Reprinted from: Transactions of the 42nd North American Wildlife and Natural Resources Conference, 1977
Published by the Wildlife Management Institute, Washington, D.C.

Effects of Petroleum on Birds

Robert C. Szaro
U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, Maryland

Introduction

Oil pollution is in the forefront of public attention because of the recent series of oil spills from tankers such as the *Argo Merchant*. Oil spillage frequently destroys the marine flora and fauna; the most striking single effect is the oiling of large numbers of marine birds. For example, as many as 30,000 birds died after the grounding of the *Torrey Canyon* at Seven Stones Reef, Great Britain in 1967 (Bourne, Parrack and Potts 1967). Moreover, between 1967 and 1971 at least 250,000 seabirds died worldwide as a direct result of oil spillage (Clark 1973). More recent oil spills have had equally devastating effects on marine birds.

Oil spillage resulting from the transportation of oil accounts for only 35 percent of the 6.1 million metric tons of petroleum hydrocarbons introduced into the oceans each year (Wilson and Hunt 1975). Most of the oil in the environment results from the countless discharges of petroleum occurring during normal usage. Most of these discharges are small enough to go uncataloged, but their global impact may in the long run be determined by the total amount of oil rather than by the size of the individual spills (Storrs 1973). Thus, the overall effect of oil pollution on aquatic bird populations must be examined from two points of view: (1) the disastrous effects of oil spills and (2) the sublethal and indirect effects of chronic exposure to low levels of petroleum hydrocarbons in the environment.

Bird Oiling

Seabird mortality resulting from oil spillage has been reviewed by Bourne (1968), Clark (1973), and Vermeer and Vermeer (1975). These reviews emphasize the high susceptibility of certain species of birds to oil pollution. Of all aquatic birds, alcids, diving ducks, and penguins appear to be the chief victims of oil pollution (Vermeer and Anweiler 1975; Vermeer and Vermeer 1975, Frost, Siegfried and Cooper 1976). The reasons why these birds constitute the most frequent and largest numbers of casualties is related to their presence in heavily trafficked sea lanes, their large numbers, the high proportion of time spent on the water, and their behavior. These birds dive while feeding so they become covered with oil when they break the surface in an oil slick. In contrast, gulls are much less vulnerable to oil slicks than diving birds because of their habit of flying over surface pollution and therefore avoiding contact (Bourne 1968). In an oil spill area in Nova Scotia, purple sandpipers (*Erolia maritima*) accumulated a thin coating of oil, but no sandpipers were observed flightless or dead (Smith and Bleakney 1968). Typically, wading birds are merely stained and not coated with oil (Vermeer and Vermeer 1975).

The main physical effects of oiling are loss of buoyancy and insulation. These effects result from the matting of the feathers, which destroys the waterproofing and insulating properties of the plumage (Hartung 1967). When the feather barbules are oiled, they adhere to each other and allow water both to saturate the outer contour feathers and to penetrate down feathers (McEwan and Koelink 1973). Heavily oiled and watersoaked birds frequently stop feeding and go to shore, where they are isolated from their food supply (Erickson 1963). Many of the birds never make it to shore, but drown on the open ocean because they lose buoyancy (Vermeer and Vermeer 1975). Heavily oiled mallards (*Anas platyrhynchos*) and scaup (*Aythya affinis*) lost almost twice as much heat as did control birds (Hartung 1967, McEwan and Koelink 1973). Thus a heavily oiled duck would need to almost double its dietary intake in order to make up for heat losses, but intake is usually reduced rather than increased and oiled ducks die rapidly.

Chemical Analysis

The analysis of animal tissues for oil is difficult because of the complex nature of oil and the presence of naturally occurring hydrocarbons in animal tissues. Petroleum has been identified by gas chromatography in a composite sample of liver, kidney, fat, heart and brain of an oil-exposed common murre (*Uria aalge*) in the liver and kidney of an oil-soaked surf scoter (*Melanitta perspicillata*) and in the liver of a Western grebe (*Aechemophorous occidentalis*) (Snyder, Fox, and Soave 1973). However, the analytical methodology for detecting and quantitating petroleum hydrocarbons in avian tissues is in the early developmental stages and standardization of techniques has not been accomplished. Petroleum hydrocarbons have been detected in various organs of oil-dosed mallards.* The quantitation of specific hydrocarbons in liver and muscle tissue has been achieved but the analysis of complex hydrocarbon mixtures such as crude oil and refined petroleum products is still in the qualitative stages.**

Systemic Effects of Oil Ingestion

Oil ingestion has been implicated in the high mortality of seabirds (Hartung and Hunt 1966; Snyder, Fox, and Soave 1973; Vermeer and Vermeer 1975). Hartung (1963) demonstrated that oiled ducks ingest oil from their feathers. He oiled the breast feathers with a suspension of lampblack in mineral oil, and after 6 hours of preening, the duck's bill, tongue, and throat were black, and the lining of its intestinal tract showed traces of carbon particles. He found the same result by another method in which the breast feathers of three black ducks (*Anas rubripes*) were contaminated with oil containing radiolabeled iodine (Hartung 1963). Within 24–36 hours the feces exhibited high levels of radiation, up to 12 times normal background levels. About 50 percent of the oil was preened off within eight days; the amount ingested decreased logrithmically from the first day.

Under natural conditions ducks can readily acquire 7 g or more of oil on their plumage (Hartung 1964). A coating of 7 g should result in the ingestion of approximately 1.5 g of oil during the first day (Hartung and Hunt 1966).

A single dose of 1.2 ml of No. 2 fuel oil did not harm canvasbacks (*Aythya valisineria*) (Hunt 1961). At a higher dosage of 2.4 ml, the body weights of the control and treated ducks did not differ significantly after a period of 31 days, but reaction time to stimuli slowed and muscular coordination deteriorated. Hartung

*Laseter 1976: personal communication.
**Gay and Belisle 1976: personal communication.

and Hunt (1966) tested a number of industrial oils for their toxic effects on waterfowl. All the oils (including No. 1 fuel oil, diesel oil, motor oil, and cutting oil) caused lipid pneumonia, gastrointestinal irritation, