But coastal species have shown many more symptoms of reproductive failures than have mountain species.

Question: B. G. Greenberg, School of Public Health, University of North Carolina, Chapel Hill

I wonder if Dr. Risebrough can help us to transfer or extrapolate the findings about thickness of eggshells in bird studies to human populations. Is the formation of bone structure or teeth likely to be affected and are there other organs affected?

Reply: R. W. Risebrough

The shell thinning phenomenon appears to be associated with the membranes of the shell gland. Most likely the transport of calcium ions is inhibited, perhaps by inactivation of an ATPase. Another potentially sensitive process is the transport of bicarbonate or carbonate across the membrane and the maintenance of the pH gradient. The deposition of calcium carbonate in the eggshell is, therefore, a very different process from the deposition of calcium phosphate crystals in bone. If a human system were sensitive to DDE it would, therefore, most likely have membranes across which ions are transported. But even the relatively uncontaminated pelicans from Florida have much more DDE than does the North American human population.