EXPOSURE OF MARINE BIRDS TO ENVIRONMENTAL POLLUTANTS

UNITED STATES DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE
Wildlife Research Report 9

WILDLIFE RESEARCH REPORTS

This series comprises reports of research relating to birds, mammals, and other wildlife and their ecology, and specialized bibliographies on these, issued for wildlife research and management specialists. The Service distributes these reports to official agencies, to libraries, and to researchers in fields related to the Service's work; additional copies may usually be purchased from the Division of Public Documents, U.S. Government Printing Office.

Library of Congress Cataloging in Publication Data

Ohlendorf, Harry M
Exposure of marine birds to environmental pollutants.

(Wildlife research report; 9)
Bibliography: p.
1. Sea birds—Physiology. 2. Pollution—Environmental aspects. 3. Pollution—Toxicology. 4. Oil spills and wildlife. I. Risebrough, Robert W., joint author. II. Vermeer, Kees, joint author. III. Title. IV. Series.
QL698.044 598.2'2'4 78-5240

EXPOSURE OF MARINE BIRDS TO ENVIRONMENTAL POLLUTANTS

By Harry M. Ohlendorf
Robert W. Risebrough
Kees Vermeer

UNITED STATES DEPARTMENT OF THE INTERIOR

FISH AND WILDLIFE SERVICE

Wildlife Research Report 9

Washington, D.C. • 1978

Contents

Exposure of Marine Birds to Environmental Pollutants[1]

by

Harry M. Ohlendorf

U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, Maryland 20811

Robert W. Risebrough and Kees Vermeer

Canadian Wildlife Service
Department of the Environment
Ottawa, Ontario (RWR)
Delta, British Columbia (KV)

Abstract

It is unlikely that any marine birds remain uncontaminated by the synthetic organochlorine compounds that have become ubiquitous pollutants. Marine birds also are increasingly exposed to petroleum compounds as a result of the exploitation of undersea petroleum deposits, increased tanker traffic, and expansion of coastal petrochemical industries.

Lethal and reproductive effects of organochlorines on marine birds have been most pronounced in coastal areas receiving effluents discharged by manufacturing plants. For example, particularly severe DDT contamination in southern California and elevated levels of dieldrin and related chemicals in the Netherlands have killed local marine birds or inhibited their reproduction. Eggshell thinning, apparently resulting from exposure to DDE, is widespread among estuarine species, and eggshells of peregrine falcons (*Falco peregrinus*) have become thinner in all areas of the species' range thus far studied. In more contaminated coastal areas, reproductive success of the peregrine falcon is low. Adverse effects of organochlorines on the reproduction of other species also have been found.

The oiling of feathers and the associated mortality of marine birds are not the only adverse effects of oil pollution; ingestion of oil may cause death by dehydration by interfering with ion transport and water balance in the gut. Surfactants used to disperse oil spills also have serious consequences for marine birds. Dissolved oil fractions may kill or poison the biota the birds feed on. The physiological effects of the incorporation of more persistent compounds into marine food webs are unknown.

Contamination of marine birds by most metals and certain trace elements has not been documented, although elevated mercury levels have been observed in birds of certain estuarine and local marine environments. The significance of elevated mercury levels and small plastic particles found in the stomachs and pellets of marine birds is not yet known.

[1] A summary of this paper was presented at the 13-15 May 1975 international symposium on "Conservation of Marine Birds of Northern North America," in Seattle, Washington. The paper was written in 1975-76, and certain portions have been amended or updated as references were published. Proceedings of the symposium are being prepared for publication, but this paper on environmental pollutants is being published separately because of its great length and the delay in publication of the entire Proceedings.

Marine birds are exposed to several types of environmental pollutants: petroleum hydrocarbons, organochlorines, heavy metals, and others. Because few data are available for northern North America, potential problems for marine birds there must be judged from observations in other geographical areas.

Certain marine birds may serve as indicators of environmental pollution on a global scale because (1) they usually can be identified even in an advanced state of decomposition, after a long period of submergence in seawater, or when thickly covered with oil; (2) they are geographically widespread, often are very numerous, and feed on a wide range of marine organisms; and (3) most species nest colonially and lay large, distinctively marked eggs that are often easily collected and constitute distinct units for comparison between species (Vermeer and Reynolds 1970; Prestt 1971).

Eggs serve as particularly useful sample units for analysis of organochlorines and certain heavy metals, particularly mercury, because they do not decompose rapidly and are easily handled. Some marine bird species lay additional clutches if the first is removed; therefore, eggs may be taken without severe adverse effects on populations. This characteristic is of particular importance because studies are sometimes not begun until it is apparent that a population is declining (Prestt 1971).

Organochlorine concentrations in the egg are about equal to whole body concentrations found in the female at the time the egg was laid (Keith and Gruchy 1972). Although some microbes have the ability to metabolize organochlorine pesticides under certain conditions (Matsumura 1974), putrefaction does not significantly affect residue analysis for DDT and its metabolites (Mulhern and Reichel 1970). During incubation, however, the developing embryo appears to metabolize DDT to DDD and DDE (Abou-Donia and Menzel 1968; Blus et al. 1974b). Chemical residue concentrations can be adjusted for the loss of moisture and lipids that occurs during incubation (L. F. Stickel et al. 1973).

Eggs may not be the best tissue for measurement of all metals, because certain heavy metals apparently are not readily transferred to them (Anderlini et al. 1972). This, however, is not true of mercury. Under certain circumstances, feathers may be the best tissue to analyze for mercury residues (Stickel 1971). However, unlike liver and muscle tissue, mercury residues in feathers tend to reflect body burdens at the time the feathers were growing. The liver, which is a major organ of metabolism, or muscle appear to be the best tissues for measuring current exposure to mercury (Backstrom 1969; Vermeer and Armstrong 1972b; Fimreite 1974). Other heavy metals may be concentrated in other tissues. For example, residues in the bones, kidneys, and brain, as well as in the liver, appear to be the best measure of exposure to lead (Longcore et al. 1974b). The transfer of petroleum hydrocarbons to eggs has not been reported, but may be expected to occur.

Unless otherwise indicated, all chemical residues in the present report are expressed on a wet-weight basis.

Petroleum Hydrocarbons

Because much of the current information concerning the significance of oil pollution in the estuarine and marine environment has been included in recent reviews (National Academy of Sciences 1973, 1975a; Moore and Dwyer 1974; Evans and Rice 1974; Vermeer and Vermeer 1974a, 1974b; Farrington 1977), we have avoided an extensive review here. However, some of the general information, taken in part from these reviews, is pertinent to our subject and has been included along with that more specifically related to birds.

Crude oil and petroleum products are complex mixtures of chemicals with individual compounds numbering in the tens of thousands with wide molecular weight ranges (Farrington 1977). No one method of analysis is available that will provide reliable estimates of the concentration of the entire range of petroleum compounds, and there has yet to be a complete analysis of a single crude oil. Therefore, reports of the presence or absence of petroleum pollution should be carefully evaluated to be certain that the methods of chemical analysis employed would indeed provide the information reported.

Vanadium and nickel are present in appreciable quantities (> 100 ppm) as organometallics indigenous to crude oil, and other trace metals are picked up during production or transportation of crude oil (Whisman and

Cotton 1971).

Oil pollutants have been detected in sediments, water, and organisms in areas of large oil spills as well as from areas where no large spills have occurred in past months or years (Farrington 1977). These areas are near sources of small spills and chronic inputs. No more than an estimated 300 analyses for petroleum pollutants in sediment, water, and organism samples have been reported in the literature exclusive of reports of visible sheens on the water.

The scarcity of published measurements of the extent and severity of oil pollution in sediments and organisms is probably related to the difficulty of making meaningful analytical measurements to detect petroleum pollution (Goldberg 1972).

Sources of Oil in United States Waters

The amount of oil entering the marine environment from known sources has recently been estimated on a worldwide basis (5.3 million metric tons) as well as for the United States (1.3 million metric tons; National Academy of Sciences 1973, 1975a; Farrington 1977).

The largest amounts of oil come from normal transport and refining operations and are intentional discharges (Farrington 1977). Accidents account for only 3% of the oil that reaches marine waters of the United States and for only 5% of the world total. Oil tanker operations account for 26 times as much oil as offshore production in the United States and 24 times as much in the world total.

The oil that reaches the coastal waters from rivers and from land operations accounts for 65% of the total (Farrington 1977). The oil that reaches the oceans from the air, by dry fallout and rain, is estimated to be less than 5% of the total.

The relative importance of the various sources of oil entering the marine environment varies with location and time (Farrington 1977). For example, a large well blowout would introduce a massive amount of oil to a given location and even if averaged over a 10-year period would be the dominant source for that geographical location.

The effect of the oil from the various sources can be very different (Farrington 1977). For example, accidental spills may have both immediate acute effects and long-term chronic effects. Municipal or industrial effluents, in contrast, may have no measurable immediate impact but may have long-term chronic effects as the concentration of the petroleum chemicals builds up in the ecosystem.

Two important points relate to control of oil pollution discharges (Farrington 1977):

(1) The largest source is the chronic dribbling of oil into the coastal zone by industrial and municipal effluents, urban runoff, and river runoff carrying oil from inland areas. A substantial amount of oil, therefore, will be discharged to the coastal zone regardless of source. This amount will increase as oil consumption increases unless control steps are taken. Evidence suggests that chronic low-level pollution could be potentially more damaging to ecosystems than isolated catastrophic spills (Evans and Rice 1974).

(2) It may be safer for the total marine environment to drill and produce oil in offshore areas than to import equal quantities of oil. Approximately 0.014% of the oil produced offshore is discharged to the marine environment, in contrast to about 0.16% of the oil transported by tanker. However, this does not take into account the ecological damage that may occur in coastal areas as a result of the construction and maintenance of pipelines and onshore facilities.

Mystery oil spills, those of unknown source, account for 30% of the oil spills in U.S. waters (National Academy of Sciences 1973, 1975a). There are two possible ways to identify mystery oil spills. The first method is to tag oil tanker cargoes, pipeline loads, and storage tank contents with microscopic spheres or special chemicals. However, the size of the bureaucracy necessary to ensure accurate records renders this method impractical (Farrington 1977).

The second method is to make detailed chemical analysis of the spilled oils and potential sources. The chemical characteristics are then compared and the best match of a potential source with the spilled oil is attempted. This technique, which is called "passive tagging," makes use of the unique chemical composition of each oil to distinguish one from another and to match oils from source

and spill samples. The technique is also referred to as "fingerprinting," which is perhaps unfortunate. Many nonscientists in the field of oil pollution control have mistakenly equated "fingerprinting" in identifying mystery oil spill sources with fingerprinting in criminology. Although there are some similarities, the identification of oils is very difficult and in its infancy as a technique (Lee et al. 1974; Farrington 1977). However, following an extensive investigation by the U.S. Coast Guard, charges have been filed in the first case that was based on chemical similarities in spilled oil and a sample taken from a suspect tanker (Anon. 1975).

Transfer and Dissipation of Oil in the Marine Environment

A basic understanding of the various pathways of transfer and fate of oil has been derived from laboratory studies, field studies, and the application of knowledge of processes in the marine environment (Farrington 1977).

Many of the processes that act on the oil result in a fractionation and selective removal of certain components more rapidly than others (Farrington 1977). Lower molecular weight components of the type found in kerosene, gasoline, and in varying concentrations in crude oils and fuel oils will evaporate more rapidly than the heavier molecular weight components such as those that make up the bulk of lubricating oils. The lower molecular weight components also are more soluble than the heavier components (Moore and Dwyer 1974; Farrington 1977). When oil is placed in contact with seawater, the lower molecular weight aromatic hydrocarbons are dissolved or accommodated in the water to a greater extent than are the saturated hydrocarbon components (Boylan and Tripp 1971; Frankenfeld 1973; Boehm and Quinn 1974; Anderson et al. 1974a, 1974b; Lee et al. 1974; American Petroleum Institute 1973). When a spill occurs, however, oil may enter marine sediments and be released essentially unchanged months later (Blumer et al. 1970).

Extensive laboratory research has been directed toward a better understanding of the biodegradation of oil, and of the individual compounds or classes of compounds in oil (Davis 1967; ZoBell 1969; Ahearn and Meyers 1973; National Academy of Sciences 1973, 1975a). Several species of microorganisms, e.g., bacteria and yeasts, will completely degrade certain components of oil under the right conditions in the laboratory or in the field (Farrington 1977).

Bacteria capable of partially degrading oil have been isolated from several locations in the world's oceans (Farrington 1977). However, the rates of degradation in the various types of coastal areas are unknown. The potential pathogenicity of some species of bacteria that might increase in number near or in an oil spill area also is unknown and little is known about the toxicity of the chemicals produced by microbial degradation of oil (National Academy of Sciences 1975a). Knowledge of the biochemical pathways and products of the biochemical degradation of oil is only rudimentary (Davis 1967; ZoBell 1969; Ahearn and Meyers 1973; National Academy of Sciences 1973, 1975a; Farrington 1977).

Oil may enter marine organisms by ingestion of contaminated food and may also enter from water across membrane surfaces such as gills (Farrington 1977).

Oil incorporation into some shellfish, lobsters, and fish is reversible to some extent when the animals are placed in clean water for a period of time. Most, but not all, of the oil taken up from water by the animals was discharged within weeks to months in clean water (Blumer et al. 1970; Lee et al. 1972a, 1972b; Anderson 1973; National Academy of Sciences 1973; Stegeman and Teal 1973; Anderson et al. 1974b; Fossato 1975). However, oysters exposed for 2 months to oil from an oil spill did not appreciably reduce their oil content even after 6 months in cleaner waters (Blumer 1971; Blumer and Sass 1972). The more toxic cyclic hydrocarbons were retained longer than the less toxic straight chain compounds (Blumer et al. 1970).

Fish tested in the laboratory partially metabolized several different aromatic hydrocarbons of the type found in crude oils and fuel oils (Lee et al. 1972b). Mussels, however, did not metabolize these compounds under similar conditions, showing the undesirability of extrapolating from one group of organisms to another (Lee et al. 1972a). Equal caution is advisable in extrapolating from results of tests of those few compounds that have been tested because differences in the molecular structure can have profound effects on the rates at

which they are absorbed and metabolized (Farrington 1977).

Aside from these few examples, we have found no studies of retention of petroleum hydrocarbons after oil spills. Neither have we found studies of the uptake, retention, and discharge of petroleum hydrocarbons taken in with food. Some data suggest that food web magnification of oil does not occur in certain communities of marine organisms (Lee et al. 1972a, 1972b; Burns and Teal 1971, 1973; Anderson 1973; Stegeman and Teal 1973). There may, however, be magnification of the higher boiling fractions of the contaminants higher up in the food web (Burns and Teal 1971).

Chemical communication is highly important among marine organisms, for both interspecific and intraspecific message systems. Because very low concentrations of organic stimuli are required for communication, such processes are especially susceptible to interference by pollutants at low concentrations (Blumer 1971; Blumer et al. 1973; Jacobson and Boylan 1973; Atema and Stein 1972, 1974).

Small quantities of crude oil (0.9 ml in 100 liters of seawater) interfere with some specific, possibly chemosensory, behavior of the lobster (*Homarus americanus*). The delay period between noticing food and going after it doubled when oil was added. The water-soluble fraction of the oil alone (in the 50-ppb range) did not have a noticeable effect on behavior and feeding times. Morphological changes in odor receptors after oil exposure were not detected by light and electron microscopy. The results indicate that small amounts of oil mixed in seawater constitute a bad odor in the lobster's environment, depressing its appetite and chemical excitability (Blumer et al. 1973).

Exposure of Marine Birds to Oil

Following the 1969 spill of 650,000 to 700,000 liters of No. 2 fuel oil into Buzzards Bay and the adjacent Wild Harbor Marsh near West Falmouth, Massachusetts, essentially all the marsh organisms living in the contaminated area were affected; they all accumulated oil hydrocarbons in their tissues. Two processes apparently occur as the oil passes through the marsh ecosystem: a progressive loss in the straight chain hydrocarbons in relation to the branched chain, cyclic, and aromatic hydrocarbons, and a greater retention of the higher-boiling fractions of the contaminants by organisms higher in the food chain (Burns and Teal 1971).

Although its feathers were not oiled, a herring gull (*Larus argentatus*) that was collected in Wild Harbor had substantial amounts (584 ppm) of the whole spectrum of fuel oil hydrocarbons in its muscle but contained mostly those with straight and slightly branched chains. The brain of this bird also contained high residues (535 ppm), but with a higher proportion of the aromatic compounds. Another herring gull, collected outside the spill area, had much lower oil hydrocarbon residues in its muscle (10 ppm) and brain (15 ppm), and the aromatic compounds were not detected in the brain (Burns and Teal 1971).

Three birds that died in the 1971 San Francisco Bay oil spill contained very high residues of oil hydrocarbons in their tissues. A composite sample of liver, kidney, brain, fat, and heart tissue of a common murre (*Uria aalge*) contained 8,820 ppm, composite liver and kidney tissue of a surf scoter (*Melanitta perspicillata*) contained 1,250 ppm, and the liver of a western grebe (*Aechmophorus occidentalis*) contained 9,100 ppm oil hydrocarbons. The composite tissues (liver, kidney, brain, fat, and muscle) of a murre that had not been exposed to the oil spill contained no detectable oil hydrocarbons (Snyder et al. 1973).

Body fat of herring gulls breeding on Lake Ontario in 1973 contained a number of aromatic hydrocarbons including several polynuclear aromatics. Naphthalene, 2-methyl-naphthalene, acetonaphthalene, and biphenyl were identified from their retention times (Fox et al. 1975). The sources of these aromatic hydrocarbons remain undetermined. Accumulation in the food chain from water or sediments through fish is probable, but these compounds, which presumably are of petroleum origin, may have been ingested at garbage dumps. Thus, it appears likely that aquatic birds living in oil-polluted environments may accumulate residues of the relatively more persistent compounds.

Biological Effects of Oil on Marine Birds

Aside from the reports on mortality and rehabilitation of oiled birds, the biological ef-

fects of oil on marine birds are little known. Important biological effects include both acute and chronic toxicity as well as adverse effects on physiology, reproduction, and behavior. Indirect effects involving the food web and changes in habitat and food supply are relatively unknown.

There is a distinct possibility that oil and other environmental contaminants such as the organochlorine compounds may act synergistically (Farrington 1977).

Circumstantial evidence suggests that oil pollution has seriously reduced populations of certain species of marine birds in some areas (Tuck and Livingston 1959; Tuck 1960; Hawkes 1961; Buck and Harrison 1967; Parslow 1967, 1970; Bourne 1968; Clark 1973).

An oil spill can have significant effects on populations of marine birds such as the alcids, which often are numerous among the birds that die in spills. Although alcids are long-lived and have few predators once they are at sea, they often do not breed until 3 or more years old, most lay only a single egg per clutch, not all adults breed every year, and they produce an average of only one chick per five breeding adults. These species require more than 50 years to double their population under optimal conditions. More than half a century would be required for a colony to recover its numbers (excluding immigration) if reduced by one-half as the result of a large oil spill (Clark 1969).

The potential effects of oil spills on aquatic birds and their feeding habitat on the Canadian west coast were assessed by Vermeer and Vermeer (1975). They concluded that the present shipping of oil plus the increased tanker traffic along the entire British Columbia coast that is expected to be in progress in 1977 will result in enough oil spillage to threaten the coastal populations of seabirds with destruction.

Concentrations of seabirds will be most vulnerable to spills (Vermeer and Vermeer 1975). Three major colonies along the coast of British Columbia are the Langara Region, the southeast coast of the Queen Charlotte Islands, and the Scott Islands. Alcids and storm petrels (*Oceanodroma* spp.) are the most numerous seabirds along the British Columbia coast. Alcids are among the birds most vulnerable to oil pollution, whereas storm petrels are less threatened by spills because they spend more time in the air and dive only occasionally. Waterfowl, especially diving ducks, will be vulnerable to spills during the winter as they concentrate in large numbers in estuaries and inlets along the British Columbia coast. The large wintering populations of ducks, geese, and grebes along the Fraser Delta foreshore and Boundary Bay will be vulnerable because of their nearness to tanker and shipping traffic. Approximately 1 million loons, shearwaters, phalaropes, ducks, gulls, and geese migrate north in the spring along west Vancouver Island. These migrants, because of their concentration in large numbers, may be very temporarily but critically vulnerable to oil pollution.

The birds most likely to be directly affected by spills are breeding populations of alcids and wintering diving ducks, whereas ducks, geese, and shorebirds, which feed in the intertidal zone, may be hardest hit indirectly through destruction of their feeding habitat (Vermeer and Vermeer 1975). Of the ducks threatened by destruction of their feeding habitat, sea ducks are most vulnerable because they rely most on the marine habitat for feeding purposes.

Feather-oiling

Large numbers of marine birds die each year as a result of oil spills. Estimates of mortality are based primarily on beach counts of oiled birds, but such estimates may be highly inaccurate because a significant percentage, perhaps 50-90%, of the dead birds never wash ashore (Clark and Kennedy 1968; Coulson et al. 1968; Tanis and Morzer Bruyns 1968; Hope-Jones et al. 1970).

An estimated 30,000 marine birds, of which about 97% were common murres and razorbills (*Alca torda*), died as a result of the Torrey Canyon disaster (Bourne et al. 1967). Earlier, in the winter of 1951-52, approximately 100,000 birds were lost to oil pollution on the coasts of the British Isles (ZoBell 1962). At least 10,000 birds, including alcids, ducks, gulls, and kittiwakes, were killed by oil apparently derived from ballast pumped from tankers entering Cook Inlet, Alaska, during February and March 1970 (U.S. Department of the Interior 1970).

The population decline of murres (*Uria* spp.) along the coast of Newfoundland has been as-

sociated with oil pollution (Tuck 1960), although the effects probably are not related only to those caused by feather-oiling. Numerous other instances of mortality related to oil are documented in reviews on this subject (Clark and Kennedy 1968; Aldrich 1970; Vermeer and Vermeer 1974a).

The effects of oiled plumage on marine birds vary with the properties of the oil, degree of contamination, quantity absorbed, environmental conditions, and the original condition of the bird. Even a small patch of oil on the feathers may mean that without care the bird will die (Tuck 1960; Smith 1975), but in some instances it appears that birds are able to clean their own plumage (Phillips 1974; Smith 1975). Oiling of a bird's plumage increases metabolism and causes an increased loss of body heat to the surrounding cold water that can readily be fatal (Lincoln 1936; Hartung 1967; Boyle 1969; Greenwood 1970; McEwan and Koelink 1973).

Feather-oiling appears to be a more significant problem in cold-water areas than in areas where water is warmer. Warm water apparently causes the spilled liquid oil to form tarballs that are comparatively less hazardous to birds (Bourne and Bibby 1975).

Damage to feathers may result long after exposure and may be reflected by abnormal wear of the plumage (Bourne 1974).

After the Torrey Canyon grounding in March 1967, 7,849 oiled birds were captured for cleaning and rehabilitation. One month later, however, fewer than 6% were still alive (Clark and Kennedy 1971).

An estimated 3,180,000 liters of bunker C fuel oil were spilled in the massive 1971 oil spill that occurred near the entrance to San Francisco Bay. The California Department of Fish and Game estimated that 7,000 aquatic birds were exposed to the fuel oil, and more than 4,000 of these were taken into captivity for treatment and rehabilitation. Two weeks after the spill, 90% of the birds had died in spite of efforts to save them, and within 3 months mortality exceeded 96% (Orr 1971; Snyder et al. 1973). Grebes, murres, and loons apparently died more rapidly than the other species affected, and ducks appeared most hardy (Snyder et al. 1973).

Progress has since been made in the rehabilitation of oiled birds, and modified methods are being used (Hay 1975). In 1973, the International Bird Rescue Research Center treated 523 oiled birds of which 49% survived (Smith 1975).

Toxicology, Physiology, and Pathology

The great diversity of chemical compounds in oil increases the difficulty of determining its toxicological and physiological effects. In addition, oil dispersants used to clean up a spill area are also toxic and the toxicity of oil plus dispersant usually is greater than the toxicity of either alone (Clark and Kennedy 1968; Tarzwell 1970; Linden 1975). There also are important species differences in susceptibility (Swedmark et al. 1973).

The toxicity of some oils to ducks has been measured under different environmental conditions. Single doses of several industrial oils produced lipid pneumonia, gastrointestinal irritation, fatty livers, and adrenal cortical hyperplasia. Birds that received a cutting oil in combination with diesel oil exhibited acinar atrophy of the pancreas. Those that received diesel oil and a fuel oil developed toxic nephrosis. Cholinesterase activity was significantly inhibited by administration of the cutting oil and somewhat depressed by the diesel oil (Hartung and Hunt 1966).

Ducks that had been killed by oil pollution exhibited changes that were similar to those in the experimentally fed birds, suggesting that toxicity of oils is a major factor in mortality of exposed birds (Hartung and Hunt 1966). Toxicity apparently is reduced through aging of the oil because the more volatile compounds are also the more toxic (Clark and Kennedy 1968).

Birds that died after the San Francisco Bay oil spill in 1971 were examined for pathological changes that might have resulted from exposure to oil. Intoxication from oil ingestion appeared to be an important factor contributing to the high mortality, although the evidence was circumstantial. Birds that died in the period of high mortality had ingested oil and exhibited dehydration, ulceration of the intestinal mucosa, enteritis, hepatic fatty changes, and renal tubular nephrosis (Snyder et al. 1973). Similar pathological changes as well as adrenal lesions and pulmonary diseases have been observed in other oiled seabirds (Guillon 1967; Beer 1968).

Following the large 1974 oil spill in the

Straits of Magellan, a high percentage of South American tern (*Sterna hirundinacea*) chicks on an island in the spill area died (Smithsonian Institution 1974). Although the cause of mortality is unknown, it is possible that the small fish that the terns ate and fed to their young were contaminated with some fraction of the spilled crude oil in concentrations that did not harm the adults but were toxic to the young. It is possible, however, that the chicks died of starvation after the adults were killed or were unable to catch enough food for the young.

The rates at which water and sodium are transported across the intestinal mucosa increase when Pekin ducklings (*Anas platyrhynchos*) are transferred from fresh water to a diet containing hypertonic saline drinking water (Crocker et al. 1974). These rate increases seem to be essential for the successful adaptation of ducklings to saline drinking water. Ducklings given a single oral dose of a crude oil (0.2 ml) at the start of maintenance on saline drinking water did not develop the characteristic rate increases. In addition, high mucosal transfer rates that had been developed in ducklings fed saline water for 3 days ceased 24 h after they received crude oil. Although commercial dispersant (5 ppm or 20 ppm) in fresh or saline drinking water had no effect on ducklings, the presence of dispersed crude oil (12.5-50.0 ppm) in the water prevented the development of high mucosal transfer rates in the ducklings given saline water.

A reduction of the mucosal transfer rates in seawater-adapted ducklings, through the action of ingested crude oil, may limit the amount of free water available to the body (Crocker et al. 1974). Although the high mortality among oil-contaminated seabirds may be due to a variety of pathological conditions, dehydration resulting from impairment of mucosal transfer mechanisms may be an important factor contributing to their death.

Crude oils from eight different geographical locations reduced the rates of sodium and water transfer across the intestinal mucosa of Pekin ducklings to different degrees (Crocker et al. 1975). Administration of Kuwait crude oil caused the greatest degree of inhibition, and North Slope, Alaska, crude oil caused the smallest.

Distillation fractions derived from two chemically different crude oils were administered to ducklings in volumes that corresponded to their relative abundance in the crude oil from which they were derived (Crocker et al. 1975). The greatest inhibitory effect on mucosal transfer was not associated with the same distillation fractions from each oil. A highly naphthenic crude oil from the San Joaquin Valley, California, showed the greatest inhibitory activity in the least abundant (2%), low boiling point (< 245 C) fraction. The most abundant (47%), highest boiling point (> 482 C) fraction showed the least inhibitory activity. In contrast, a highly paraffinic crude oil from Paradox Basin, Utah, showed the greatest inhibitory effect with the highest boiling point fraction and a minimal effect with the lowest boiling point fraction. The relative abundances of these two fractions in the Paradox Basin crude oil represented 27 and 28%.

Mucosal transfer inhibition by water-soluble extracts of San Joaquin Valley and Paradox Basin crude oils was roughly proportional to the inhibitory potency of the low boiling point fraction of the oil (Crocker et al. 1975). Weathered samples of these oils showed greater effects than corresponding samples of unweathered oils even though most of the low molecular weight material from both oils was either evaporated or solubilized in the underlying water during the 36-h weathering period.

Reproduction

During the nesting season, small amounts of oil on the plumage of birds can have very serious effects on reproduction. The oil compounds that are involved, however, are essentially unknown and no extensive tests have been reported.[2]

Oil washed ashore on a small island in West Germany where terns (chiefly *Thalasseus sandvicensis* and *Sterna hirundo*) and European oystercatchers (*Haematopus ostralegus*) were nesting. During copulation, many of the adult terns became dorsally smeared with oil from their mate's oiled feet, but no direct losses among adult terns were attributed to the oil. More than 70% of the young terns

[2]Reproductive effects have, however, been studied since this manuscript was written (see Szaro 1977).

were contaminated with oil and many of them were unable to fly. Some of the eggs laid along the high-tide mark failed to hatch after they became contaminated with oil (Rittinghaus 1956).

After ingestion of a relatively nontoxic lubricating oil (2 g/kg), one mallard (*Anas platyrhynchos*) and two Pekin ducks stopped laying for about 2 weeks. Very small quantities of oil coated on mallard eggs reduced their hatchability to 21%, compared with 80% for unoiled eggs. Experimentally oiled mallards continued to incubate their clutches, but their eggs failed to hatch (Hartung 1965).

In an experimental application to test the effects of 2,4-D and diesel fuel on eggs of ring-necked pheasants (*Phasianus colchicus*), there was no adverse effect by the 2,4-D on hatchability, but application of the diesel fuel reduced hatchability to zero (Kopischke 1972).

Behavior

Exposure to oil causes some obvious changes in behavior patterns of birds because they abandon all activities to attempt to clean the oil from their feathers by preening (Smith 1975). There may be other serious but less readily observed direct effects that influence the birds' ability to locate food, to migrate, or to perform other essential activities.

As discussed earlier, small amounts of oil in the water cause significant changes in behavior of certain marine organisms. Modified behavior among any of the numerous species of animals in the food webs may have serious indirect implications for the welfare of marine birds that depend upon them.

Organochlorines

By 1971, and perhaps earlier, it became unlikely that any bird dependent upon marine food webs anywhere in the world was free of contamination by the synthetic organochlorine compounds that have become ubiquitous pollutants in the global ecosystems (Sladen et al. 1966; Risebrough and Berger 1971; Bogan and Bourne 1972; Bourne and Bogan 1972; Bennington et al. 1975; Risebrough 1977; Walker 1977; White and Risebrough 1977). More information is available on the global distribution patterns of organochlorines than for other chemicals in marine birds. Several direct biological effects of organochlorines on marine birds are known. Other relevant information is available on the distribution of these pollutants in estuarine, freshwater, and terrestrial ecosystems, as well as their biological effects on other birds.

The most abundant synthetic organochlorine compound in tissues and eggs of marine birds is frequently *p,p'*-DDE, a derivative of *p,p'*-DDT, which is the principal component of the commercial insecticidal mixture (Risebrough et al. 1968; Jensen et al. 1969; Koeman et al. 1969). Other DDT compounds frequently present in marine birds are *p,p'*-DDD, *p,p'*-DDT, and *o,p'*-DDT (Bennington et al. 1975).

Polychlorinated biphenyls (PCB's), or chlorobiphenyls, consist of a mixture of compounds differing in chlorine content and the position of chlorine atoms on the parent biphenyl molecule. Pentachlorobiphenyls and hexachlorobiphenyls usually constitute the majority of the chlorobiphenyls present in marine birds, but trichlorobiphenyls and tetrachlorobiphenyls are occasionally present (Risebrough and de Lappe 1972; White and Risebrough 1977).

A number of other synthetic organochlorine compounds have been detected in marine birds, but almost always at levels substantially lower than those of the DDT and PCB compounds. Hexachlorobenzene (HCB), which has been found in tissues of great cormorants (*Phalacrocorax carbo*), sandwich terns (*Thalasseus sandvicensis*), and common eiders (*Somateria mollissima*) from coastal areas of the Netherlands (Koeman and van Genderen 1972; Koeman et al. 1972a), has been considered a potentially hazardous marine pollutant (National Academy of Sciences 1975b). The HCB is used as a fungicide but may enter the marine environment in significant quantities as a component of the tarry waste products from the manufacture of chlorinated hydrocarbons such as perchloroethylene and carbon tetrachloride that are frequently discharged at sea (Environmental Protection Agency 1973).

Chlorinated styrenes were identified by gas chromatography/mass spectrometry in tissues of common eiders, sandwich terns, and great cormorants from the Netherlands (Ten Noever de Brauw and Koeman 1972) and in

tissues of great blue herons (*Ardea herodias*) from Lake St. Clair, Michigan (Reichel et al. 1977); their source apparently remains unknown. Chlorinated naphthalenes also were identified in tissues of the great cormorant (Koeman et al. 1973).

Mirex was measured in eggs of herons and white ibis (*Eudocimus albus*) from estuarines of the U.S. Atlantic and Gulf coasts (Ohlendorf et al. 1974) and also in the blubber of a seal (*Phoca vitulina*) from the Netherlands (Ten Noever de Brauw et al. 1973). In addition to its use as an insecticide, mirex, under various trade names, is used as a flame retardant.

Dieldrin was found in eggs and tissues of several species of marine birds inhabiting coastal waters of Great Britain (Robinson et al. 1967) and of New Zealand, and also in pelagic species such as the sooty shearwater (*Puffinus griseus*) breeding in sub-Antarctic islands of New Zealand (Bennington et al. 1975). It is accumulated by ospreys (*Pandion haliaetus*) and bald eagles (*Haliaeetus leucocephalus*) feeding on coastal marine fish of the eastern United States (Mulhern et al. 1970; Belisle et al. 1972; Cromartie et al. 1975; Wiemeyer et al. 1975).

Endrin was detected in brown pelicans (*Pelecanus occidentalis*) from Florida (Schreiber and Risebrough 1972), the Gulf of California (Risebrough et al. 1968), and Louisiana (J. D. Newsom, personal communication). White pelicans (*Pelecanus erythrorhynchos*) from Louisiana also contained endrin (J. D. Newsom, personal communication).

Chlordane compounds, principally oxychlordane and *cis*-chlordane, were found in eggs of herons from the eastern U.S. estuaries (Ohlendorf et al. 1974) and in fish and common terns (*Sterna hirundo*) from Long Island Sound (R. W. Risebrough and P. Robinson, unpublished data).

Heptachlor epoxide, toxaphene, and the several isomers of hexachlorocyclohexane (benzene hexachloride or BHC) are occasionally found in estuarine environments. Heptachlor epoxide and BHC isomers have been reported in Antarctic birds breeding in the South Orkneys (Tatton and Ruzicka 1967) but their identification has not been confirmed (Risebrough 1977).

The occurrence, distribution, and effects of organochlorines on wildlife, principally terrestrial, freshwater, and estuarine species, have been summarized in other recent reviews (Prestt and Ratcliffe 1972; L. F. Stickel 1972, 1973; Blus et al. 1977b; W. H. Stickel 1975; Ketchum et al. 1975; L. F. Stickel and F. E. Hester, unpublished manuscript). Earlier studies of the transport of PCB's to the marine environment also have been reviewed (Nisbet and Sarofim 1972; Panel on Hazardous Trace Substances 1972). More recently the environmental effects of PCB's (Peakall 1975), their chemical properties (Hutzinger et al. 1974), and the transfer of organochlorine compounds to the marine environment and their incorporation into marine food webs have been reviewed (Risebrough et al. 1976a).

Although levels of organochlorine compounds other than those of the DDT and PCB groups may occasionally be present at levels deleterious to birds in estuaries, levels in the offshore marine environment are usually well below those considered harmful to marine birds. In the present review, principal emphasis will therefore be placed on the DDT and PCB compounds.

Exposure of Marine Birds to Organochlorines

Organochlorine residue data are available from coastal regions, but there have been relatively few studies of chlorinated hydrocarbon contamination of marine birds in areas that are far from known pollution sources.

All eggs of the Adelie penguin (*Pygoscelis adeliae*) from widely separated localities in the Antarctic (Risebrough and Carmignani 1972; Risebrough 1977), eggs and tissues of birds from the Aleutians (White and Risebrough 1977) and from sub-Antarctic areas of New Zealand (Bennington et al. 1975), and tissues of birds from the eastern North Atlantic (Bourne and Bogan 1972; Bogan and Bourne 1972) contained residues of DDT compounds. All samples also contained detectable levels of chlorobiphenyl compounds, frequently at levels higher than the total concentration of the DDT group. The DDT and chlorobiphenyl compounds also were detected in all samples obtained from remote terrestrial and freshwater Arctic ecosystems (Risebrough and Berger 1971; Walker 1977).

Although the data are few and sample sizes

Table 1. *Mean PCB and DDE residues (ppm lipid weight) in cormorants* (Phalacrocorax *spp.*)

Locality, date	Species	N	Tissue	Percent lipid	PCB's	DDE	PCB/DDE
Amchitka, 1971[a]	Red-faced cormorant (*P. urile*)	1	Yolk	20.0	19.0	3.8	5.0
Amchitka, 1974[a]	Red-faced cormorant	1	Pectoral muscle	—	21.0	3.5	6.0
Agattu, 1974[a]	Red-faced cormorant	1	Pectoral muscle	3.8	14.0	2.4	6.0
Amchitka, 1974[a]	Pelagic cormorant (*P. pelagicus*)	1	Pectoral muscle	4.0	8.0	0.8	10.0
Auckland Islands, 1972[b]	Auckland Island shag	4	Egg lipid	100.0	0.3	0.9	0.3
Iceland, 1973[c]	Shag (*P. aristotelis*)	10	Egg	5.0	23.0	3.8	6.0
	Great cormorant	13	Egg	4.8	10.0	3.0	3.0
Peru, 1969[d]	Guanay (*P. bougainvillei*)	4	Egg lipid	100.0	15.0	12.2	1.2
Southern California, 1969[e]	Double-crested cormorant	7	Egg lipid	100.0	87.0	754.0	0.1
Greenland, 1972[f]	Great cormorant	3	Body fat	—	23.0	9.8	2.3

[a]White and Risebrough 1977.
[b]Bennington et al. 1975.
[c]J. A. Sproul et al., unpublished manuscript.
[d]R. W. Risebrough et al., unpublished manuscript.
[e]Gress et al. 1973.
[f]Braestrup et al. 1974.

are frequently small, a general picture of global marine contamination by DDT and PCB compounds can be presented. Some of the available data on cormorants have been summarized (Table 1). With the exception of the residue values reported for the double-crested cormorant (*Phalacrocorax auritus*) in southern California, where the birds were exposed to industrial contamination from the Los Angeles area (Gress et al. 1973), the samples were obtained from areas reasonably remote from point sources of contamination. The DDE residues in the Auckland Island shags (*Phalacrocorax carunculatus*) were somewhat lower than in cormorants from Amchitka and Agattu at the equivalent latitude in the northern hemisphere. However, PCB values in the southern hemisphere birds were lower by 1-2 orders of magnitude.

Other data from biocenotic equivalents in the two areas support the conclusion that DDE levels are slightly lower in the southern than in the northern hemisphere but that PCB values are lower by 1-2 orders of magnitude (Bennington et al. 1975; White and Risebrough 1977). The DDE levels in an egg of a New Zealand falcon (*Falco novaeseelandiae*) were equivalent to those in eggs of peregrines from Amchitka, but PCB levels were an order of magnitude lower. Comparable differences were found between auklets (*Aethia pusilla* and *Cyclorrhynchus psittacula*) and the tufted puffin (*Lunda cirrhata*) of the Aleutians and the diving petrel (*Pelecanoides urinatrix*) from the Snares Islands of southern New Zealand.

The DDE levels in eggs of the guanay were somewhat higher than those from New Zealand or the Aleutians, suggesting local sources of DDT contamination in Peru (R. W. Risebrough et al., unpublished manuscript).

A comparison of the cormorant samples from the Aleutians and the eggs of two cormorants (*Phalacrocorax carbo* and *P. aristotelis*) breeding in Iceland suggests that levels of DDE and PCB contamination in the two oceanic areas are similar. In five species of fish obtained from Amchitka in 1974, DDE residues ranged from 1 to 5 ppb; PCB resi-

dues ranged from 8 to 20 ppb (White and Risebrough 1977). Residues of DDE in seven species of fish obtained from the coastal waters of Iceland in 1973 ranged from 1 to 9 ppb; PCB levels ranged from 8 to 20 ppb (J. A. Sproul et al., unpublished manuscript). On a lipid basis, PCB residues expressed as tri-, tetra-, or penta-chlorobiphenyls ranged from 0.3 to 2 ppm in the Amchitka fish and from 0.2 to 3 ppm in the Icelandic fish. Body fat of great cormorants from Greenland (Braestrup et al. 1974) contained comparable PCB levels and somewhat higher DDE levels than great cormorants from Iceland.

A comparison of DDE and PCB residue levels in black-legged kittiwakes (*Rissa tridactyla*), fulmars (*Fulmarus glacialis*), and thick-billed murres (*Uria lomvia*) from Amchitka and Iceland suggests somewhat higher levels in the Icelandic birds, although residues are of the same order of magnitude and with comparable ratios (J. A. Sproul et al., unpublished manuscript; White and Risebrough 1977). The differences may reflect a higher level of contamination in those areas of the ocean where the Atlantic birds spend the winter months.

Earlier data (Risebrough et al. 1968) suggested that DDT compounds were more abundant than PCB's in Pacific waters. However, many of the samples were from coastal California waters where DDT contamination was particularly severe.

Residue levels and PCB:DDE ratios in the breast muscles of Icelandic birds obtained in 1973 were comparable to those in birds obtained earlier from areas north of Britain, indicating that no decline in residue concentrations in birds had occurred over that short interval (Bourne and Bogan 1972; J. A. Sproul et al., unpublished manuscript).

From the Pacific, the visceral fat of 7 black-footed albatrosses (*Diomedea nigripes*) and 22 Laysan albatrosses (*D. immutablis*) from Midway Island contained mean DDE levels of 22 and 8 ppm, and mean PCB levels of 14 and 2 ppm (Fisher 1973). These species are restricted to the North Pacific, usually about 20° N and feed primarily on squid. In Hawaii, DDE concentrations in four eggs of the dark-rumped petrel (*Pterodroma phaeopygia*) ranged from 0.07 to 1.14 ppm (0.6-11.5 ppm, lipid weight) (King and Lincer 1973). PCB values were not reported.

From the tropical Atlantic, DDE and PCB levels in the breast muscle of 28 adult sooty terns (*Sterna fuscata*), breeding on the Dry Tortugas, were 2.5 and 7.8 ppm (lipid weight); mean percentage of lipid was 2.6% (P. G. Connors et al., unpublished manuscript).

In these areas of the Atlantic and Pacific, comparatively remote from sources of contamination, PCB residue concentrations generally exceeded those of the DDT compounds. In the New Zealand (including the sub-Antarctic islands) samples, however, DDT residues were frequently present at higher concentrations than the sum of PCB's (Bennington et al. 1975).

In the Antarctic, few eggs of the Adelie penguin obtained in 1970 or earlier from widely separated localities contained PCB's at detectable levels (Risebrough et al. 1976a). Maximum amounts of PCB's in eggs of the Adelie penguin obtained from Cape Crozier in October 1967 were less than one-eighteenth of the concentration of DDT compounds (Risebrough et al. 1968). Subsequent analysis of some of these eggs revealed the presence of PCB. PCB's were detected also in eggs of the Adelie penguin, chinstrap penguin (*Pygoscelis antarctica*), and the gentoo penguin (*P. papua*) obtained in the Antarctic Peninsula in 1975, although at concentrations less than those of the DDT compounds (Risebrough et al. 1976b). The preponderance of DDT compounds in the Antarctic, the most remote area receiving chlorinated hydrocarbons from atmospheric or oceanic transport, apparently reflects the relative use of these two groups of compounds in the southern hemisphere.

In coastal areas local conditions usually determine the contamination patterns. For example, liquid chemical wastes discharged by insecticide manufacturing plants in California and the Netherlands subsequently entered the sea and caused significant organochlorine contamination of coastal birds. High DDT concentrations were found in northern anchovies (*Engraulis mordax*) from Los Angeles Harbor in 1965 (Risebrough et al. 1967). Subsequent investigations documented exceptionally high levels of DDT compounds in the coastal birds, including the brown pelicans (Risebrough 1972; Anderson et al. 1975) and double-crested cormorants (Gress et al. 1973). When the company began to dispose of its liquid wastes in a sanitary landfill in 1970, input of DDT compounds into the sea began

to decline (Carry and Redner 1970; Redner and Payne 1971; D. R. Young et al., unpublished manuscript); residues in fish and in the brown pelicans also began to decline (Anderson et al. 1975).

In 1964, sandwich terns and spoonbills (*Platalea leucorodia*) were found dying on the island of Texel in the Dutch Wadden Sea. The birds were in tremors and convulsions, signs comparable to those found in other birds poisoned by organochlorine insecticides (Koeman and van Genderen 1965, 1966). Studies of the distribution of chlorinated hydrocarbons in birds, fish, and mussels (*Mytilus edulis*) from localities along the Dutch and West German coasts and in the eggs of seabirds of Great Britain indicated a point source of contamination by dieldrin, endrin, and telodrin. Telodrin, an insecticide not used in Europe at that time, was being manufactured with dieldrin and endrin in a factory near the mouth of the Rhine River. When it was discovered that these residues were coming from the insecticide plant, measures were taken to eliminate discharge; residue levels in the local seabirds began to decline and the sharp decrease in population numbers was halted (Koeman et al. 1968).

In addition to these two incidences, coastal contamination from local but diffuse sources has resulted in high levels of organochlorines in birds in Japan, North America, and Europe. Levels of PCB in Japanese birds, including several species of gulls, were comparable to those in industrial areas of North America and Europe (Fujiwara 1974). The PCB residues in breast muscle of eight little egrets (*Egretta garzetta*) that were found dead or dying in Tokyo Bay ranged from 0.3 to 180 ppm (22-1,600 ppm, lipid basis) with a geometric mean of 9 ppm. Residues of PCB in the breast muscle of eight black-tailed gulls (*Larus crassirostris*) ranged from 3-39 ppm, with a geometric mean of 13 ppm (Doguchi 1973).

In western North America comparatively high levels of DDT and PCB contamination were found in common murres (Gress et al. 1971) and the ashy storm petrels (*Oceanodroma homochroa*) (Coulter and Risebrough 1973) breeding on the Farallon Islands and in great egrets (*Casmerodius albus*) and great blue herons breeding at a coastal site (Faber et al. 1972) near local sources of pollution.

Most eggs of marine birds from the Strait of Georgia contained more PCB's and DDE and had a higher PCB:DDE ratio than did eggs from the west coast of Vancouver Island and from the Queen Charlotte Islands (Table 2). This comparison within a relatively small region (i.e., the Pacific Coast of British Columbia) further illustrates the principle that eggs from birds nesting farther at sea are likely to contain lower levels of organochlorines than those nesting nearer the mainland. Average PCB levels in these samples almost always exceeded those of DDE.

Fish, crabs, and shellfish were collected from the lower Fraser River, its estuary, and selected areas of Georgia Strait (Albright et al. 1975). Generally, PCB's were present at higher levels than DDE, and greatest concentrations of these compounds occurred in biota from waters adjacent to the city of Vancouver. With one exception, animals from Georgia Strait and those away from the immediate influence of Fraser River water contained no detectable levels of chlorinated hydrocarbons.

High levels of DDE and PCB in double-crested cormorants from the Bay of Fundy (Zitko and Choi 1972; Zitko et al. 1972) most likely have resulted from past DDT use in New Brunswick and from diffuse sources of PCB's along the eastern North American coastline. Similarly, contamination levels in ospreys (Wiemeyer et al. 1975; Spitzer et al. 1977) in coastal Connecticut, Massachusetts, New York, and New Jersey most likely were derived from local sources of contamination.

Bald eagles found sick or dead in the United States during 1966-72 were analyzed for organochlorines (Mulhern et al. 1970; Belisle et al. 1972; Cromartie et al. 1975); DDE, DDD, dieldrin, and PCB's were detected in most of the 145 eagle carcasses. Eighteen of the eagles contained possibly lethal levels (greater than 4 ppm) of dieldrin. Since 1964 when data were first collected, 8 of the 17 eagles obtained from Maryland, Virginia, South Carolina, and Florida possibly died from dieldrin poisoning. All four specimens from Maryland and Virginia were from the Chesapeake Bay Tidewater area.

In December 1973, eight ruddy ducks (*Oxyura jamaicensis*) killed in an oil spill on the Delaware River (White and Kaiser 1976), contained DDE (1.1-4.5 ppm) and PCB's (2.8-10 ppm). Levels of DDT and DDD were below

Table 2. *Mean PCB and DDE residues (ppm lipid weight) in seabird eggs from the Strait of Georgia, the west coast of Vancouver Island, and the Queen Charlotte Islands in British Columbia, 1970* (K. Vermeer, unpublished data).

Locality	Species	N	Percent lipid	PCB's	DDE	PCB/DDE
Strait of Georgia						
Mandarte Island	Double-crested cormorant	3	6.9	207.0	59.0	3.5
	Pelagic cormorant	10	5.3	50.0	15.0	3.3
	Glaucous-winged gull (*Larus glaucescens*)	10	6.0	41.5	12.5	3.3
Mittlenatch Island	Pelagic cormorant	10	4.4	122.0	12.0	10.2
	Pigeon guillemot (*Cepphus columba*)	10	10.5	34.0	6.0	5.7
	Glaucous-winged gull	10	8.0	19.0	6.0	3.2
Vancouver Island						
Cleland Island	Leach's petrel (*Oceanodroma leucorhoa*)	10	12.7	8.5	17.0	0.5
	Pigeon guillemot	1	10.8	24.0	12.0	2.0
	Tufted puffin	1	10.0	6.5	4.0	1.6
	Glaucous-winged gull	10	8.6	30.0	18.5	1.6
Queen Charlotte Islands						
Skedans Island	Fork-tailed petrel (*O. furcata*)	2	29.6	51.0	14.0	3.6
	Pigeon guillemot	2	11.7	3.6	1.4	2.6
	Glaucous-winged gull	10	8.3	6.0	4.0	1.5
Lucy Island	Rhinoceros auklet (*Cerorhinca monocerata*)	10	15.0	13.0	18.0	0.7
	Glaucous-winged gull	10	9.7	6.0	3.0	2.0
Northwest Rocks	Glaucous-winged gull	3	8.7	4.2	2.6	1.6

0.34 ppm in all but one sample. Dieldrin and HCB were present in seven samples, but neither exceeded 0.36 ppm.

In a survey of organochlorine residues in 21 aquatic bird species at 31 locations in Alberta, Saskatchewan, and Manitoba, DDE and dieldrin levels were higher in eggs of larids and fish-eating birds than in those of geese and ducks, presumably reflecting different trophic levels between those two groups of birds (Vermeer and Reynolds 1970).

On the Niagara Peninsula, an area of Ontario that is intensively developed for agriculture and heavy industry and has a large urban population, eggs were collected in 1972 from 20 species of birds having a variety of feeding habits (Frank et al. 1975). Representative species were obtained from both the terrestrial and aquatic food chains. Highest total DDT residues were in the eggs of aquatic carnivores, including common tern (22.4 ppm), herring gull (10.4 ppm), black-crowned night heron (*Nycticorax nycticorax*; 7.8 ppm), and black tern (*Chlidonias niger*; 7.6 ppm). Herbivores and insectivores contained lower total DDT residues regardless of the environment in which they fed. The highest mean residues of PCB's also were in carnivores in the aquatic food chain, including herring gulls (74 ppm), common terns (42 ppm), and black-crowned night herons (27 ppm).

Eggs of anhingas (*Anhinga anhinga*), herons, and ibises were collected during the 1972 nesting season at coastal and inland localities from Florida to New Jersey (Ohlendorf et al. 1974). Measurable residues of DDE

occurred in all 209 eggs. The highest mean value (4.0 ppm) was found in great egrets from New Jersey. Among the coastal localities, levels of DDE as well as total DDT progressively declined toward the south. The PCB's occurred second most frequently and also reached their highest mean level (4.2 ppm) in the great egret eggs from New Jersey. Other pollutants occurred less frequently and at lower levels.

In Great Britain, where the presence of organochlorine pollutants in seabird eggs was first demonstrated (Moore and Tatton 1965), organochlorine residues in seabird eggs from a number of colonies have been monitored. Populations of common puffin (*Fratercula arctica*) in Great Britain declined (Flegg 1971, 1972), but those birds analyzed have not shown excessively high contamination levels. Birds from Saint Kilda contained 7.6 ppm of PCB (61 ppm in fat), but seven other puffins contained lower concentrations. Five eggs obtained from Saint Kilda in 1969 contained lower residues of PCB's, DDE, and dieldrin than did eggs of either the common murre or the razorbill (Parslow et al. 1972). Eggs of the murre from Lundy, Skomer, and Berry Head contained lower PCB levels than did eggs of the kittiwakes from the same location, but DDE levels were lower in the kittiwakes (Parslow 1973). In some localities on the British coast, eggs of murres contained levels of PCB's that were as high as those reported from California and the Baltic, but DDE levels were lowest in Britain.

Biological Effects of Organochlorines on Marine Birds

Although there is a considerable amount of information on residue concentration and reproductive effects of organochlorines in marine birds, there is relatively little information on toxicology, physiology, and pathology in these species. Therefore, it is particularly relevant to consider also such effects in the more frequently studied terrestrial species.

Toxicology, Physiology, and Pathology

Evidence is substantial that PCB's may have contributed to the mortality of contaminated birds. Great cormorants found dead in the Netherlands may have died of PCB poisoning (Koeman et al. 1973). Residues in the brain and liver were equivalent to those in birds poisoned through feeding of the PCB preparation Clophen A60. Chlorinated dibenzofurans, however, were present in the commercial PCB mixture (Vos et al. 1970) and may have contributed to the mortality of the experimental birds. Therefore, the residue levels in tissues may not be equivalent in the toxicological sense.

The occasional "wrecks" of seabirds, particularly of common murres, are usually associated with storms. In 1970 more than 100,000 murres died in Bristol Bay, Alaska, following stormy weather (Bailey and Davenport 1972). There had been no oil spills in the area. The birds were emaciated and apparently had starved as a result of an inability to find food during the prolonged storm, but they were not analyzed for organochlorines.

In Great Britain, PCB concentrations in the livers of gannets (*Morus bassanus*) that died during large-scale mortality incidents in 1972 ranged from 3,300-9,600 ppm, lipid weight; DDE concentrations ranged from 260-520 ppm, lipid weight (Parslow et al. 1973). Organochlorine concentrations of this magnitude might contribute to the death of marine birds either through direct poisoning following mobilization of fat or through more subtle sublethal effects on the birds at a time of environmental stress.

Because concentrations of chemicals in the body are greatly affected by weight gains and losses, it is sometimes more useful to compare total body loads. Estimated body contents of PCB's and DDE in five murres found dead during a 1969 wreck in the Irish Sea were 2,700 μg (range 800-8,900) and 673 μg (314-1,535) (Holdgate 1971). Five birds that were shot in the same general area had 3,500 μg (800-7,200) of PCB's and 1,484 μg (468-3,211) of DDE. However, eight other murres that died in the wreck had an average estimated body burden of 4,660 μg of PCB, twice as much as in nine other apparently healthy birds that were collected (Parslow and Jefferies 1973). Depletion of body fat during times of hunger could be expected to mobilize chlorinated hydrocarbons, providing additional stress when the birds are poorly equipped to cope with it. The overall contribution of chlorinated hydrocarbons, particularly

PCB's, to such mortality remains to be determined.

A glaucous gull (*Larus hyperboreus*) found in convulsions on Bear Island in the Arctic contained 311 ppm of PCB's and 67 ppm DDE in the liver (Bogan and Bourne 1972); its weakened condition and abnormal coordination were attributed to these high levels. The glaucous gulls in this colony were feeding on the eggs of other seabirds.

Necropsy findings and the high level of DDD (200 ppm) in the brain of a common loon (*Gavia immer*) found in a soybean field in Madison County, Mississippi, indicate that the bird died of DDD poisoning (Prouty et al. 1975).

Experiments have been conducted with captive birds to determine which tissue might contain chemical residues that are diagnostic of organochlorine poisoning (L. F. Stickel et al. 1966; W. H. Stickel et al. 1969, 1970, 1973; Stickel and Stickel 1970). There is little doubt that many closely related compounds have a lethal additive effect in the nervous system (Ludke 1976; J. L. Ludke and W. H. Stickel, personal communication). Chemical residues in the brain, in association with pathological conditions of the body, may reveal that the compounds caused death. Lethal ranges have been established for DDT, DDD, DDE, dieldrin, and mirex. Suggestions also have been made for weighing and summing brain residues of DDT, DDD, and DDE for interpretation of field specimens.

Recent studies of the induction of hepatic enzymes by PCB's have been reviewed (Peakall 1975). Levels of PCB in many seabirds may be assumed to be sufficient to increase the activity of various mixed function oxidase enzymes. Elevated activity levels of these enzymes also enhance steroid metabolism and degrade nonpolar compounds of foreign origin. The biological consequences of increased steroid metabolism are unknown but birds may compensate for the higher level of steroid metabolism by increasing levels of synthesis.

Teratogenic effects observed in experimental feeding studies with PCB's have included malformations of the eye, legs, and beaks (Carlson and Duby 1973; Tumasonis et al. 1973; Cecil et al. 1974). Such abnormalities may have been caused by contaminant dibenzofurans in the PCB mixtures (Vos et al. 1970; Bowes et al. 1975). Similar abnormalities have been found in common terns breeding in Long Island Sound (Hays and Risebrough 1972) but the cause remains unknown; a link with PCB or chlorinated dibenzofurans has not yet been proven.

Diets containing 10 and 30 ppm (dry weight) DDE were fed to black ducks (*Anas rubripes*), and diets containing 1, 5, and 10 ppm (dry weight) DDE were fed to mallards (Longcore et al. 1971a). Among the results were the following changes in black duck eggshell composition: (1) significant increase in the percentage of magnesium, (2) significant decreases in barium and strontium, (3) increases (which approached significance) in average percentage of eggshell sodium and copper, (4) a decrease in shell calcium that approached significance, (5) patterns of mineral correlations that in some instances were distinct to dosage groups, and (6) inverse correlations in the control group between eggshell thickness, magnesium, and sodium.

Changes in mallard eggshells were: (1) significant increase in percentage of magnesium at 5 and 10 ppm DDE, (2) significant decrease in aluminum at 5 and 10 ppm DDE, (3) a significant decrease of calcium in the 10 ppm DDE group, and (4) an increase in average percentage of sodium in eggshells from DDE-dosed ducks that approached significance.

Blood samples were taken for 2 successive years from canvasback ducks (*Aythya valisineria*) trapped in the Chesapeake Bay (Dieter et al. 1976). The first winter (1972-73), five plasma enzymes known to respond to organochlorine poisoning were examined. Alterations in enzyme activity indicated tissue damage (specifically in membrane permeability) at the cellular level. Abnormal enzyme elevations suggested that 20% of the population sampled (23 of 115 ducks) might contain elevated levels of organochlorine contaminants, but no residue analyses were performed. The second winter (1973-74), two of the same enzymes, aspartate aminotransferase and lactate dehydrogenase, were assayed in 95 blood samples. The PCB concentrations in representative blood samples were significantly ($P < 0.05$) correlated with plasma aspartate aminotransferase activity.

Male coturnix quail (*Coturnix coturnix*) were fed diets containing graded levels of DDE, PCB (Aroclor 1254), malathion, and

mercuric chloride (Dieter 1974). At 12 weeks, increases in each of the activities of five plasma enzymes (creatine kinase, aspartate aminotransferase, cholinesterase, fructose-diphosphate aldolase, and lactate dehydrogenase) of birds were proportional to the log dose of the respective agents. In addition, the pattern of enzyme responses in the experimental groups had changed, and was illustrative of the specific type of substance that had been fed. The data suggest that qualitative and quantitative identification of environmental contaminants in birds, and perhaps a variety of wild animals, may be possible by utilization of multiple plasma enzyme assays. Residue analyses after 12 weeks of feeding showed that DDE accumulated in carcasses and livers at concentrations up to fourfold higher than those in the diets. In contrast, residues of Aroclor 1254 attained in carcasses were identical to, and in livers one-half of, the concentration in the feed.

Wild-trapped starlings (*Sturnus vulgaris*) were fed concentrations of DDE or Aroclor 1254 (5, 25, and 100 ppm, dry weight) that were found to be sublethal when fed to pen-reared coturnix quail for 12 weeks (Dieter 1975). Although the experimental design had been to compare plasma enzyme responses at 3, 7, and 12 weeks, reliable measurements could only be made through 7 weeks of the experiment because of unexpected mortality. Variations in enzyme response were greater in wild than in pen-reared birds, but not enough to mask the toxicant-induced changes in enzyme activity. Cholinesterase, lactate dehydrogenase, creatine kinase, and aspartate aminotransferase activities increased in those fed the organochlorine compounds. Evaluation of enzymatic profiles appears to be a potentially valuable technique to monitor the presence of toxicants in wild populations, especially if used to complement standard chemical residue analyses. After feeding for 7 weeks, liver residues of either organochlorine compound were about threefold higher than the concentrations fed daily. However, 4 times as much DDE as Aroclor 1254 had accumulated in the carcasses.

Dietary DDE at levels from 10 to 1,000 ppm (dry weight) inhibited nasal gland secretion in mallards maintained in fresh water (Friend et al. 1973). However, in subsequent studies on the effects of dietary DDE (10-250 ppm, dry weight) on osmoregulation and nasal gland function in mallards, Pekin ducks, black guillemots (*Cepphus grylle*), and common puffins, DDE had minimal effects on plasma electrolyte levels and total nasal gland Na,K-ATPase activities in each of these species (Miller et al. 1976). Liver DDE levels in experimental ducks and guillemots were comparable with those reported for seabirds found dead after kills; levels in starved puffins were much higher. Therefore, DDE at environmental levels may not affect osmoregulation of nasal gland Na,K-ATPase in ducks or in these two species of marine birds.

Coturnix quail were fed 1 ppm (dry weight) dieldrin, 2 ppm DDE, or the two chemicals together (Ludke 1974). When fed alone, both dieldrin and DDE reached their highest concentrations in the birds' livers after 28 days on treatment, followed by a slight decrease after 56 days. In whole-body samples (carcass minus liver), dieldrin residues increased steadily throughout the treatment period. Dieldrin residues in the birds exposed to dieldrin alone were always similar to residues in birds that were exposed to dieldrin in combination with DDE. In birds fed DDE, either alone or in combination with dieldrin, DDE residues in the carcass increased similarly for 28 days. After 56 days, DDE residues were significantly greater in the birds fed the dieldrin and DDE mixture. The continued increase of DDE residues when both DDE and dieldrin were fed suggests an interaction in which dieldrin promotes an increased uptake or retention of DDE.

No weight loss or mortality occurred among bobwhite (*Colinus virginianus*) fed a control diet or those fed chlordane (10 ppm, dry weight) alone. However, birds that were fed endrin (10 ppm, dry weight) or a combination of chlordane and endrin lost weight and died within a few days (Ludke 1976). Moribund individuals had lost considerable body weight and contained much less body fat than did individuals that were not exhibiting signs of intoxication when sacrificed. Birds that died from intoxication averaged weight losses of 32.2% (endrin-treated) and 31.4% (chlordane + endrin-treated) when compared with the control group. Individuals that survived exposure had significantly lower brain residues than those that died. Residues of endrin were significantly lower (by 38%) in brains of

birds that died from endrin plus chlordane than in those dying from endrin alone. These data indicate that closely related toxicants may have an accumulative effect at the site of action.

Two of 14 male American kestrels (*Falco sparverius*) died after 14 and 16 months on a diet containing 2.8 ppm DDE (Porter and Wiemeyer 1972). The brains of the two birds contained DDE residues of 213 and 301 ppm compared with an average of 14.9 ppm (range, 4.5-26.6 ppm) for 11 of the adult males that were sacrificed after 12 to 16 months on dosage. Each of the two birds that died had lost about one-third of its weight since treatment began and necropsy revealed typical characteristics (reduced pectoral muscle and badly depleted fat reserves) of organochlorine poisoning.

Endrin was consistently the most toxic of 89 pesticidal chemicals that were tested for their lethal dietary toxicity to young bobwhites, coturnix quail, ring-necked pheasants, and mallards (Heath et al. 1972a). Aldrin and dieldrin were among the six most toxic chemicals of those tested on all species, and toxaphene was the only other organochlorine that was particularly toxic to mallards. Major species differences in vulnerability to various chemicals such as were demonstrated in this study must be considered whenever toxicity of particular chemicals to avian species is unknown. Further testing made this point increasingly clear (Hill et al. 1975). Among the more toxic organochlorine compounds, nearly all are alicyclic hydrocarbons. Of these chemicals tested, most of the aromatic chlorinated hydrocarbons are among the less toxic.

Toxicities of six PCB compounds (Aroclor 1232, 1242, 1248, 1254, 1260, and 1262) to penned mallards, pheasants, bobwhite, and coturnix quail were generally less than that of DDT (Heath et al. 1972b). Aroclor toxicity was positively correlated with chlorine percentage (last two digits of Aroclor number) for the 2-week-old birds that were fed treated diets for 5 days. The joint toxicity of Aroclor 1254 and DDE on coturnix was additive, not synergistic. When 18 chemicals (including 8 organochlorines) were fed in 13 pairs to coturnix quail and ring-necked pheasant, the effects of the organochlorines also were additive rather than synergistic (Kreitzer and Spann 1973).

To learn if the percentage of chlorine in a mixture of PCB's alone determines toxicity, Hill et al. (1974) fed coturnix quail diets containing Aroclor 1248, 1254, or 1260 at levels that added equal amounts of chlorine to the feed. Sublethal concentrations produced no detectable effects. Lethal concentrations with equal chlorine showed Aroclor 1248 to be the least toxic of the three compounds at the highest chlorine concentrations. At lower concentrations, Aroclor 1254 was the most toxic Aroclor. Although chlorine percentage of a PCB is positively correlated with its avian toxicity, PCB toxicity is apparently not simply a function of chlorination. Toxicity also is related to the positions the chlorine atoms occupy on the benzene rings. Toxicity of hexachlorobiphenyl mixtures to bird embryos has been shown to be correlated with their dibenzofuran content (Vos and Koeman 1970; Vos et al. 1970).

Experiments with coturnix quail were used to simulate the stresses on wild birds of breeding condition and of weight loss due to migration (Gish and Chura 1970). Light conditions in the laboratory were manipulated to stimulate reproductive development in one group of birds and suppress development in another group. Within each of these groups, some birds were partially starved before dosage and some were fully fed. Birds were then fed dietary levels of 0, 700, 922, 1,214, or 1,600 ppm (dry weight) of DDT for a period of 20 days or until death. Birds partially starved before dosage were more susceptible to DDT intoxication than nonstarved ones. Similarly, males died earlier than females, and the lighter birds died earlier than the heavier ones. The heavier birds of each sex not only survived longer than lighter individuals receiving the same treatments, but they also lost a greater proportion of their weight before death. During the early portion of the dosage period, females in breeding condition were less sensitive to DDT than were nonbreeding females and males. After 10 days on dosage, however, the cumulative mortality of females in breeding condition rapidly approached that of males and of females not in breeding condition.

Reproduction

Field and experimental evidence indicates that declines in eggshell thickness observed in

certain species in North America and Great Britain since the mid-1940's have been largely caused by residues of *p,p'*-DDE or other compounds or metabolites of the DDT group (Cooke 1973). At moderate or high levels of DDE, shell thinning is severe and eggs may break during incubation. High DDE levels have been recorded in California; species affected there have included brown pelicans (Risebrough et al. 1971), double-crested cormorants (Gress et al. 1973), great egrets, and great blue herons (Faber et al. 1972). As indicated previously, much of the DDE probably originated from an insecticide manufacturing plant in southern California. DDE levels associated with the shell thinning of eggs of the common murres (Gress et al. 1971) and ashy storm petrels (Coulter and Risebrough 1973) on the Farallon Islands in central California may also have originated in part from this particular source.

Eggshell thinning has occurred in several other species that occur in freshwater or estuarine habitats or that nest on coastal islands. In 1967, shell thickness in herring gull eggs from five States decreased with increases in chlorinated hydrocarbon residues (Hickey and Anderson 1968). Comparison of eggshells taken before 1946 with those taken since then reveals that several species including the peregrine falcon, brown pelican, double-crested cormorant, black-crowned night heron, bald eagle, and osprey have sustained shell-thickness and shell-weight decreases of 20% or more, at least for brief periods (Anderson and Hickey 1972). In some of these, regional population declines are known. However, in seabird species that depend upon marine food chains in Iceland, there was no evidence of shell thinning in 1973 (J. A. Sproul et al., unpublished manuscript).

Shell thickness was significantly and inversely correlated with the concentration of DDE in 40 great blue heron eggs from Alberta (Vermeer and Reynolds 1970; Vermeer and Risebrough 1972).

In the Upper Great Lakes States, 9 of 13 species of fish-eating birds were found in 1969-70 to have sustained statistically significant decreases in eggshell thickness since 1946 (Faber and Hickey 1973). Maximum changes in a thickness index occurred in great blue herons (-25%), red-breasted mergansers (*Mergus serrator;* -15%), and double-crested cormorants (-15%). Heron eggs taken in Louisiana generally displayed a smaller post-1946 change than herons in the Middle West. Although DDE was a prominent factor for most groups, especially herons, in relation to the eggshell thinning observed, dieldrin and PCB's also were associated with thinning in some species. This relationship, however, may have been due to correlation in concentrations of these chemicals and concentrations of DDE.

The thinning of eggshells of the brown pelican has proven to be related to the concentrations of DDE in the eggs (Blus et al. 1971; Blus et al. 1972a, 1972b). Nearly all brown pelican eggs collected from 13 colonies in South Carolina, Florida, and California in 1969 and from 17 colonies in South Carolina and Florida in 1970 exhibited eggshell thinning (Blus 1970; Blus et al. 1974a). Of the 100 eggs analyzed for residues of pollutants, all eggs contained measurable quantities of DDE; most eggs contained measurable quantities of DDD, DDT, dieldrin, or PCB's. DDE appears to have been responsible for virtually all the eggshell thinning.

Nest success of brown pelicans in South Carolina was related to residues of DDE and dieldrin in sample eggs (Blus et al. 1974b). Residues of DDE seemed primarily responsible for nest failure; however, deleterious effects of this pollutant on nest success was not satisfactorily separated from those induced by dieldrin. Significant intercorrelation of all five organochlorine residues identified in the eggs complicated the relationship of residues to nest success. Maximum DDE residues in an egg from a successful nest were 2.4 ppm and in an egg from an unsuccessful nest, 8.5 ppm. Comparable maximum residues for dieldrin in sample eggs were 0.54 ppm (successful) and 0.99 ppm (unsuccessful). Residues of DDD, DDT, or PCB's in sample eggs were not significantly related to nest success. Reproductive success in the brown pelican colony was subnormal in the 2 years of study (1971 and 1972) but reproductive success was normal in those nests in which the sample egg contained either 2.5 ppm or less of DDE, or 0.54 ppm or less of dieldrin.

Residues of DDE, DDD, DDT, dieldrin, and PCB's exhibited a significant decline in South Carolina brown pelican eggs from 1969 through 1973 (Blus et al. 1977a), but the de-

crease in DDD was greatest. In 1973, the pelicans experienced excellent reproductive success for the first time in many years, and the decline in residues was related to this improvement. DDE was implicated as the agent responsible for most pollutant-induced nest failure; residues above 3.7 ppm in the sample egg were associated with total failure of those eggs remaining in the nest. The improvement in reproductive success was not associated with an increase in average eggshell thickness.

The peregrine falcon appears to be affected by shell thinning in all areas of its nearly global range thus far examined, including areas in the Aleutians (Peakall et al. 1975), Greenland (Walker et al. 1973), and coastal Chile (Walker et al. 1973) where they depend on marine food chains. On the Auckland Island in the sub-Antarctic, one egg of the New Zealand falcon contained DDE residues that were similar to those associated with shell thinning in the closely related peregrine (Bennington et al. 1975).

Peregrine falcons that breed along the coast of Scotland feed largely on seabirds, and these populations have declined in numbers at a time when populations that were preying on land birds in the interior remained stable (Ratcliffe 1972). A decline in reproductive success of the white-tailed eagle (*Haliaeetus albicilla*) in Germany has most likely been caused by DDE (Koeman et al. 1972b). In the Baltic, where white-tailed eagle populations declined during this century (Henriksson et al. 1966), very high concentrations of PCB and DDT compounds have been measured in eagles that were found dead (Jensen et al. 1972).

During an early study, the population of the Bermuda petrels (*Pterodroma cahow*) was undergoing an unexplained decline that was attributed to the presence of DDT (Wurster and Wingate 1968), but reproductive success subsequently improved. Reexamination of the tissues that had been analyzed for DDT, and analysis of dead chicks and unhatched eggs obtained subsequently, showed no changes in either DDT or PCB concentrations during the periods of poor reproductive success and subsequent recovery. Moreover, residues were comparatively low when related to those of other species of petrels in more contaminated areas (D. Wingate and R. W. Risebrough, unpublished data).

Shell thinning of eggs of the osprey in the northeastern United States where reproduction has been low and where population numbers have declined is also related to DDE concentrations (Spitzer et al. 1977). Dieldrin and PCB's also may have contributed to the rapid population decline in the affected areas in the Northeast, principally Connecticut (Wiemeyer et al. 1975).

In the Northeast, shell thinning has been documented in eggs of the gannets breeding on Bonaventure Island (J. A. Keith, personal communication). The breeding population of gannets, after increasing over the previous 80 years, declined by 16% between 1969 and 1973 (Nettleship 1975). In the recent past, DDT was extensively used in forest spray operations in adjacent areas of New Brunswick.

Patterns of reproductive failure in declining populations of several European and North American raptorial species were duplicated experimentally with captive American kestrels that were given a diet containing dieldrin and DDT, two commonly used organochlorine insecticides (Porter and Wiemeyer 1969). Major effects on reproduction were increased egg disappearance, increased egg destruction by parent birds, and reduced eggshell thickness.

In other experimental studies, DDE has caused significant eggshell thinning in captive screech owls (*Otus asio*) (McLane and Hall 1972) and American kestrels (Wiemeyer and Porter 1970). The levels of DDE found in the kestrel eggs in the second reproductive season of that study are similar to those found in British peregrine falcon eggs (Ratcliffe 1967).

Bald eagle eggs collected in 1968 from nests in Wisconsin, Maine, and Florida all contained residues of DDE, DDD, dieldrin, heptachlor epoxide, and PCB's (Krantz et al. 1970). Many also contained traces of DDT. Eggs from five nonproductive nests in Maine contained much higher residues than did eggs collected from either productive or nonproductive nests in Wisconsin and Florida.

Twenty-three bald eagle eggs collected in Alaska, Maine, Michigan, Minnesota, and Florida during 1969 and 1970 were analyzed for organochlorines and mercury (Wiemeyer et al. 1972). All eggs contained residues of DDE, dieldrin, PCB's, and mercury. Average residue concentrations were lowest in eggs

from Alaska. Significant eggshell thinning has occurred among eggs in samples from most major areas. Some eggs contained DDE residues of the same magnitude as those that produced shell thinning in experimental species. High dieldrin residues in some eggs could have an adverse effect on reproductive success.

Egg failure was the major cause of poor reproductive success of ospreys on the Potomac River during 1970 (Wiemeyer 1971). Many eggs disappeared between visits to the nests; some were found broken or damaged in the nests, and others failed to hatch.

Osprey eggs were exchanged between Connecticut and Maryland nests in 1968 and 1969 to determine which environmental factors might have contributed to the decline in reproductive success of Connecticut ospreys (Wiemeyer et al. 1975). Incubation of 30 Connecticut osprey eggs by Maryland ospreys did not improve the hatching rate. Forty-five Maryland osprey eggs incubated by Connecticut ospreys hatched at their normal rate. The results of the egg exchanges and associated observations indicated that the most probable cause of the poor reproduction of Connecticut ospreys was related to contamination of the birds and their eggs. Residues of DDT and its metabolites, dieldrin, and PCB's were generally higher in fish from Connecticut than from Maryland. There were no major changes in residue content of Connecticut eggs collected in 1968-69 compared with those collected in 1964. One Connecticut osprey had a concentration of dieldrin in its brain that was in the lethal range. The average shell thickness of recently collected osprey eggs from Connecticut had declined 18%, and those from Maryland had declined 10% from pre-1947 norms. Dieldrin, DDE, and PCB's are three environmental pollutants that have most likely been important factors in the greatly reduced reproductive success and rapid population decline of Connecticut ospreys.

All black duck eggs that were collected in 1971 from the northeastern United States and Canada contained DDE residues (Longcore and Mulhern 1973). Means for States and Provinces ranged from 0.09 to 5.94 ppm, with mean concentrations exceeding 1.0 ppm in eggs from Maine, New York, New Jersey, and Delaware. The highest DDE concentration (14.0 ppm) was in an egg from Delaware. The DDD and DDT residues averaged <0.5 ppm for each collection area. No mirex residues and only trace amounts of dieldrin and heptachlor epoxide were detected. Of the 61 eggs, 57 contained PCB's; means ranged from <0.05 ppm in samples from Nova Scotia to 3.30 ppm in those from Massachusetts, with trace amounts occurring in nearly half the samples. Mean organochlorine pesticide residues were lower in the 1971 samples than in those analyzed in an earlier study in 1964. Average shell thickness of eggs collected in 1964 (0.321 mm) was significantly less ($P < 0.01$) than that of eggs collected before 1940 (0.348 mm) or in 1971 (0.343 mm).

Eggs of captive black ducks fed diets containing DDE at 10 and 30 ppm (dry weight; approximately 3 and 9 ppm wet weight) experienced significant shell thinning and an increase in shell cracking when compared with eggs of untreated black ducks (Longcore et al. 1971b). Survival of ducklings from dosed parents in terms of "percentage of 21-day ducklings of embryonated eggs" was 40-76% lower than survival of ducklings from undosed parents. Average DDE residues in eggs from hens fed 10 and 30 ppm DDE were 46 ppm and 144 ppm.

In another experiment, black duck hens fed 10 ppm (dry weight) of DDE in the diet laid eggs with shells 22% thinner at the equator, 30% thinner at the cap, and 33% thinner at the apex than those of controls (Longcore and Samson 1973). Natural incubation increased shell cracking more than fourfold as compared with mechanical incubation. Hens removed cracked eggs from nests, and one hen terminated incubation. Hens fed DDE produced one-fifth as many ducklings as did the controls. The DDE in eggs of dosed hens averaged 64.9 ppm.

Concentrations of 10 and 40 ppm DDE (dry weight) in the feed of penned mallard ducks caused significant eggshell thinning and cracking and a marked increase in embryo mortality (Heath et al. 1969). In other studies, eggshell thinning also occurred in mallards fed DDE (Haegele and Hudson 1974), DDT (Tucker and Haegele 1970; Davison and Sell 1974), or dieldrin (Lehner and Egbert 1969; Muller and Lockman 1972; Davison and Sell 1974), but low dietary levels (25 and 50 ppm) of Aroclor 1254 produced no measurable reproductive effects (Heath et al. 1972b).

Diets containing various levels of DDT (at 20 ppm, dry weight, or greater), or dieldrin (at 10 ppm, dry weight) caused significant reduction in eggshell thickness, weight, and calcium in mallard ducks (Davison and Sell 1974). The reduction in eggshell thickness was linear with increasing dose of dieldrin through all levels studied.

Mallards were fed untreated feed or feed containing 40 ppm (dry weight) DDE, 40 ppm PCB, or 40 ppm DDE + PCB beginning a month before laying (Risebrough and Anderson 1975). Mean shell thickness indices were similar in the control and PCB groups, but they were reduced by 17% in the DDE group and 19% in the DDE + PCB group. The contents of 12 eggs randomly selected from the DDE group contained 373 ppm DDE (lipid basis), and 13 eggs from the DDE + PCB group contained mean residues of 344 ppm DDE + 364 ppm PCB (lipid basis). Egg production was similar in all groups for about the first 7 weeks, then it dropped markedly in the DDE + PCB group. Part, but not all, of this group's lower production of intact eggs was caused by egg eating. This behavior accounted for 18 of 282 eggs observed lost in the DDE + PCB group, 6 of 394 eggs in the PCB group, and none in the control and DDE groups. Although there was no significant change in shell thinning or DDE residues when PCB was added to the diet, the reduction in the number of intact eggs produced by the DDE + PCB group suggests that the two compounds may nevertheless interact to influence reproductive success.

Behavior

In England, gray herons (*Ardea cinerea*) have been observed breaking their own eggs, and others dropped their live young from the nest (Milstein et al. 1970; Prestt 1970). Such aberrant behavior may be related to sublethal organochlorine residues in the birds, as these authors suggested. The birds did not eat the eggshells, but tossed even the fragments from the nest. Therefore, the alternative possibility of calcium "hunger" does not seem to be true in herons.

Mallard ducks fed a diet containing 3 ppm DDE (dry weight; equal to about 0.6 ppm in a natural succulent diet) laid eggs that contained an average of 5.8 ppm DDE; ducklings that hatched from these eggs differed from controls in behavioral tests designed to measure responses to a maternal call and to a frightening stimulus (Heinz 1976b). In response to the maternal call, ducklings from parents fed DDE were hyper-responsive; compared with controls, a greater percentage approached the call and a greater percentage of those that approached remained near the call for the remainder of the test. In a test of avoidance behavior, ducklings whose parents were fed DDE traveled shorter distances from the frightening stimulus than did controls.

Coturnix quail chicks were given sublethal amounts of chlordane, dieldrin, endrin, DDE, or Aroclor 1254 in their feed, beginning at 7 days of age, and their avoidance response to a moving silhouette was measured daily for 14 days (Kreitzer and Heinz 1974). The birds were on dosage for 8 days, and on untreated feed for 6 days immediately thereafter. Group avoidance response was significantly suppressed (P from 0.01 to <0.001) by chlordane, dieldrin, endrin, and Aroclor 1254, but no effect of DDE on the birds' behavior could be detected. The behavior of the endrin-treated birds returned to normal after 2 days on untreated feed. The data indicated partial recovery for birds treated with dieldrin and chlordane, but none for those treated with Aroclor 1254.

Heavy Metals

The sources, occurrence, food web transfer, and toxicology of heavy metals and other trace elements must be understood to evaluate the significance of these chemicals to marine birds. These more general aspects have received considerable attention in recent symposia and reviews (Larsson 1970; Nelson et al. 1971; Gavis and Ferguson 1972; Eisler 1973; National Research Council of Canada 1974; Leland et al. 1975). Consequently, our discussion will be restricted to the more specific aspects of the exposure of aquatic birds to these chemicals, but will include some interpretive information relative to terrestrial avian species.

Most techniques that are used for measuring mercury residues in environmental samples determine levels of total mercury, regardless of the chemical form in which it

occurs. The various forms of mercury, however, differ widely in their toxicities. Unless otherwise specified, mercury concentrations presented here represent concentrations of total mercury.

Exposure of Marine Birds to Heavy Metals

Animals acquire heavy metals from the foods they eat, from the water that surrounds them, and possibly from the air they breathe. Quantities accumulated differ greatly among organisms, depending upon exposure and physiology (White and Stickel 1975).

Mercury in tissues of living organisms is often primarily in the more toxic methyl mercury form (Westoo 1967; Fimreite 1974), and methyl mercury is readily incorporated into the bodies of aquatic organisms (Leland et al. 1975). Most of the mercury in fish is in the form of methyl mercury (Koeman et al. 1975), but the high mercury concentrations discovered in the livers of six dead great cormorants and in livers of three others that were collected in the Netherlands were not primarily methyl mercury (Koeman et al. 1973). Mercury concentrations, primarily in forms other than methyl mercury, increased with age in some marine mammals and were correlated with concentrations of selenium and bromine (Koeman et al. 1975; Martin et al. 1976). Perhaps, like some marine mammals, cormorants may be able to detoxify methyl mercury by a chemical mechanism in which selenium and bromine are involved. However, mercury and selenium concentrations in livers of common murres and of a razorbill (Koeman et al. 1975) and in liver and breast muscle of sooty terns of known age were not correlated (P. G. Connors et al., unpublished manuscript). Inorganic and organic mercury from industrial sources may be converted into methyl mercury by some organisms, including birds (Jensen and Jernelov 1969; Kiwimae et al. 1969).

Mercury concentration increases with body weight, or age, in fish (Bache et al. 1971; Fimreite et al. 1971), crayfish (Vermeer 1972), and herons (Hoffman 1974). The concentration increases at higher trophic levels in fish, other aquatic organisms, fish-eating birds, or ducks (de Goeij 1971; Fimreite et al. 1971; Fimreite 1974; Hoffman 1974; Kleinert and DeGurse 1972; Vermeer et al. 1973; Baskett 1975).

Mercury concentrations in various tissues of the body are correlated with each other (Fimreite 1971; Koeman et al. 1971; Vermeer and Armstrong 1972a; Fimreite 1974; Heinz 1974, 1976a; Hoffman 1974). Eggs normally contain between a fifth and a ninth of the mercury concentration in the liver of the female (Fimreite et al. 1970; M. T. Finley, personal communication). Mercury in the liver of female California gulls (*Larus californicus*) averaged 5.5 times that in their eggs (Vermeer 1971a).

High mercury residues in aquatic organisms and in the related avifauna are often related to discharges from chlor-alkali plants, pulp mills, or other industrial plants that use mercury (Fimreite 1970; Fimreite et al. 1971; Nelson et al. 1971; Vermeer 1971a). Ospreys and great crested grebes (*Podiceps cristatus*) now have about 3 times as much mercury in some industrially contaminated areas as in uncontaminated areas (Larsson 1970).

In a survey of aquatic birds at 33 locations in Alberta, Saskatchewan, and Manitoba, mercury levels were generally higher in gulls (*Larus* spp.) and fish-eating birds than in ducks and geese (Vermeer 1971a). The highest mercury levels were found in herring gulls, possibly related to their scavenging and fish-eating habits.

Elevated mercury levels were found in livers of common mergansers (*Mergus merganser*; up to 86 ppm), common loons (90 ppm), and great blue herons (128 ppm) from Ontario (Fimreite 1974). Lower concentrations were found in mallards (12.5 ppm) and pintails (*Anas acuta*; 6.2 ppm). Mercury levels were higher in adults than in immatures. A chlorine plant about 80 km upstream from the collecting locality was believed to be the source of mercury found in the birds.

Mercury was present in spotted sandpiper (*Actitis macularia*) eggs collected upstream from Edmonton, Alberta, at lower concentrations (0.09 ppm) than in those eggs collected downstream (0.28 ppm), suggesting municipal or industrial contamination originating at Edmonton (Vermeer 1971b).

During another survey in Canada, highest concentrations of mercury in livers of fish-eating birds collected near sites of industrial contamination were in red-necked grebes

(*Podiceps grisegena*; Fimreite et al. 1971). Four common tern eggs averaged 0.58 ppm and two red-breasted merganser eggs averaged 0.81 ppm.

Aquatic bird eggs from the upper Great Lakes States contained higher mercury levels than those from Louisiana, although species represented from the two areas were not identical (Faber and Hickey 1973). Highest mean residues were in three species of mergansers (up to 1.6 ppm; red-breasted merganser). For those species with all eggs containing less than 0.25 ppm of mercury, the residues were considered to represent background levels. Mercury exceeded 1 ppm in one or more eggs of black-crowned night heron, hooded merganser (*Lophodytes cucullatus*), common merganser, and red-breasted merganser. Highest levels (up to 1.9 ppm) were in addled eggs of red-breasted mergansers.

Many birds dependent upon aquatic areas in the Lake St. Clair, Michigan, region have high residues of mercury in their tissues (Dustman et al. 1972). In 1970, carcasses, livers, and eggs were collected and analyzed. Mercury levels in great blue herons (up to 175 ppm in the liver; 23 ppm in the carcass) and common terns (up to 39 ppm in the liver; 7.5 ppm in the carcass) far exceeded those in any other species. The levels are comparable to those in birds in Sweden that died under experimental dosage with methyl mercury and in birds that died under field conditions in several Scandinavian countries with signs of mercury poisoning (Henriksson et al. 1966; Borg et al. 1969; Holt 1969). Mercury residues in eggs of all of the five common terns (up to 6.2 ppm), five of nine mallards (up to 2.7 ppm), three of the five black-crowned night herons (up to 1.1 ppm), and the single egg of a pied-billed grebe (*Podilymbus podiceps;* 4.0 ppm) were in the range of residues (0.5-3.1 ppm) in eggs of ring-necked pheasants whose reproductivity was reduced by mercury in experimental studies (Borg. et al. 1969; Fimreite 1971; Spann et al. 1972).

In 1973, eggs of some of these species were again collected at Lake St. Clair, following restrictions on industrial discharges of mercury into the St. Clair River (Stendell et al. 1976). Mercury levels in the eggs were appreciably lower than were found in these species in 1970. Common terns contained the highest residues (up to 1.3 ppm). Mallard eggs contained relatively low residue levels (< 0.05 to 0.26 ppm). Black-crowned night heron eggs (up to 0.76 ppm) and great egret eggs (up to 0.45 ppm) contained intermediate amounts.

Mercury levels generally are low in most species of ducks and geese but higher levels have been found in those species that consume a greater proportion of animal material in their diet (Kleinert and DeGurse 1972; Krapu et al. 1973; Fimreite 1974; Heath and Hill 1974). Among North American waterfowl species, the highest levels have been found in mergansers. Common mergansers from Onterio had up to 86 ppm mercury in their livers (Fimreite 1974). Hooded mergansers from Clay Lake, Ontario, contained up to 12.3 ppm and common goldeneyes (*Bucephala clangula*) up to 7.8 ppm in their breast muscle (Vermeer et al. 1973). Food items were also analyzed and crayfish (*Oronectes virilis*), which the hooded mergansers eat, contained the highest average concentration of mercury (7.1 ppm).

Mercury has been found in the visceral fat of black-footed albatrosses and Laysan albatrosses from Midway Atoll, North Pacific Ocean (Fisher 1973). Average residue levels in the Laysan albatrosses were 0.104 ppm and those in the black-footed were 0.075 ppm.

Mercury has been found in the livers of birds collected around the British coast (Dale et al. 1973). The highest concentration (26 ppm; converted from 122 ppm dry weight, see Holdgate 1971) was in a red-breasted merganser. Common eiders, which feed on mussels that are known to accumulate mercury, also had high concentrations (10 ppm). All of the more pelagic species, including black-legged kittiwakes, fulmars, and auks (*Alca torda* and *Alle alle*) had less than 2.2 ppm. Three gannets had slightly higher levels (up to 2.9 ppm). Herring gulls from oceanic islands contained relatively low mercury residues (up to 2.6 ppm) like the pelagic birds, but those from near shore had higher residues.

Common puffins collected around the coast of Britain contained up to 7.7 ppm (Parslow et al. 1972), and eiders from the Tay region had up to 0.45 ppm mercury in their livers (Jones et al. 1972).

Elevated levels of mercury have been found in birds of the Baltic region (Jensen et al. 1972). In 1969, mercury content of common murre secondaries had doubled the levels from 1906-1325. Mercury levels in murre eggs

were approximately the same in 1968 and 1969, averaging about 0.52 ppm, with the upper extreme concentration of 0.67 ppm. Even higher levels of mercury (3.7 ppm) were found in muscle tissue of great cormorants than in muscle of murres (0.9 ppm) or black guillemots (1.8 ppm) from the Baltic. Mercury in feathers (up to 51 ppm), muscle (up to 26 ppm), and brains (up to 14 ppm) of white-tailed eagles exceeded the levels in other species. Mercury concentrations in the kidneys (48-123 ppm) and muscle tissue (1.9-8.5 ppm) of other white-tailed eagles from the same area further indicate that the species may have serious mercury pollution problems (Henriksson et al. 1966). Bald eagles in the United States also occasionally contain high levels of mercury (up to 43 ppm) in their carcasses (Belisle et al. 1972).

Mercury levels (figures not specifically stated) in the muscle of eiders and sandwich terns of the Dutch Wadden Sea appear 3 to 5 times higher than the levels considered representative of natural background (de Goeij 1971). Analyses were also made of various organs of three common murres and one razorbill that were found as oiled birds along the Dutch coast (Koeman et al. 1975): mercury in the livers did not exceed 2.5 ppm; selenium in the liver of one common murre was 4.6 ppm, but the levels of these metals were not correlated with each other.

There were no significant geographical or species differences in two essential heavy metals (copper and zinc) in Antarctic and North American petrels (Anderlini et al. 1972). Silver, cobalt, and lead were difficult to detect at the low levels that were found, but there were no detectable differences in their concentrations. Cadmium, chromium, nickel, and mercury levels in petrels suggested a correlation of increasing concentration with increased exposure to industrialized areas. Higher concentrations of these metals in ashy petrels are probably the result of their feeding in the proximity of San Francisco Bay.

Livers from ruddy ducks killed by an oil spill on the Delaware River contained detectable levels of lead, cadmium, and mercury (White and Kaiser 1976). Lead ranged from 0.19 to 0.61 ppm, cadmium from 0.27 to 1.60 ppm, and mercury from 0.06 to 0.74 ppm. Residues of these metals were similar to those found in canvasbacks from the Chesapeake Bay region (D. H. White and R. C. Stendell, unpublished manuscript).

Mercury residues in the livers of six gannets from the Irish Sea (4 ppm; 18.4 ppm dry weight) averaged higher than in two gannets from eastern Scotland (1.6 ppm; 7.3 ppm dry weight) that died during unrelated large-scale mortality incidents (Parslow et al. 1973). Average levels of copper (7.4 ppm; 34 ppm dry weight) and zinc (64.8 ppm; 298 ppm dry weight) in the livers of gannets from the Irish Sea also were higher than in two others from eastern Scotland (2.8 ppm copper; 26.3 ppm zinc). The differences in the metal concentrations between the two groups were considered the result of the differences in liver sizes. Although the cause of the gannet deaths could not be established, heavy metal concentrations in the birds apparently were responsible for the death of only one individual with high mercury levels (22 ppm; 98 ppm dry weight). Lead and cadmium concentrations were below the limits of detection in all of these birds, but another gannet that died in an earlier incident had measurable residues of lead (0.2 ppm) and cadmium (2.0 ppm).

Mercury concentrations in the livers and kidneys of common murres that died in the seabird wreck in the Irish Sea during autumn 1969 did not exceed 5 ppm (23 ppm dry weight) (Holdgate 1971). Some of the birds showed relatively high levels of particular metals and in some the highest concentrations were above the level at which poisoning may have occurred. However, in general, the range of mercury levels in the casualties of the incident and in the healthy birds shot for comparison overlap. The levels of mercury (up to 5 ppm), lead (8.7 ppm), cadmium (2.8 ppm), and arsenic (8.3 ppm) in the livers and kidneys of some birds appeared elevated.

Biological Effects of Heavy Metals on Marine Birds

Toxicology, Physiology, and Pathology

In 1953 a severe neurological disorder caused by mercury poisoning was first recognized among people living in the vicinity of Minamata Bay, Japan (Kurland et al. 1960). Toxic effects and similar histopathological changes have been reported for fish, birds, and mammals that died as a result of mercury

poisoning, but no particular studies have been made on the toxicity of heavy metals to seabirds (Parslow et al. 1973). However, in certain terrestrial species, symptoms of poisoning might be expected when mercury concentrations in liver or kidney tissues reach about 30 ppm (W. H. Stickel 1971). By contrast, normal levels are less than 1 ppm.

Although death may not have been caused by mercury poisoning, mercury in the livers of adult great egrets found dead in California ranged between 2 and 9.5 ppm (Faber et al. 1972). Mercury in the liver (22 ppm; 98 ppm dry weight) of a gannet from the Irish Sea could have caused the bird's death (Parslow et al. 1973).

Female mallards fed 3 ppm mercury (dry weight) as methyl mercury in their diet had average mercury residues of 11.1 ppm in their livers, 14.7 ppm in their kidneys, 5.0 ppm in their muscles, 4.6 ppm in their brains, and 5.5 to 7.4 ppm in their eggs (Heinz, 1976a). Males had higher residues, and many of the ducklings from these parents died within 1 week after hatching (Heinz 1974, 1976a). The ducklings also had high levels of mercury in their tissues.

In short-term tests of lethal dietary toxicity of pesticidal chemicals, Ceresan M, a fungicide containing ethyl mercury, was relatively more toxic to young mallards than were 37 other compounds (Heath et al. 1972a). Only endrin and Dasanit were more toxic. In similar subsequent tests, Morsodren, another organomercurial fungicide, was also highly toxic to young mallards (Hill et al. 1975).

Mercury potentiated the toxicity and biochemical effects of parathion in coturnix quail fed a sublethal concentration of Morsodren (4 ppm dry weight as methyl mercury) for 18 weeks (Dieter and Ludke 1975). Mean residue concentrations in these birds were 21 ppm of mercury in the liver and 8.4 ppm in the carcass. The computed LD_{50} of parathion was 5.86 mg/kg in birds not fed Morsodren and 4.24 in those fed the heavy metal. When challenged with a sublethal oral dose of parathion (1.0 mg/kg), Morsodren-fed birds exhibited significantly greater inhibition of plasma and brain cholinesterase activity than controls.

After administration of various mercury compounds to domestic chickens (*Gallus gallus*), the methyl mercury compounds were rather evenly distributed among the organs, whereas the other mercury compounds, organic and inorganic, gave very high concentrations in the liver and kidneys compared with other organs (Kiwimae et al. 1969). Differences in the proportion of methyl mercury compounds to total mercury occurred in the white and the yolk of the eggs from these hens. Although the proportion in the white was similar to that in the blood and the muscles, the proportion in the yolk was similar to that in the liver and the kidneys. The albumen contained mainly methyl mercury compounds in concentrations that varied with the compound given to the hens. The methyl mercury concentration in albumen was always much lower when other compounds were administered than when the hens were given the same quantity of methyl mercury hydroxide.

In another study, mercury was not detectable in the albumen but was present at high levels in the yolk following intravenous injection of mercuric nitrate into laying coturnix quail (Nishimura et al. 1971).

Evaluation of enzymatic profiles appears to be a potentially valuable technique for monitoring the presence of toxicants in wild populations, especially if used to complement standard chemical residue analysis (Dieter 1975). Lactate dehydrogenase activity increased twofold and cholinesterase activity decreased in birds fed Morsodren. After feeding for 3 weeks, mercury in starling carcasses reflected the concentrations fed daily, whereas the concentration in the livers was 2 to 4 times that in the diet.

A decrease in cholinesterase activity occurred in male coturnix quail that were fed diets for 12 weeks containing graded levels of mercuric chloride (Dieter 1974). At 12 weeks the decrease was proportional to the log dose received, although this was not true after 2 and 4 weeks on the treated diet. Mercury residues attained in the tissues were 5% or less of those in the feed.

There was a marked sexual difference in rates of mercury loss in coturnix quails (Backstrom 1969). Males lost little of the mercury in their bodies, especially from the brain and muscle, in 30 days, but females had a marked loss in this period, largely because of excretion in eggs. Ring-necked pheasants lost 33% to 50% of the mercury from their livers and

kidneys in 2 months, and approximately 99% was lost in 6 months (Borg et al. 1969). Ospreys apparently have a similar loss rate of mercury (Johnels et al. 1968).

When methyl mercury dicyandiamide was fed to mallard ducks at a concentration of 3 ppm mercury (dry weight), mercury accumulated in the eggs to an average of 7.2 and 5.5 ppm in 2 successive years (Heinz and Locke 1976). Mercury in the eggs caused brain lesions in ducklings. Lesions included demyelination, neuron degeneration, necrosis, and hemorrhage in the meninges overlying the cerebellum. Brains of dead ducklings contained an average of 6.2 and 5.2 ppm mercury in the 2 successive years.

Upon necropsy, ring-necked pheasants that were killed after receiving 4.2 ppm mercury (dry weight) in their diet for 350 days appeared normal, but those that received greater concentrations (12.5, 37.4, or 112 ppm, dry weight) died during the experiment and showed variable amounts of subcutaneous edema and decreasing amounts of subcutaneous and abdominal adipose tissue as survival time on the treated diet increased (Spann et al. 1972). Birds that died on the higher dosages showed signs of neurological disturbance, including ataxia and torticollis, before death.

Lead poisoning has long been recognized as a serious problem for waterfowl (Wetmore 1919; Jordan and Bellrose 1951; Bellrose 1959). Histopathological changes occur in the kidneys of mallards as a result of lead shot ingestion (Locke et al. 1966, 1967). In addition, significant changes in activity of three enzymes often used to assess hepatic damage occurred in mallard ducks following oral administration of lead shot (Rozman et al. 1974).

The ingestion of one number 4 lead shot by each of 80 pen-reared mallards that were fed whole-kernel corn caused 19% mortality within an average of 20 days (Longcore et al. 1974a). Coating or alloying lead with other metals only delayed mortality among dosed ducks. Disintegrable lead shot with water-soluble binder and lead-containing biochemical additives were as toxic to mallards as commercial lead shot.

Lead levels in brains, tibiae, and breast muscle of mallard ducks that died and in tibiae of those that were sacrificed increased significantly from dosage with one number 4 lead shot (about 1.4 g) until death (Longcore et al. 1974b). In mallard ducks, lead levels exceeding 3 ppm in the brain, 6 to 20 ppm in the kidney or liver, or 10 ppm in clotted blood from the heart indicated acute exposure to lead.

One month after dosage, mean lead levels in mallards given one number 4 all-lead shot were about twice those in tissues of mallards given one number 4 lead-iron shot that contained about 50% lead (Finley et al. 1976a). Necropsy of sacrificed ducks failed to reveal any of the tissue lesions usually associated with lead poisoning in waterfowl. Lead in the blood of ducks dosed with all-lead shot averaged 0.64 ppm, and 0.28 ppm in ducks given lead-iron shot. Lead residues in livers and kidneys of females given all-lead shot were significantly higher than in males. In both dosed groups, lead levels in wingbones of the females were about 10 times those in males, and were significantly correlated with the number of eggs laid after dosage. It appeared that after the laying hens ingested sublethal amounts of lead shot, high lead deposition in the bone occurred as a result of mobilization of calcium from the bone during eggshell formation. Lead levels in contents and shells of eggs laid by hens dosed with all-lead shot were about twice those in eggs laid by hens dosed with lead-iron shot. Lead levels in eggshells best reflected levels of lead in the blood.

The inverse correlation between delta-aminolevulinic acid dehydratase (ALAD) activity and blood lead concentrations was highly significant in canvasback ducks from the Chesapeake Bay (Dieter et al. 1976). ALAD is an important enzyme in hemoglobin synthesis. The activity of this enzyme in the blood provides a sensitive and precise estimate of lead contamination in waterfowl. In mallards, lead concentrations in blood were strongly correlated with erythrocyte ALAD activity, suggesting that biochemical response to two types of lead shot (one all-lead, the other containing 50% lead) depends upon the quantity of lead present (Finley et al. 1976b).

Reproduction

Mercury levels (3.5 to 11 ppm) in the eggs of Swedish white-tailed eagles that failed to hatch indicate that the decline in reproduction of this species could be attributed to mercury poisoning (Borg et al. 1969). A corresponding

decline in this species in Finland also was associated with mercury contamination (Henriksson et al. 1966). However, as discussed earlier, organochlorines also may be partially responsible for the observed decline.

There were apparently no young produced by common loons in Clay and Ball Lakes, Ontario, in 1970 and 1971 (Fimreite 1974). (Both lakes receive effluent from a chlorine plant.) Fledging success of common terns at Ball Lake was 10% of normal, but fledging was normal at nearby Wabigoon Lake, where birds contained lower residues. Average total mercury in the eggs was 3.6 and 1.0 ppm; average methyl mercury was 2.4 and 0.8 ppm in the two colonies.

There are considerable differences between species in susceptibility to mercury pollutants. Mercury concentrations as high as 16 ppm in western Ontario herring gull eggs apparently did not affect their hatchability (Vermeer et al. 1973), but 0.5 to 1.5 ppm mercury in ring-necked pheasant eggs reduced hatchability, reduced egg weight and production, and produced a large number of eggs without shells (Fimreite 1971).

Concentrations of mercury found in the livers of abnormal young terns (*Sterna hirundo* and *S. dougallii*) from Great Gull Island (in Long Island Sound) ranged from 0.2 to 1.2 ppm, but were not thought to have caused the abnormalities (Hays and Risebrough 1972). Livers of normal young terns were not analyzed. Hatchability in the Great Gull Island colony has consistently been greater than 90%, but hatchability of common tern eggs in Lake Ontario colonies has been low. Concentrations of heavy metals in common terns were studied to determine the reason for the difference in hatchability. Concentrations of cadmium, chromium, cobalt, copper, lead, mercury, nickel, silver, and zinc in bone, liver, breast muscle, and kidneys of adult birds from the two locations were similar (Conners et al. 1975). Therefore, these metals apparently were not responsible for the differences in hatchability.

Although the reproductive effects of mercury in other species are largely unknown, mercury residues of 0.5 ppm were associated with poor reproductive success in an experimental study with ring-necked pheasants (Fimreite 1971). Average mercury residues in field-collected eggs of four species of aquatic-related birds on the Niagara peninsula, Ontario, were between 0.5 and 1 ppm (Frank et al. 1975). These included red-winged blackbird (*Agelaius phoeniceus;* 0.68 ppm), herring gull (0.74 ppm), black-crowned night heron (0.64 ppm), and common tern (0.83 ppm).

Mercury was found in measurable quantities in all of the 100 brown pelican eggs from 13 colonies in South Carolina, Florida, and California (Blus et al. 1974a). Six of the 21 pelican eggs from South Carolina contained 0.5 ppm or more of mercury. Sixteen of the 49 pelican eggs from Florida contained 0.5 ppm or more of mercury, and 1 on the verge of hatching contained 1.43 ppm.

Methyl mercury at low dietary levels (0.5 or 3.0 ppm, dry weight, equal to about 0.1 or 0.6 ppm mercury on the basis of a natural succulent diet) caused lowered reproductive success in experimental mallards and black ducks. Mallards fed 3 ppm mercury in their diet during one reproductive season showed reproductive impairment, but none was evident among birds fed 0.5 ppm (Heinz 1974). Adverse effects in the group fed 3 ppm included a decrease in egg laying, an increase in embryonic mortality, and reduced duckling survival. These effects resulted in the production of less than half (46.5%) as many 1-week-old ducklings as the controls. Levels of mercury reached about 1 ppm in eggs of the birds fed 0.5 ppm mercury and between 6 and 9 ppm in the eggs from ducks fed 3 ppm mercury.

The hens from the first reproductive season were kept on diets containing mercury into a second season (Heinz 1976a). During the second season, levels of mercury in eggs from hens on these diets averaged 0.79 and 5.46 ppm. On a dry-weight basis, the concentration of mercury in eggs was about 5 times that in the feed. There were no significant differences in egg production or hatching success among control birds and those fed mercury. However, duckling survival decreased: ducklings from hens fed 3 ppm mercury during the two reproductive seasons were less likely to survive to 1 week of age than were controls or ducklings from parents fed 0.5 ppm mercury.

Mallards whose parents were fed a diet containing 0.5 ppm mercury (dry weight) were themselves fed a diet containing 0.5 ppm mercury (dry weight) from 9 days of age through their first reproductive season (Heinz 1976c). Mercury in the eggs of these hens fed mercury

averaged 0.86 ppm. Hens fed mercury made less efficient use of feed and laid a greater percentage of their eggs outside their nest boxes compared with controls. They also produced fewer 1-week-old ducklings than did controls, although there had been no difference in duckling production by their parents fed 0.5 ppm mercury in the preceding years. The ducklings from dosed parents did not grow as fast as did those from controls.

Black ducks given a diet containing 3 ppm mercury (dry weight) as methyl mercury hatched fewer eggs than did controls and fewer of their ducklings survived (Finley and Stendell 1978). Average mercury residues in brain, liver, and muscle of ducklings that died (3.7, 9.4, and 4.9 ppm) were about twice those in tissues of ducklings sacrificed at 4 weeks of age (1.6, 5.7, and 2.1 ppm).

Mercury residues in pheasant eggs were 0.9 to 3.1 ppm following administration of 4.2 ppm mercury (dry weight) in their diet (Spann et al. 1972). The birds exhibited greatly reduced egg production and increased embryo mortality in the few eggs laid.

Mercury residues in bobwhite eggs from birds fed a dietary concentration of 1.7 ppm (dry weight; administered as ethyl mercury p-toluene sulfonanilide) averaged 1.6 ppm (J. W. Spann and R. G. Heath, unpublished manuscript). There was a significantly greater mortality among young whose parents received mercury in the diet. The principal period of increased mortality included the last 5 days of incubation and the first day after hatching.

Behavior

The behavior of mallard ducks whose parents were fed a control diet or a diet containing 0.5 or 3.0 ppm mercury (dry weight) as methyl mercury was studied (Heinz 1975). There was no significant difference among controls and ducklings from mercury-treated parents in the percentage of ducklings that approached the tape-recorded maternal call. However, control ducklings moved back and forth toward the call more than ducklings from mercury-treated parents and also spent more time in the end of the runway near the loudspeaker than ducklings whose parents were fed a diet containing 0.5 ppm mercury. Compared to control ducklings, those from parents fed a diet containing either mercury concentration were hyper-responsive in avoidance behavior tests.

Among mallard ducklings produced in the 2nd year of the study in which hens were fed a control diet or a diet that contained 0.5 or 3 ppm mercury (dry weight), the findings were similar (Heinz 1976a). There were no significant differences among controls and groups fed mercury in approach responses toward a recorded maternal call and ducklings from mercury-treated parents were hyper-responsive compared with controls in avoidance behavior.

In the second generation, there were no significant differences between controls and ducklings from parents fed 0.5 ppm mercury in approach responses to tape-recorded maternal calls, in avoidance of a frightening stimulus, or in open-field behavior (Heinz 1976c).

Plastic and Other Artifacts

Small plastic beads and irregularly shaped particles up to 0.5 cm in diameter are commonly found in plankton samples from widely separated oceanic areas, including the northwestern Atlantic, Sargasso Sea, Bristol Channel (United Kingdom), and the coastal waters of southern New England (Carpenter and Smith 1972; Carpenter et al. 1972; Morris and Hamilton 1974; Colton et al. 1974). The particles are primarily composed of polystyrene or polyethylene compounds and have about the same density as seawater. Their various colors include white, green, brown, blue, red, or clear (Carpenter and Smith 1972; Morris and Hamilton 1974; Colton et al. 1974). The polystyrene spherules evidently are of industrial origin, because they have been found in the effluents from manufacture of polystyrene (Hayes and Cormons 1974; Morris and Hamilton 1974). Their abundance in the British Channel water was lowest near the seaward end and greatest in the inner part of the Channel, near the Holm Islands. Benthic sediments near the Holm Islands contained as many as 20,000 beads/m^2 (Morris and Hamilton 1974).

Small fish ingest the beads and particles (Carpenter et al. 1972; Kartar et al. 1973).

Plastic particles have been found in the stomachs of fork-tailed petrels, horned puffins (*Fratercula corniculata*), and parakeet auklets (*Cyclorrhynchus psittacula*) from the Aleutians (G. J. Divoky and C. M. White, personal communication), as well as in the stomachs of adult and nestling Leach's petrels from Newfoundland and New Brunswick (Rothstein 1973).

Gulls and terns regurgitate indigestible parts of their food, such as bits of shell and fish bones. Polystyrene particles have also been found in these pellets (Hays and Cormons 1974).

It is not known whether the birds ingest the plastic particles directly, but petrels apparently do. Other marine birds may acquire particles in their stomachs by consuming fish that have previously ingested the plastic particles.

Evidence of harmful effects of plastic particles to any species is lacking, except for the possibility of intestinal blockage in smaller fish (Carpenter et al. 1972). However, they do accumulate in the environment, are eaten by fish, and are found in the stomachs of marine birds. It has been suggested that the plastics industry develop products that are degradable, but the most likely outcome of such an effort would be introduction of finished products that would disintegrate into smaller particles similar to those described here (Hays and Cormons 1974).

Rubber thread cuttings may represent a hazard to marine birds. Common puffins, in particular, appear to mistake them for fish and swallow them. These elastic threads form knots and the tangled mass may remain in the stomach. In one case the entangled elastic was tightly packed into the gizzard exit; in another it had formed a ball of rubber in the gizzard itself. Although the rubber threads may not kill the birds, there is a possibility that they make them less able to withstand other stresses (Parslow and Jefferies 1972).

Although other artifacts, such as trash scattered on beaches or jetsam washed ashore, may contribute significantly to the mortality of certain species of marine birds (Gochfeld 1973), in other circumstances, such debris may enhance the habitability of an area. An apparent increase in the number of black guillemots breeding in the Barrow, Alaska, area appears to be associated with the local increase in man-made debris. The birds typically nest in cavities in rock cliffs and crevices in talus slopes. Because such nest sites are absent in the Barrow area, guillemots have nested in an empty oil drum, under a collapsed building, and under other types of man-made debris (Divoky et al. 1974).

No explanation has been found for the appearance along the Northumberland (United Kingdom) coast of severely debilitated common murres whose plumage has been extensively abraded. Fluoride, discharged by a nearby aluminum smelter, was considered a possible cause because the birds had a strong odor resembling chlorine, another closely related halogen compound. There also were similarities between the signs observed in the affected birds and those observed in cases of acute or chronic fluorosis in other animals. The implication of fluoride was dismissed, however, in part, on the basis of low fluoride residue levels in bone, skin, internal organs, and digestive tract of the affected birds. Further, normal murre feathers were not damaged by soaking in various fluorine compounds, in samples of smelter effluent, and in undiluted scrubber liquid (Croxall 1972).

Recommendations

The levels of any pollutant, or combination of pollutants, in the marine environment should remain below a level that damages the viability of any population or species of marine bird. Thus the global use of organochlorine compounds must be regulated, if necessary, to restrict input into the sea. The undersea exploitation of petroleum, the marine transport of petroleum, and the activities of coastal refining and petrochemical industries must also be regulated to prevent harm to local populations of marine birds.

Much remains to be learned about the exposure of marine birds to environmental pollutants in northern North America. The most critical areas for study include the effects of chronic sublethal exposure to petroleum hydrocarbons, certain organochlorines, and mercury. The possible synergistic effects of these compounds in marine birds should also be intensively studied.

A long-term program to monitor increasing or decreasing levels of any particular pollu-

tant in the marine environment, with particular reference to the levels that affect the most sensitive species of marine bird, is necessary. A portion of this program might be carried out by using the eggs of marine birds, because colonies of some species are large and eggs may be obtained on a regular basis. The variance of pollutant distributions and the mathematical nature of these distributions are imperfectly known and the statistics of sampling have not yet been adequately formulated. Moreover, it would be desirable to carry out such programs in conjunction with other programs that examine changes in pollutant levels in the marine environment like the "Mussel Watch" (Goldberg 1975), which is following changes in the levels of plutonium isotopes, petroleum compounds, chlorinated hydrocarbons, and selected metals in mussels from U.S. coastal localities.

Priorities in future research might be given to more intensive studies within local areas to obtain a better understanding of the dynamics of pollutant accumulation by birds. Of primary concern is the need to determine whether petroleum compounds are accumulated in food webs, including marine birds, and whether such compounds exert deleterious physiological effects. Because petroleum compounds seem to have longer-lasting effects in colder water, the impending exploitation of oil resources in the offshore and North Slope areas accentuates the urgent need for information on the environmental consequences of chronic as well as acute exposure.

The environmental effects of small plastic particles that are commonly found in oceanic areas, including northern North America, should be investigated.

The relationships between chronic exposure to environmental pollutants and other environmental stresses are relatively unknown, as are relationships and effects of pollutants on many of the essential organisms in the food webs upon which marine and estuarine birds depend.

An annual symposium on the marine birds of northern North America should be held to serve as a forum for presentation of new information. The symposium would contribute significantly to conservation of the area's natural resources by facilitating exchange of information and coordination of further research.

Acknowledgments

D. J. Snyder assembled much of the required literature and also typed the manuscript. M. T. Finley, J. L. Ludke, L. F. Stickel, and D. H. White reviewed the manuscript and offered useful suggestions.

References

Abou-Donia, M. B., and D. B. Menzel. 1968. The metabolism *in vivo* of 1,1,1-trichloro-2,2-bis-(p-chlorophenyl)ethane (DDT), 1,1-dichloro-2,2-bis-(p-chlorophenyl)ethane (DDD) and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) in the chick by embryonic injection and dietary ingestion. Biochem. Pharmacol. 17(10):2143-2161.

Ahearn, D. G., and S. P. Meyers. 1973. The microbial degradation of oil pollutants. Publ. No. LSU-SG-73-01, Center for Wetland Resources, Louisiana State University, Baton Rouge.

Albright, L. J., T. G. Northcote, P. C. Oloffs, and S. Y. Szeto. 1975. Chlorinated hydrocarbon residues in fish, crabs, and shellfish of the lower Fraser River, its estuary, and selected locations in Georgia Strait, British Columbia — 1972-73. Pestic. Monit. J. 9(3):134-140.

Aldrich, J. W. 1970. Review of the problem of birds contaminated by oil and their rehabilitation. U.S. Fish Wildl. Serv. Resour. Publ. 87. 23 pp.

American Petroleum Institute. 1973. Effects of oil and chemically dispersed oil on selected marine biota — a laboratory study. API Publ. No. 4191 (prepared by Battelle Pacific Northwest Laboratories, Richland, Washington).

Anderlini, V. C., P. G. Connors, R. W. Risebrough, and J. H. Martin. 1972. Concentrations of heavy metals in some Antarctic and North American sea birds. Pages 49-61 *in* B. C. Parker, ed., Proceedings of the Colloquium on Conservation Problems in Antarctica. Allen Press, Lawrence, Kansas.

Anderson, D. W., and J. J. Hickey. 1972. Eggshell changes in certain North American birds. Proc. Int. Ornith. Congr. 15:514-540.

Anderson, D. W., J. R. Jehl, Jr., R. W. Risebrough, L. A. Woods, Jr., L. R. DeWeese, and W. G. Edgecomb. 1975. Brown pelicans: improved reproduction off the southern California coast. Science 190(4216):806-808.

Anderson, J. W. 1973. Uptake and depuration of specific hydrocarbons from fuel oil by the bivalves *Rangia cuneata* and *Crassostrea virginica.* Pages 690-708 *in* Background papers for a workshop on inputs, fates, and effects of petroleum in the marine environment, Vol. II. Ocean Affairs Board, National Academy of Sciences, Washington, D.C.

Anderson, J. W., J. M. Neff, B. A. Cox, H. E. Tatem, and G. M. Hightower. 1974a. Characteristics of dispersions and water-soluble extracts of crude and refined oils and their toxicity to estuarine crustaceans and fish. Mar. Biol. 27(1):75-88.

Anderson, J. W., J. M. Neff, and S. R. Petrocelli. 1974b. Sublethal effects of oil, heavy metals, and PCBs on marine organisms. Pages 83-121 *in* M. A. Q. Khan and J. P. Bederka, Jr., eds. Survival in toxic environments. Academic Press, New York. 553 pp.

Anonymous. 1975. Tanker skipper tracked, arrested in July oil spill. Washington Post, 8 November.

Atema, J., and L. Stein. 1972. Sublethal effects of crude oil on lobster (*Homarus americanus*) behavior. Woods Hole, Massachusetts Oceanographic Institution, Tech. Rep. WHOI-72-74. 81 pp.

Atema, J., and L. Stein. 1974. Effects of crude oil on the feeding behavior of the lobster *Homarus americanus*. Environ. Pollut. 6(2):77-86.

Bache, C. A., W. H. Gutenmann, and D. J. Lisk. 1971. Residues of total mercury and methylmercuric salts in lake trout as a function of age. Science 172(3986):951-952.

Backstrom, J. 1969. Distribution studies of mercuric pesticides in quail and some fresh-water fishes. Acta Pharmacol. Toxicol. 27(Suppl. 3). 103 pp.

Bailey, E. P., and G. H. Davenport. 1972. Die-off of common murres on the Alaska Peninsula and Unimak Island. Condor 74(2):215-219.

Baskett, T. S. 1975. Mercury residues in breast muscles of wild ducks. Pestic. Monit. J. 9(2):67-78.

Beer, J. V. 1968. Post-mortem findings in oiled auks dying during attempted rehabilitation. Pages 123-129 *in* Symposium on The Biological Effects of Oil Pollution on Littoral Communities, February 17-19, 1968. Pembroke, Wales.

Belisle, A. A., W. L. Reichel, L. N. Locke, T. G. Lamont, B. M. Mulhern, R. M. Prouty, R. B. DeWolf, and E. Cromartie. 1972. Residues of organochlorine pesticides, polychlorinated biphenyls, and mercury and autopsy data for bald eagles, 1969 and 1970. Pestic. Monit. J. 6(3):133-138.

Bellrose, F. C. 1959. Lead poisoning as a mortality factor in waterfowl populations. Bull. Ill. Nat. Hist. Surv. 27(3):235-288.

Bennington, S., P. G. Connors, C. Connors, and R. W. Risebrough. 1975. Patterns of chlorinated hydrocarbon contamination in New Zealand sub-Antarctic and coastal marine birds. Environ. Pollut. 8(2): 135-147.

Blumer, M. 1971. Scientific aspects of the oil spill problem. Environ. Aff. 1:54-73.

Blumer, M., J. M. Hunt, J. Atema, and L. Stein. 1973. Interaction between marine organisms and oil pollution. EPA Office of Research and Monitoring, Ecol. Res. Ser. Rep. No. EPA-R3-73-042. 97 pp.

Blumer, M., and J. Sass. 1972. The West Falmouth oil spill. Woods Hole, Massachusetts Oceanographic Institution, Tech. Rep. 72-19.

Blumer, M., G. Souza, and J. Sass. 1970. Hydrocarbon pollution of edible shellfish by an oil spill. Mar. Biol. 5(3):195-202.

Blus, L. J. 1970. Measurements of brown pelican eggshells from Florida and South Carolina. BioScience 20(15):867-869.

Blus, L. J., A. A. Belisle, and R. M. Prouty. 1974a. Relations of the brown pelican to certain environmental pollutants. Pestic. Monit. J. 7(3/4):181-194.

Blus, L. J., C. D. Gish, A. A. Belisle, and R. M. Prouty. 1972a. Logarithmic relationship of DDE residues to eggshell thinning. Nature 235(5338):376-377.

Blus, L. J., C. D. Gish, A. A. Belisle, and R. M. Prouty. 1972b. Further analysis of the logarithmic relationship of DDE residues to eggshell thinning. Nature 240(5377):164-166.

Blus, L. J., R. G. Heath, C. D. Gish, A. A. Belisle, and R. M. Prouty. 1971. Eggshell thinning in the brown pelican: implication of DDE. BioScience 21(24):1213-1215.

Blus, L. J., B. S. Neely, Jr., A. A. Belisle, and R. M. Prouty. 1974b. Organochlorine residues in brown pelican eggs: relation to reproductive success. Environ. Pollut. 7(2):81-91.

Blus, L. J., B.S. Neely, Jr., T. G. Lamont, and B. Mulhern. 1977a. Residues of organochlorines and heavy metals in tissues and eggs of brown pelicans, 1969-73. Pestic. Monit. J. 11(1):40-53.

Blus, L. J., S. N. Wiemeyer, J. A. Kerwin, R. C. Stendell, H. M. Ohlendorf, and L. F. Stickel. 1977b. Impact of estuarine pollution on birds. Pages 57-71 *in* Estuarine pollution control and assessment. Proceedings of a Conference, Vol. 1. U.S. Environmental Protection Agency, Office of Water Planning and Standards, Washington, D.C.

Boehm, P.D., and J. G. Quinn. 1974. The solubility behaviour of No. 2 fuel oil in seawater. Mar. Pollut. Bull. 5(7):101-105.

Bogan, J. A., and W. R. P. Bourne. 1972. Organochlorine levels in Atlantic seabirds. Nature 240(5380):358-359.

Borg, K., H. Wanntorp, K. Erne, and E. Hanko. 1969. Alkyl mercury poisoning in terrestrial Swedish wildlife. Viltrevy 6(4):301-379.

Bourne, W. R. P. 1968. Oil pollution and bird populations. Pages 99-121 *in* Symposium on the Biological effects of oil pollution on littoral communities, February 17-19, 1968. Pembroke, Wales.

Bourne, W. R. P. 1974. Guillemots with damaged primary feathers. Mar. Pollut. Bull. 5(6):88-90.

Bourne, W. R. P., and C. J. Bibby. 1975. Temperature and the seasonal and geographical occurrence of oiled birds on West European beaches. Mar. Pollut. Bull. 6(5):77-80.

Bourne, W. R. P., and J. A. Bogan. 1972. Polychlorinated biphenyls in North Atlantic seabirds. Mar. Pollut. Bull. 3(11):171-175.

Bourne, W. R. P., J. D. Parrack, and G. R. Potts. 1967. Birds killed in the *Torrey Canyon* disaster. Nature 215:1123-1125.

Bowes, G. W., M. J. Mulvihill, B. R. Simoneit, A. L. Burlingame, and R. W. Risebrough. 1975. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature 256(5515):305-307.

Boylan, D. B., and B. W. Tripp. 1971. Determination of hydrocarbons in sea water extracts of crude oils and crude oil fractions. Nature 230:44-47.

Boyle, C. L. 1969. Oil pollution of the sea: is the end in sight? Biol. Conserv. 1(4):319-329.

Braestrup, L., J. Clausen, and O. Berg. 1974. DDE, PCB and aldrin levels in arctic birds of Greenland. Bull. Environ. Contam. Toxicol. 11(4):326-332.

Buck, W. F. A., and J. G. Harrison. 1967. Some prolonged effects of oil pollution on the Medway Estuary. Pages 32-33 *in* The wildfowlers' association of Great Britain and Ireland, annual report and yearbook, 1966-1967.

Burns, K. A., and J. M. Teal. 1971. Hydrocarbon incorporation into the salt marsh ecosystem from the West Falmouth oil spill. NTIS [Natl. Tech. Inf. Serv.] Rep. No. NOAA-73032608.

Burns, K. A., and J. M. Teal. 1973. Hydrocarbons in the pelagic *Sargassum* community. Deep Sea Res. 20:207-211.

Carlson, R. W., and R. T. Duby. 1973. Embryotoxic effects of three PCBs in the chicken. Bull. Environ. Contam. Toxicol. 9(5):261-264.

Carpenter, E. J., S. J. Anderson, G. R. Harvey, H. P. Miklas, and B. B. Peck. 1972. Polystyrene spherules in coastal waters. Science 178(4062):749-750.

Carpenter, E. J., and K. L. Smith, Jr. 1972. Plastics on the Sargasso Sea surface. Science 175(4027):1240-1241.

Carry, C. W., and J. A. Redner. 1970. Pesticides and heavy metals: progress report, December 1970. County Sanitation Districts of Los Angeles County, 2020 Beverly Boulevard, Los Angeles. 51 pp.

Cecil, H. C., J. Bitman, R. J. Lillie, G. F. Fries, and J. Verrett. 1974. Embryotoxic and teratogenic effects in unhatched fertile eggs from hens fed polychlorinated biphenyls (PCB's). Bull. Environ. Contam. Toxicol. 11(6):489-495.

Clark, R. B. 1969. Oil pollution and the conservation of seabirds. Pages 76-112 *in* Proceedings of the International Conference on Oil Pollution of the Sea, 1968. Rome.

Clark, R. B. 1973. Impact of chronic and acute oil pollution on seabirds. Pages 619-634 *in* Background papers for a workshop on inputs, fates, and effects of petroleum in the marine environment, Vol. II. Ocean Affairs Board, National Academy of Science, Washington, D.C.

Clark, R. B., and J. R. Kennedy. 1968. Rehabilitation of oiled seabirds. Department of Zoology, University of Newcastle upon Tyne, England. 57 pp.

Clark, R. B., and J. R. Kennedy. 1971. How oiled seabirds are cleaned. Research Unit on the Rehabilitation of Oiled Seabirds. Department of Zoology, University of Newcastle upon Tyne, England. 48 pp.

Colton, J. B., Jr., F. D. Knapp, and B. R. Burns. 1974. Plastic particles in surface waters of the northwestern Atlantic. Science 185(4150):491-497.

Connors, P. G., V. C. Anderlini, R. W. Risebrough, M. Gilbertson, and H. Hays. 1975. Investigations of metals in common tern populations. Can. Field-Nat. 89(2):157-162.

Cooke, A. S. 1973. Shell thinning in avian eggs by environmental pollutants. Environ. Pollut. 4(2):85-152.

Coulson, J. C., G. R. Potts, I. R. Deans, and S. M. Fraser. 1968. Exceptional mortality of shags and other seabirds caused by paralytic shellfish poison. British Bands 61(9):381-404.

Coulter, M. C., and R. W. Risebrough. 1973. Shell thinning in eggs of the ashy petrel (*Oceanodroma homochroa*) from the Farallon Islands. Condor 75(2):254-255.

Crocker, A. D., J. Cronshaw, and W. N. Holmes. 1974. The effect of a crude oil on intestinal absorption in ducklings (*Anas platyrhynchos*). Environ. Pollut. 7(3):165-177.

Crocker, A. D., J. Cronshaw, and W. N. Holmes. 1975. The effect of several crude oils and some petroleum distillation fractions on intestinal absorption in ducklings (*Anas platyrhynchos*). Environ. Physiol. Biochem. 5(2):92-106.

Cromartie, E., W. L. Reichel, L. N. Locke, A. A. Belisle, T. E. Kaiser, T. G. Lamont, B. M. Mulhern, R. M. Prouty, and D. Swineford. 1975. Residues of organochlorine pesticides and polychlorinated biphenyls and autopsy data for bald eagles, 1971-72. Pestic. Monit. J. 9(1):11-14.

Croxall, J. P. 1972. Guillemots killed by fluoride? Mar. Pollut. Bull. 3(10):149-152.

Dale, I. M., M. S. Baxter, J. A. Bogan, and W. R. P. Bourne. 1973. Mercury in seabirds. Mar. Pollut. Bull. 4(5):77-79.

Davis, J. B. 1967. Petroleum microbiology. Elsevier Publishers, New York.

Davison, K. L., and J. L. Sell. 1974. DDT thins shells of eggs from mallard ducks maintained on *ad libitum* or controlled-feeding regimens. Arch. Environ. Contam. Toxicol. 2(3):222-232.

de Goeij, J. J. M. 1971. Determination of mercury in samples from the Dutch environment. Presented at the American Nuclear Society Topical Meeting on Nuclear Methods in Environmental Research, August 23-24, 1971. University of Missouri, Columbia.

Dieter, M. P. 1974. Plasma enzyme activities in coturnix quail fed graded doses of DDE, polychlorinated biphenyl, malathion and mercuric chloride. Toxicol. Appl. Pharmacol. 27(1):86-98.

Dieter, M. P. 1975. Further studies on the use of enzyme profiles to monitor residue accumulation in wildlife: plasma enzymes in starlings fed graded concentrations of morsodren, DDE, Aroclor 1254, and malathion. Arch. Environ. Contam. Toxicol. 3(2):142-150.

Dieter, M. P., and J. L. Ludke. 1975. Studies on combined effects of organophosphates and heavy metals in birds. I. Plasma and brain cholinesterase in coturnix quail fed methyl mercury and orally dosed with parathion. Bull. Environ. Contam. Toxicol. 13(3):257-262.

Dieter, M. P., M. C. Perry, and B. M. Mulhern. 1976. Lead and PCB's in canvasback ducks: relationship between enzyme levels and residues in blood. Arch. Environ. Contam. Toxicol. 5(1):1-13.

Divoky, G. J., G. E. Watson, and J. C. Bartonek. 1974. Breeding of the black guillemot in northern Alaska. Condor 76(3):339-343.

Doguchi, M. 1973. Chlorinated hydrocarbons in the environment in the Kanto Plain and Tokyo Bay, as reflected in fishes, birds and man. Pages 269-289 *in* F. Coulston, F. Korte, and M. Goto, eds. New methods in environmental chemistry and toxicology. International Academy Printing Company, Totsuka, Tokyo.

Dustman, E. H., L. F. Stickel, and J. B. Elder. 1972. Mercury in wild animals, Lake St. Clair 1970. Pages 46-52 *in* R. Hartung and B. D. Dinman, eds. Environmental mercury contamination. Ann Arbor Science Publishers, Ann Arbor, Michigan.

Eisler, R. 1973. Annotated bibliography on biological effects of metals in aquatic environments. EPA Office of Research and Monitoring, EPA-R3-73-007. 287 pp.

Environmental Protection Agency. 1973. Environmental contamination from hexachlorobenzene, July 20, 1973. Office of Toxic Substances, EPA, Washington, D.C.

Evans, D. R., and S. D. Rice. 1974. Effects of oil on marine ecosystems: a review for administrators and policy makers. Fish. Bull. 72(3):625-638.

Faber, R. A., and J. J. Hickey. 1973. Eggshell thinning, chlorinated hydrocarbons, and mercury in inland aquatic bird eggs, 1969 and 1970. Pestic. Monit. J. 7(1):27-36.

Faber, R. A., R. W. Risebrough, and H. M. Pratt. 1972. Organochlorines and mercury in common egrets and great blue herons. Environ. Pollut. 3(2):111-122.

Farrington, J. W. 1977. Oil pollution in the coastal environment. Pages 385-400 *in* Estuarine pollution control and assessment. Proceedings of a Conference, Vol. II. U.S. Environmental Protection Agency, Office of Water Planning and Standards, Washington, D.C.

Fimreite, N. 1970. Mercury uses in Canada and their possible hazards as sources of mercury contamination. Environ. Pollut. 1(2):119-131.

Fimreite, N. 1971. Effects of dietary methylmercury on ring-necked pheasants. Can. Wildl. Serv. Occas. Pap. No. 9. 37 pp.

Fimreite, N. 1974. Mercury contamination of aquatic birds in northwestern Ontario. J. Wildl. Manage. 38(1):120-131.

Fimreite, N., R. W. Fyfe, and J. A. Keith. 1970. Mercury contamination of Canadian prairie seed eaters and their avian predators. Can. Field-Nat. 84(3):269-276.

Fimreite, N., W. M. Holsworth, J. A. Keith, P. A. Pearce, and I. M. Gruchy. 1971. Mercury in fish and fish-eating birds near sites of industrial contamination in Canada. Can. Field-Nat. 85(3):211-220.

Finley, M. T., M. P. Dieter, and L. N. Locke. 1976a. Lead in tissues of mallard ducks dosed with two types of lead shot. Bull. Environ. Contam. Toxicol. 16(3):261-269.

Finley, M. T., M. P. Dieter, and L. N. Locke. 1976b. Delta-aminolevulinic acid dehydratase: inhibition in ducks dosed with lead shot. Environ. Res. 12(2):243-249.

Finley, M. T., and R. C. Stendell. 1978. Survival and reproductive success of black ducks fed mercury. Environ. Pollut. In press.

Fisher, H. I. 1973. Pollutants in North Pacific albatrosses. Pacific Sci. 27(3):220-225.

Flegg, J. J. M. 1971. Puffins in peril. B. T. O. News 46(1):1-2.

Flegg, J. J. M. 1972. Puffins on St. Kilda 1964-1971. Bird Study 19(1):7-17.

Fossato, V. U. 1975. Elimination of hydrocarbons by mussels. Mar. Pollut. Bull. 6(1):7-10.

Fox, G. A., A. P. Gilman, D. J. Hallett, R. J. Norstrom, F. I. Onuska, and D. B. Peakall. 1975. Herring gull productivity and toxic chemicals in the Great Lakes in 1975. Can. Wildl. Serv. Manuscript Rep. No. 34. 35 pp.

Frank, R., M. Van Hove Holdrinet, and W. A. Rapley. 1975. Residue of organochlorine compounds and mercury in birds' eggs from the Niagara Peninsula, Ontario. Arch. Environ. Contam. Toxicol. 3(2):205-218.

Frankenfeld, J. W. 1973. Factors governing the fate of oil at sea; variations in the amounts and types of dissolved or dispersed materials during the weathering process. Pages 485-495 *in* Proceedings of the Joint Conference on Prevention and Control of Oil Spills, 1973. American Petroleum Institute, Washington, D.C.

Friend, M., M. A. Haegele, and R. Wilson. 1973. DDE: interference with extrarenal salt excretion in the mallard. Bull. Environ. Contam. Toxicol. 9(1):49-53.

Fujiwara, K. 1974. Environmental and food contamination with PCB's in Japan. Department of Hygienic Chemistry, Kyoto City Institute of Public Hygiene. 45 pp.

Gavis, J., and J. F. Ferguson. 1972. The cycling of mercury through the environment. Water Res. 6(9):989-1008.

Gish, C. D., and N. J. Chura. 1970. Toxicity of DDT to Japanese quail as influenced by body weight, breeding condition, and sex. Toxicol. Appl. Pharmacol. 17(3):740-751.

Gochfeld, M. 1973. Effects of artifact pollution on the viability of seabird colonies on Long Island, New York. Environ. Pollut. 4(1):1-6.

Goldberg, E. D., convener. 1972. Baseline studies of heavy metal, halogenated hydrocarbon, and petroleum hydrocarbon pollutants in the marine environment and research recommendations. Deliberations of the International Decade of Ocean Exploration Baseline Conference, May 24-26, 1972. IDOE, National Science Foundation, Washington, D.C.

Goldberg, E. D. 1975. The Mussel Watch — a first step in global marine monitoring. Mar. Pollut. Bull. 6(7):111.

Greenwood, J. J. D. 1970. The problem of oiled seabirds. Paper presented at the Conference of Local Authorities and Other Bodies. Marryat Hall, Dundee, Scotland.

Gress, F., R. W. Risebrough, D. W. Anderson, L. F. Kiff, and J. R. Jehl. 1973. Reproductive failures of double-crested cormorants in Southern California and Baja California. Wilson Bull. 85(2):197-208.

Gress, F., R. W. Risebrough, and F. C. Sibley. 1971. Shell thinning in eggs of common murres, *Uria aalge*, from the Farallon Islands, California. Condor 73(3):368-369.

Guillon, J. C. 1967. Les effets tardifs de l'intoxication par le mazout chex les oiseaux [The delayed effects of crude oil poisoning in birds]. L'Homme et L'Oiseau 9:15-16.

Haegele, M. A., and R. H. Hudson. 1974. Eggshell thinning and residues in mallards one year after DDE exposure. Arch. Environ. Contam. Toxicol. 2(4):356-363.

Hartung, R. 1965. Some effects of oiling on reproduction of ducks. J. Wildl. Manage. 29(4):872-874.

Hartung, R. 1967. Energy metabolism in oil-covered ducks. J. Wildl. Manage. 31(4):798-804.

Hartung, R., and G. S. Hunt. 1966. Toxicity of some oils to waterfowl. J. Wildl. Manage. 30(3):564-570.

Hawkes, A. L. 1961. A review of the nature and extent of damage caused by oil pollution at sea. Trans. N. Am. Wildl. Nat. Resour. Conf. 26:343-355.

Hay, K. G. 1975. Petroleum industry's role in marine bird conservation. Prepared for the International Symposium on Conservation of Marine Birds in Northern North America, Seattle, May 13-15, 1975. 20 pp.

Hays, H., and G. Cormons. 1974. Plastic particles found in tern pellets, on coastal beaches and at factory sites. Mar. Pollut. Bull. 5(3):44-46.

Hays, H., and R. W. Risebrough. 1972. Pollutant concentrations in abnormal young terns from Long Island Sound. Auk 89(1):19-35.

Heath, R. G., and S. A. Hill. 1974. Nationwide organochlorine and mercury residues in wings of adult mallards and black ducks during the 1969-70 hunting season. Pestic. Monit. J. 7(3/4):153-164.

Heath, R. G., J. W. Spann. E. F. Hill, and J. F. Kreitzer. 1972a. Comparative dietary toxicities of pesticides to birds. U.S. Fish Wildl. Serv., Spec. Sci. Rep.-Wildl. 152. 57 pp.

Heath, R. G., J. W. Spann, and J. F. Kreitzer. 1969. Marked DDE impairment of mallard reproduction in controlled studies. Nature 224(5214):47-48.

Heath, R. G., J. W. Spann, J. F. Kreitzer, and C. Vance. 1972b. Effects of polychlorinated biphenyls on birds. Proc. Int. Ornithol. Congr. 15:475-485.

Heinz, G. 1974. Effects of low dietary levels of methyl mercury on mallard reproduction. Bull. Environ. Contam. Toxicol. 11(4):386-392.

Heinz, G. 1975. Effects of methylmercury on approach and avoidance behavior of mallard ducklings. Bull. Environ. Contam. Toxicol. 13(5):554-564.

Heinz, G. 1976a. Methylmercury: second-year feeding effects on mallard reproduction and duckling behavior. J. Wildl. Manage. 40(1):82-90.

Heinz, G. 1976b. Behavior of mallard ducklings from parents fed 3 ppm DDE. Bull. Environ. Contam. Toxicol. 16(6):640-645.

Heinz, G. 1976c. Methylmercury: second-generation reproductive and behavioral effects on mallard ducks. J. Wildl. Manage. 40(4):710-715.

Heinz, G., and L. N. Locke. 1976. Brain lesions in mallard ducklings from parents fed methylmercury. Avian Dis. 20(1):9-17.

Henriksson, K., E. Karppanen, and M. Helminen. 1966. High residue of mercury in Finnish white-tailed eagles. Ornis. Fenn. 43(2):38-45.

Hickey, J. J., and D. W. Anderson. 1968. Chlorinated hydrocarbons and eggshell changes in raptorial and fish-eating birds. Science 162(3850):271-273.

Hill, E. F., R. G. Heath, J. W. Spann, and J. D. Williams. 1974. Polychlorinated biphenyl toxicity to Japanese quail as related to degree of chlorination. Poult. Sci. 53(2):597-604.

Hill, E. F., R. G. Heath, J. W. Spann, and J. D. Williams. 1975. Lethal dietary toxicities of environmental pollutants to birds. U.S. Fish Wildl. Serv., Spec. Sci. Rep.-Wildl. 191. 61 pp,

Hoffman, R. D. 1974. Mercury in herons, egrets, and their nesting environment. M.S. Thesis. Ohio State University, Columbus. 78 pp.

Holdgate, M. W., ed. 1971. The seabird wreck of 1969 in the Irish Sea, autumn 1969. Nat. Environ. Res. Coun. Publ. Ser. C, No. 4. London. 17 pp.

Holt, G. 1969. Mercury residues in wild birds in Norway: 1965-1967. Nord. Veterinamed. 21(2):105-114.

Hope-Jones, P., G. Howells, E. I. S. Rees, and J. Wilson. 1970. Effect of "Hamilton Trader" oil on birds in the Irish Sea in May 1969. British Birds 63(3):97-110.

Hutzinger, O., S. Safe, and V. Zitko. 1974. The Chemistry of PCB's. CRC Press, Cleveland, Ohio.

Jacobson, S. M., and D. B. Boylan. 1973. Seawater soluble fraction of kerosene: effect on chemotaxis in a marine snail, *Nassarius obsoletus*. Nature 241(5386):213-215.

Jensen, S., and A. Jernelov. 1969. Biological methylation of mercury in aquatic organisms. Nature 223(5207):753-754.

Jensen, S., A. G. Johnels, M. Olsson, and G. Otterlind. 1969. DDT and PCB in marine animals from Swedish waters. Nature 224(5216): 247-250.

Jensen, S., A. G. Johnels, M. Olsson, and E. Westermark. 1972. The avifauna of Sweden as indicators of environmental contamination with mercury and chlorinated hydrocarbons. Proc. Int. Ornith. Congr. 15:455-465.

Johnels, A. G., M. Olsson, and T. Westermark. 1968. *Esox lucius* and some other organisms as indicators of mercury contamination in Swedish lakes and rivers. Bull. Off. Int. Epiz. 69(9-10):1439-1452.

Jones, A. M., Y. Jones, and W. D. P. Stewart. 1972. Mercury in marine organisms of the Tay region. Nature 238(5360):164-165.

Jordan, J. S., and F. C. Bellrose. 1951. Lead poisoning in wild waterfowl. Ill. Nat. History Surv., Biological Notes No. 26. 27 pp.

Kartar, S., R. A. Milne, and M. Sainsbury. 1973. Polystyrene waste in the Severn Estuary. Mar. Pollut. Bull. 4(9):144.

Keith, J. A., and I. M. Gruchy. 1972. Residue levels of chemical pollutants in North American birdlife. Proc. Int. Ornith. Congr. 15:437-454.

Ketchum, B. H., V. Zitko, and D. Saward. 1975. Aspects of heavy metal and organohalogen pollution in aquatic ecosystems. Pages 75-90 *in* A. D. McIntyre and C. F. Mills, eds. Ecological toxicology research — effects of heavy metal and organohalogen compounds. Plenum Press, New York. 323 pp.

King, W. B., and J. L. Lincer. 1973. DDE residues in the endangered Hawaiian dark-rumped petrel (*Pterodroma phaeopygia sandwichensis*). Condor 75(4):460-461.

Kiwimae, A., A. Swensson, U. Ulfvarson, and G. Westoo. 1969. Methylmercury compounds in eggs from hens after oral administration of mercury compounds. J. Agric. Food Chem. 17(5):1014-1016.

Kleinert, S. J., and P. E. DeGurse. 1972. Mercury levels in Wisconsin fish and wildlife. Wisc. Dep. Nat. Resour. Tech. Bull. 52. 22 pp.

Koeman, J. H., T. Bothof, R. de Vries, H. Van Velzen-blad, and J. G. Vos. 1972a. The impact of persistent pollutants on piscivorous and molluscivorous birds. TNO-Nieuws 27:561-569.

Koeman, J. H., J. Garssen-Hoekstra, E. Pels, and J. J. M. de Goeij. 1971. Poisoning of birds of prey by methyl mercury compounds. Meded. Fac. Landbouwwet Rijksuniv Gent. 36(1):43-49.

Koeman, J. H., R. H. Hadderingh, and M. F. I. J. Bijleveld. 1972b. Persistent pollutants in the white-tailed eagle in the Federal Republic of Germany. Biol. Conserv. 4(5):373-377.

Koeman, H. J., M. C. Ten Noever de Brauw, and R. H. deVos. 1969. Chlorinated biphenyls in fish, mussels, and birds from the River Rhine and the Netherlands coastal area. Nature 221(5186):1126-1127.

Koeman, J. H., W. S. M. van de Ven, J. J. M. de Goeij, P. S. Tijoe, and J. L. van Haaften. 1975. Mercury and selenium in marine mammals and birds. Sci. Total Environ. 3(3):279-287.

Koeman, J. H., and H. van Genderen. 1965. Some preliminary notes on residues of chlorinated hydrocarbon insecticides in birds and mammals in the Netherlands. Meded. Landbouwhogesch. Opzoekingsstn. Staat. Gent. 33(3):1879-1887.

Koeman, J. H., and H. van Genderen. 1966. Some preliminary notes on residues of chlorinated hydrocarbon insecticides in birds and mammals in the Netherlands. J. Appl. Ecol. 3(Suppl.):99-106.

Koeman, J. H., and H. van Genderen. 1972. Tissue levels in animals and effects caused by chlorinated hydrocarbon insecticides, chlorinated biphenyls and mercury in the marine environment along the Netherlands coast. Pages 1-8 *in* Marine pollution and sea life. The Whitefriars Press, Ltd. London.

Koeman, J. H., H. C. Van Velzen-Blad, R. de Vries, and J. G. Vos. 1973. Effects of PCB and DDE in cormorants and evaluation of PCB residues from an experimental study. J. Reprod. Fertil. (Suppl.) 19:353-364.

Koeman, J. H., J. Veen, E. Brouwer, L. Huisman de Brouwer, and J. L. Koolen. 1968. Residues of chlorinated hydrocarbon insecticides in the North Sea environment. Helgol. wiss. Meersunters. 17:375-380.

Kopischke, E. D. 1972. The effect of 2,4-D and diesel fuel on egg hatchability. J. Wildl. Manage. 36(4):1353-1356.

Krantz, W. C., B. M. Mulhern, G. E. Bagley, A. Sprunt, IV, F. J. Ligas, and W. B. Robertson, Jr. 1970. Organochlorine and heavy metal residues in bald eagle eggs. Pestic. Monit. J. 3(3):136-140.

Krapu, G., G. A. Swanson, and H. K. Nelson. 1973. Mercury residues in pintails breeding in North Dakota. J. Wildl. Manage. 37(3):395-399.

Kreitzer, J. F., and G. H. Heinz. 1974. The effect of sublethal dosages of five pesticides and a polychlorinated biphenyl on the avoidance response of coturnix quail chicks. Environ. Pollut. 6(1):21-29.

Kreitzer, J. F., and J. W. Spann. 1973. Tests of pesticidal synergism with young pheasants and Japanese quail. Bull. Environ. Contam. Toxicol. 9(4):250-256.

Kurland, L. T., S. N. Faro, and H. Siedler. 1960. Minamata disease. World Neurol. 1(5):370-395.

Larsson, J. E. 1970. Environmental mercury research in Sweden. Swedish Environmental Protection Board, Stockholm. 44 pp.

Lee, C. C., W. K. Craig, and P. J. Smith. 1974. Water-soluble hydrocarbons from crude oil. Bull. Environ. Contam. Toxicol. 12(2):212-217.

Lee, R. F., R. Sauerheber, and A. A. Benson. 1972a. Petroleum hydrocarbons: uptake and discharge by the marine mussell, *Mytilus edulis*. Science 177(4046):344-346.

Lee, R. F., R. Sauerheber, and G. H. Dobbs. 1972b. Uptake, metabolism, and discharge of polycyclic aromatic hydrocarbons by marine fish. Mar. Biol. 17(3):201-208.

Lehner, P. N., and A. Egbert. 1969. Dieldrin and eggshell thickness in ducks. Nature 224(5225):1218-1219.

Leland, H. V., E. D. Copenhaver, and D. J. Wilkes. 1975. Heavy metals and other trace elements. J. Water Pollut. Control Fed. 47(6):1635-1656.

Lincoln, F. C. 1936. Effects of oil pollution on waterfowl. Trans. N. Am. Wildl. Conf. 1:555-564.

Linden, O. 1975. Acute effects of oil and oil/dispersant mixture on larvae of Baltic herring. Ambio 4(3):130-133.

Locke, L. N., G. E. Bagley, and H. D. Irby. 1966. Acid-fast intranuclear inclusion bodies in the kidneys of mallards fed lead shot. Bull. Wildl. Dis. Assoc. 2(4):127-131.

Locke, L. N., H. D. Irby, and G. E. Bagley. 1967. Histopathology of mallards dosed with lead and selected substitute shot. Bull. Wildl. Dis. Assoc. 3(4):143-147.

Longcore, J. R., R. Andrews, L. N. Locke, G. E. Bagley, and L. T. Young. 1974a. Toxicity of lead and proposed substitute shot to mallards. U.S. Fish Wildl. Serv., Spec. Sci. Rep.-Wildl. 183. 23 pp.

Longcore, J. R., L. N. Locke, G. E. Bagley, and R. Andrews. 1974b. Significance of lead residues in mallard tissues. U.S. Fish Wildl. Serv., Spec. Sci. Rep.-Wildl. 182. 24 pp.

Longcore, J. R., and B. M. Mulhern. 1973. Organochlorine pesticides and polychlorinated biphenyls in black duck eggs from the United States and Canada — 1971. Pestic. Monit. J. 7(1):62-66.

Longcore, J. R., and F. B. Samson. 1973. Eggshell breakage by incubating black ducks fed DDE. J. Wildl. Manage. 37(3):390-394.

Longcore, J. R., F. B. Samson, J. F. Kreitzer, and J. W. Spann. 1971a. Changes in mineral composition of eggshells from black ducks and mallards fed DDE in the diet. Bull. Environ. Contam. Toxicol. 6(4):345-350.

Longcore, J. R., F. B. Samson, and T. W. Whittendale, Jr. 1971b. DDE thins eggshells and lowers reproductive success of captive black ducks. Bull. Environ. Contam. Toxicol. 6(6):485-490.

Ludke, J. L. 1974. Interaction of dieldrin and DDE residues in Japanese quail (*Coturnix coturnix japonica*). Bull. Environ. Contam. Toxicol. 11(4):297-302.

Ludke, J. L. 1976. Organochlorine pesticide residues associated with mortality: additivity of chlordane and endrin. Bull. Environ. Contam. Toxicol. 16(3):253-260.

Martin, J. H., P. D. Elliott, V. C. Anderlini, D. Girvin, S. A. Jacobs, R. W. Risebrough, R. L. DeLong, and W. G. Gilmartin. 1976. Mercury-selenium-bromine imbalance in premature parturient California sea lions. Mar. Biol. 35(1):91-104.

Matsumura, F. 1974. Microbial degradation of pesticides. Pages 129-154 *in* M. A. Q. Khan and J. P. Bederka, Jr., eds. Survival in toxic environments. Academic Press, Inc., New York. 553 pp.

McEwan, E. H., and A. F. C. Koelink. 1973. The heat production of oiled mallards and scaup. Can. J. Zool. 5(1):27-31.

McLane, M. A. R., and L. C. Hall. 1972. DDE thins screech owl eggshells. Bull. Environ. Contam. Toxicol. 8(2):65-68.

Miller, D. S., W. B. Kinter, D. B. Peakall, and R. W. Risebrough. 1976. DDE feeding and plasma osmoregulation in ducks, guillemots and puffins. Am. J. Physiol. 23(2):370-376.

Milstein, P. le S., I. Prestt, and A. A. Bell. 1970. The breeding cycle of the grey heron. Ardea 58(3-4):171-257.

Moore, N. W., and J. O'G. Tatton. 1965. Organochlorine insecticide residues in the eggs of sea birds. Nature 207:42-43.

Moore, S. F., and R. L. Dwyer. 1974. Effects of oil on marine organisms: a critical assessment of published data. Water Res. 8(10):819-827.

Morris, A. W., and E. I. Hamilton. 1974. Polystyrene spherules in the Bristol Channel. Mar. Pollut. Bull. 5(2):26-27.

Mulhern, B. M., and W. L. Reichel. 1970. The effect of putrefaction of eggs upon residue analysis of DDT and metabolites. Bull. Environ. Contam. Toxicol. 5(3):222-225.

Mulhern, B. M., W. L. Reichel, L. N. Locke, T. G. Lamont, A. A. Belisle, E. Cromartie, G. E. Bagley, and R. M. Prouty. 1970. Organochlorine residues and autopsy data from bald eagles 1966-68. Pestic. Monit. J. 4(3):141-144.

Muller, H. D., and D. C. Lockman. 1972. Fecundity and progeny growth following subacute insecticide ingestion by the mallard. Poult. Sci. 51(1):239-241.

National Academy of Sciences. 1973. Background papers for a workshop on inputs, fates, and effects of petroleum in the marine environment. Vol. I and II. Ocean Affairs Board, Washington, D.C.

National Academy of Sciences. 1975a. Petroleum in the marine environment: inputs, techniques for analysis, fates, and effects. Ocean Affairs Board, Washington, D.C. 107 pp.

National Academy of Sciences. 1975b. Assessing potential ocean pollutants: A report of The Study Panel on Assessing Potential Ocean Pollutants to the Ocean Affairs Commission on Natural Resources, National Research Council. Washington, D.C. 438 pp.

National Research Council of Canada. 1974. Proceedings of the International Conference on Transport of Persistent Chemicals in Aquatic Ecosystems. Ottawa, Canada, May 1-3, 1974. 320 pp.

Nelson, N. (Chairman), T. C. Byerly, A. C. Kolbye, Jr., L. T. Kurland, R. E. Shapiro, S. I. Shibko, W. H. Stickel, J. E. Thompson, L. A. Van Den Berg, and A. Weissler. 1971. Hazards of mercury. Environ. Res. 4(1):1-69.

Nettleship, D. N. 1975. A recent decline of gannets, *Morus bassanus*, on Bonaventure Island, Quebec. Can. Field-Nat. 89(2):125-133.

Nisbet, I. C. T., and A. F. Sarofim. 1972. Rates and routes of transport of PCBs in the environment. Environ. Health Perspect. 1(1):21-38.

Nishimura, M., N. Urakawa, and M. Ideka. 1971. An autoradiographic study on the distribution of mercury and its transfer to the egg in the laying quail. Jpn. J. Pharmacol. 21(5):651-659.

Ohlendorf, H. M., E. E. Klaas, and T. E. Kaiser. 1974. Environmental pollution in relation to estuarine birds. Pages 53-81 *in* M. A. Q. Khan and J. P. Bederka, eds. Survival in toxic environments. Academic Press, Inc., New York. 553 pp.

Orr, R. T. 1971. Oil, wildlife and people. Pac. Discovery 24:24-29.

Panel on Hazardous Trace Substances. 1972. PCBs — Environmental impact. Environ. Res. 5:249-362.

Parslow, J. L. F. 1967. Changes in status among breeding birds in Britain and Ireland. British Birds 60(1):2-47.

Parslow, J. L. F. 1970. Oil pollution and seabirds. Colloquium on Pollution of the Sea by Oil Spills. November 2-6, 1970, NATO Headquarters, Brussels.

Parslow, J. L. F. 1973. Pollutants in guillemot and kittiwake eggs from Lundy. Lundy Field Soc. Annu. Rep. 23:31-37.

Parslow, J. L. F., and D. J. Jefferies. 1972. Elastic thread pollution of puffins. Mar. Pollut. Bull. 3(3):43-45.

Parslow, J. L. F., and D. J. Jefferies. 1973. Relationship between organochlorine residues in livers and whole bodies of guillemots. Environ. Pollut. 5(2):87-101.

Parslow, J. L. F., D. J. Jefferies, and M. C. French. 1972. Ingested pollutants in puffins and their eggs. Bird Study 19(1):18-33.

Parslow, J. L. F., D. J. Jefferies, and H. M. Hanson. 1973. Gannet mortality incidents in 1972. Mar. Pollut. Bull. 4(3):41-44.

Peakall, D. B. 1975. PCB's and their environmental effects. Crit. Rev. Environ. Control 5:469-508.

Peakall, D. B., T. J. Cade, C. M. White, and J. R. Haugh. 1975. Organochlorine residues in Alaskan peregrines. Pestic. Monit. J. 8(4):255-260.

Phillips, J. 1974. Oiled seabirds successfully cleaning their plumage. British Birds 67(11):483.

Porter, R. D., and S. N. Wiemeyer. 1969. Dieldrin and DDT: effects on sparrow hawk eggshells and reproduction. Science 165(3889): 199-200.

Porter, R. D., and S. N. Wiemeyer. 1972. DDE at low dietary levels kills captive American kestrels. Bull. Environ. Contam. Toxicol. 8(4):193-199.

Prestt, I. 1970. Organochlorine pollution of rivers and the heron (*Ardea cinerea* L.). Pages 95-102 *in* Papers Proceedings of the International Union for Conservation of Nature and Natural Resources 11th Tech. Meeting, New Delhi, India, November 25-28, 1969.

Prestt, I. 1971. Techniques for assessment of pollution effects on seabirds. Proc. R. Soc. Lond. B. 177:287-294.

Prestt, I., and D. A. Ratcliffe. 1972. Effects of organochlorine insecticides on European birdlife. Proc. Int. Ornith. Congr. 15:486-513.

Prouty, R. M., J. E. Peterson, L. N. Locke, and B. M. Mulhern. 1975. DDD poisoning in a loon and the identification of the hydroxylated form of DDD. Bull. Environ. Contam. Toxicol. 14(4):385-389.

Ratcliffe, D. A. 1967. The peregrine situation in Great Britain. Bird Study 14(4):238-246.

Ratcliffe, D. A. 1972. The peregrine population of Great Britain in 1971. Bird Study 19:117-157.

Redner, J. A., and K. Payne. 1971. Chlorinated hydrocarbons. Progress Report, December 1971. County Sanitation Districts of Los Angeles County, 2020 Beverly Boulevard, Los Angeles.

Reichel, W. L., R. M. Prouty, and M. L. Gay. 1977. Identification of polychlorinated styrene compounds in heron tissues by gas chromatograph-mass spectrometry. J. Assoc. Off. Anal. Chem. 60(1):60-62.

Risebrough, R. W. 1972. Effects of environmental pollutants upon animals other than man. Pages 443-463 *in* L. Le Cam, J. Neuman, and E. L. Scott, eds. Proceedings of the 6th Berkeley Symposium, Mathematical Statistics and Probability. University of California Press, Berkeley and Los Angeles. 599 pp.

Risebrough, R. W. 1977. Transfer of organochlorine pollutants to Antarctica. Pages 1203-1210 *in* G. A. Llano, ed. Adaptations within Antarctic Ecosystems: Proceedings of the 3rd Scientific Committee for Antarctic Research Symposium on Antarctic Biology. Gulf Publishing Co., Houston. 1252 pp.

Risebrough, R. W., and D. W. Anderson. 1975. Some effects of DDE and PCB on mallards and their eggs. J. Wildl. Manage. 39(3):508-513.

Risebrough, R. W., and D. D. Berger. 1971. Evidence for aerial fallout of polychlorinated biphenyls (PCB) in the eastern Canadian Arctic. Can. Wildl. Serv. Manuscr. Rep. 23.

Risebrough, R. W., and G. M. Carmignani. 1972. Chlorinated hydrocarbons in Antarctic birds. Pages 63-78 *in* B. C. Parker, ed. Proceedings of the Colloquium, Conservation Problems in Antarctica. Allen Press, Lawrence, Kansas.

Risebrough, R. W., and B. W. de Lappe. 1972. Accumulation of polychlorinated biphenyls in ecosystems. Environ. Health Perspect. 1:39-45.

Risebrough, R. W., B. W. de Lappe, and W. Walker, II. 1976a. Transfer of higher molecular weight chlorinated hydrocarbons to the marine environment. Pages 261-321 *in* H. L. Windom and R. A. Duce, eds. Marine Pollutant Transfer. D. C. Heath and Company, Lexington, Massachusetts. 391 pp.

Risebrough, R. W., D. B. Menzel, D. J. Martin, and H. S. Olcott. 1967. DDT residues in Pacific sea birds: a persistent insecticide in marine food chains. Nature 216(5115):589-591.

Risebrough, R. W., P. Reiche, D. B. Peakall, S. G. Herman, and M. N. Kirven. 1968. Polychlorinated biphenyls in the global ecosystem. Nature 220(5172):1098-1102.

Risebrough, R. W., F. C. Sibley, and M. N. Kirven. 1971. Reproductive failure of the brown pelican on Anacapa Island in 1969. Am. Birds 25(1):8-9.

Risebrough, R. W., W. Walker, II, T. Schmidt, B. W. de Lappe, and C. W. Connors. 1976b. Transfer of chlorinated biphenyls to Antarctica. Nature 264:738-739.

Rittinghaus, H. 1956. Etwas uber die "indirekte" verbreitung der olpest in einem seevogelschutzgebiet [On the "indirect" spread of oil pollution in a seabird sanctuary]. Ornithol. Mitt. 8(3):43-46.

Robinson, J., A. Richardson, A. N. Crabtree, J. C. Coulson, and G. R. Potts. 1967. Organochlorine residues in marine organisms. Nature 214:1307-1311.

Rothstein, S. I. 1973. Plastic particle pollution of the surface of the Atlantic Ocean: evidence from a seabird. Condor 75(3):344-366.

Rozman, R. S., L. N. Locke, and S. F. McClure, III. 1974. Enzyme changes in mallard ducks fed iron or lead shot. Avian Dis. 18(3):435-445.

Schreiber, R. W., and R. W. Risebrough. 1972. Studies of the brown pelican. Wilson Bull. 84(2):119-135.

Sladen, W. J. L., C. M. Menzie, and W. L. Reichel. 1966. DDT residues in Adelie penguins and a crabeater seal from Antarctica. Nature 210(5037):670-673.

Smith, D. C. 1975. Rehabilitating oiled aquatic birds. Pages 241-247 *in* Proceedings of the 1975 Conference on Prevention and Control of Oil Spills. American Petroleum Institute, Washington, D.C.

Smithsonian Institution Center for Short-lived Phenomena. 1974. Metula oil spill. Event 121-74, Cards 1935-1936.

Snyder, S. B., J. G. Fox, and O. A. Soave. 1973. Mortalities in waterfowl following bunker C fuel exposure. Stanford Medical Center, Stanford, California. 27 + xxi pp.

Spann, J. W., R. G. Heath, J. F. Kreitzer, and L. N. Locke. 1972. Ethyl mercury p-toluene sulfonanilide: lethal and reproductive effects on pheasants. Science 175(4019):328-331.

Spitzer, P. R., R. W. Risebrough, J. W. Grier, and C. R. Sindelar, Jr. 1977. Eggshell thickness-pollutant relationships among North American ospreys. Pages 13-19 *in* J. Ogden, ed. Transactions of the North American Osprey Research Conference, College of William and Mary, Williamsburg, Va., 10-12 February, 1972.

Stegeman, J. J., and J. M. Teal. 1973. Accumulation, release, and retention of petroleum hydrocarbons by the oyster *Crassostrea virginica*. Mar. Biol. 22:37-44.

Stendell, R. C., H. M. Ohlendorf, and E. E. Klaas. 1976. Mercury in eggs of aquatic birds, Lake St. Clair — 1973. Pestic. Monit. J. 10(1):7-9.

Stickel, L. F. 1972. Biological data on PCBs in animals other than man. Pages 158-172 *in* Polychlorinated biphenyls and the environment by Interdepartmental Task Force on PCBs, Washington, May 1972. NTIS [Natl. Tech. Inf. Serv.] No. COM-72-10419.

Stickel, L. F. 1973. Pesticide residues in birds and mammals. Pages 254-312 *in* C. A. Edwards, ed. Environmental pollution by pesticides. Plenum Press, London and New York. x + 542 pp.

Stickel, L. F., and W. H. Stickel. 1970. Distribution of DDT residues in tissues of birds in relation to mortality, body condition, and time. Pages 57-65 *in* W. B. Deichmann, ed. Pesticides symposia. Helios and Associates, Miami.

Stickel, L. F., W. H. Stickel, and R. Christensen. 1966. Residues of DDT in brains and bodies of birds that died on dosage and in survivors. Science 151(3717):1549-1551.

Stickel, L. F., S. N. Wiemeyer, and L. J. Blus. 1973. Pesticide residues in eggs of wild birds: adjustment for loss of moisture and lipid. Bull. Environ. Contam. Toxicol. 9(4):193-196.

Stickel, W. H. 1971. Ecological effects of methylmercury contamination. Environ. Res. 4(1):31-41.

Stickel, W. H. 1975. Some effects of pollutants in terrestrial ecosystems. Pages 25-74 *in* A. D. McIntyre and C. F. Mills, eds. Ecological toxicology research — effects of heavy metal and organohalogen compounds. Plenum Press, New York. 323 pp.

Stickel, W. H., J. A. Galyen, R. A. Dyrland, and D. L. Hughes. 1973. Toxicity and persistence of mirex in birds. Pages 437-467 *in* Pesticides and the environment: a continuing controversy. Inter-American Conference on Toxicology and Occupational Medicine.

Stickel, W. H., L. F. Stickel, and F. B. Coon. 1970. DDE and DDD residues correlated with mortality of experimental birds. Pages 287-294 *in* W. B. Deichmann, ed. Pesticides symposia. Helios and Associates, Miami.

Stickel, W. H., L. F. Stickel, and J. W. Spann. 1969. Tissue residues of dieldrin in relation to mortality in birds and mammals. Pages 174-204 *in* M. W. Miller and G. G. Berg, eds. Chemical fallout; current research on persistent pesticides: Charles C. Thomas, Springfield, Illinois. xxii + 531 pp.

Swedmark, M., A. Granmo, and S. Kollberg. 1973. Effects of oil dispersants and oil emulsions on marine animals. Water Res. 7(11):1649-1672.

Szaro, R. C. 1977. Effects of petroleum on birds. Trans. N. Am. Wildl. Nat. Resour. Conf. 42:374-381.

Tanis, J. J. C., and M. F. Morzer Bruyns. 1968. The impact of oil pollution on sea birds in Europe. Pages 67-74 *in* International Conference on Oil Pollution of the Sea, October 7-9, 1968. Rome.

Tarzwell, C. M. 1970. Toxicity of oil and oil dispersant mixtures to aquatic life. Pages 263-272 *in* Transactions of the International Seminar Water Pollution by Oil, May 4-8, 1970. Aviemore, Scotland.

Tatton, J. O'G., and J. H. A. Ruzicka. 1967. Organochlorine pesticides in Antarctica. Nature 215(5099):346-348.

Ten Noever de Brauw, M. C., and J. H. Koeman. 1972. Identification of chlorinated styrenes in cormorant tissues by a computerized gas chromatography mass spectrometry system. Sci. Total Environ. 1(4):427-432.

Ten Noever de Brauw, M. C., C. Van Ingen, and J. H. Koeman. 1973. Mirex in seals. Sci. Total Environ. 2(2):196-198.

Tuck, L. M. 1960. The murres: their distribution, populations and biology, a study of the genus *Uria*. Can. Wildl. Serv. No. 1.

Tuck, L. M., and J. A. Livingston. 1959. Oil pollution in Newfoundland. Pages 76-79 *in* Proceedings of the International Conference on Oil Pollution of the Sea, July 3-4, 1959. Copenhagen.

Tucker, R. K., and M. A. Haegele. 1970. Eggshell thinning as influenced by method of DDT exposure. Bull. Environ. Contam. Toxicol. 5(3):191-194.

Tumasonis, C. F., B. Bush, and F. D. Baker. 1973. PCB levels in egg yolks associated with embryonic mortality and deformity of hatched chicks. Arch. Environ. Contam. Toxicol. 1(4):312-324.

U.S. Department of the Interior. 1970. Kodiak oil pollution incident, February-March 1970: Summary Report, Federal Water Quality Administration, Washington, D.C. 23 pp.

Vermeer, K. 1971a. A survey of mercury residues in aquatic bird eggs in the Canadian prairie provinces. Trans. N. Am. Wildl. Nat. Resour. Conf. 36:138-152.

Vermeer, K. 1971b. Spotted sandpipers as possible indicators of mercury contamination of rivers. Blue Jay 29(2):59-60.

Vermeer, K. 1972. The crayfish, *Orconectes virilis*, as an indicator of mercury contamination. Can. Field-Nat. 86(2):123-125.

Vermeer, K., and F. A. J. Armstrong. 1972a. Mercury in Canadian prairie ducks. J. Wildl. Manage. 36(1):179-182.

Vermeer, K., and F. A. J. Armstrong. 1972b. Correlation between mercury in wings and breast muscles in ducks. J. Wildl. Manage. 36(4):1270-1273.

Vermeer, K., F. A. J. Armstrong, and D. R. M. Hatch. 1973. Mercury in aquatic birds at Clay Lake, Western Ontario, J. Wildl. Manage. 37(1):58-61.

Vermeer, K., and L. M. Reynolds. 1970. Organochlorine residues in aquatic birds in the Canadian prairie provinces. Can. Field-Nat. 84(2):117-130.

Vermeer, K., and R. W. Risebrough. 1972. Additional information on egg shell thickness in relation to DDE concentrations in great blue heron eggs. Can. Field-Nat. 86(4):384-385.

Vermeer, K., and R. Vermeer. 1975. Oil threat to birds on the Canadian west coast. Can. Field-Nat. 89(3):278-298.

Vermeer, R., and K. Vermeer. 1974a. Oil pollution of birds — an abstracted bibliography. Can. Wildl. Serv. Manuscr. Rep. 29. 68 pp.

Vermeer, R., and K. Vermeer. 1974b. The biological effects of oil pollution on aquatic organisms: a summarized bibliography. Can. Wildl. Serv. Manuscr. Rep. 31. 68 pp.

Vos, J. G., and J. H. Koeman. 1970. Comparative toxicologic study with polychlorinated biphenyls in chickens with species reference to porphyria, edema formation, liver necrosis and tissue residues. Toxicol. Appl. Pharmacol. 17(3):656-668.

Vos, J. G., J. H. Koeman, H. L. Van der Maas, M. C. Ten Noever de Brauw, and R. H. de Vos. 1970. Identification and toxicological evaluation of chlorinated dibenzofuran and chlorinated naphthalene in two commercial polychlorinated biphenyls. Food Cosmet. Toxicol. 8(6):625-633.

Walker, W., II. 1977. Chlorinated hydrocarbon pollutants in Alaskan gyrfalcons and their prey. Auk 94(3):442-447.

Walker, W., II, R. W. Risebrough, J. T. Mendola, and G. W. Bowes. 1973. South American studies of the peregrine, an indicator species for persistent pollutants. Antarct. J. U. S. 8:29-31.

Westoo, G. 1967. Determination of methylmercury compounds in foodstuffs. II. Determination of methylmercury in fish, egg, meat and liver. Acta Chem. Scand. 21:1790-1800.

Wetmore, A. 1919. Lead poisoning in waterfowl. U.S. Dep. Agric. Bull. 793. 12 pp.

Whisman, M. L., and F. O. Cotton. 1971. Bumines data promise help in identifying petroleum-spill sources. Pages 111-113 *in* Oil and Gas Journal, December 27, 1971.

White, C. M., and R. W. Risebrough. 1977. Polychlorinated biphenyls in the ecosystems. Pages 615-625 *in* M. L. Merritt and R. G. Fullers, eds. The environment of Amchitka Island, Alaska, Atomic Energy Research and Development Administration; Technical Information Service, TID-26712.

White, D. H., and T. E. Kaiser. 1976. Residues of organochlorines and heavy metals in ruddy ducks from the Delaware River, 1973. Pestic. Monit. J. 9(4):155-156.

White, D. H., and L. F. Stickel. 1975. Impacts of chemicals on waterfowl reproduction and survival. Trans. Waterfowl Symp. 1:132-142.

Wiemeyer, S. N. 1971. Reproductive success of Potomac River ospreys — 1970. Chesapeake Sci. 12(4):278-280.

Wiemeyer, S. N., B. M. Mulhern, F. J. Ligas, R. J. Hensel, J. E. Mathisen, F. C. Robards, and S. Postupalsky. 1972. Residues of organochlorine pesticides, polychlorinated biphenyls, and mercury in bald eagle eggs and changes in shell thickness — 1969 and 1970. Pestic. Monit. J. 6(1):50-55.

Wiemeyer, S. N., and R. D. Porter. 1970. DDE thins eggshells of captive American kestrels. Nature 227(5259):737-738.

Wiemeyer, S. N., P. R. Spitzer, W. C. Krantz, T. G. Lamont, and E. Cromartie. 1975. Effects of environmental pollutants on Connecticut and Maryland ospreys. J. Wildl. Manage. 39(1):124-139.

Wurster, C. F., Jr., and D. B. Wingate. 1968. DDT residues and declining reproduction in the Bermuda petrel. Science 159(3818):979-981.

Zitko, V., and P. M. K. Choi. 1972. PCB and *p,p'*-DDE in eggs of cormorants, gulls, and ducks from the Bay of Fundy, Canada. Bull. Environ. Contam. Toxicol. 7(1):63-64.

Zitko, V., O. Hutzinger, and P. M. K. Choi. 1972. Contamination of the Bay of Fundy — Gulf of Maine area with polychlorinated biphenyls, polychlorinated terphenyls, chlorinated dibenzodioxins, and dibenzofurans. Environ. Health Perspect. 1:47-50.

ZoBell, C. E. 1962. The occurrence, effects, and fate of oil polluting the sea. Pages 85-118 *in* International Conference on Water Pollution Research, September 1962. London.

ZoBell, C. E. 1969. Microbial modification of crude oil in the sea. Pages 317-326 *in* Proceedings of the Joint Conference on Prevention and Control of Oil Spills, 1969. American Petroleum Institute, Washington, D.C.

GPO 845-756

As the Nation's principal conservation agency, the Department of the Interior has responsibility for most of our nationally owned public lands and natural resources. This includes fostering the wisest use of our land and water resources, protecting our fish and wildlife, preserving the environmental and cultural values of our national parks and historical places, and providing for the enjoyment of life through outdoor recreation. The Department assesses our energy and mineral resources and works to assure that their development is in the best interests of all our people. The Department also has a major responsibility for American Indian reservation communities and for people who live in island territories under U.S. administration.

UNITED STATES
DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE
EDITORIAL OFFICE
AYLESWORTH HALL, CSU
FORT COLLINS, COLORADO 80523

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF THE INTERIOR
INT 423

NOTE: Mailing lists are computerized. Please return address label with change of address.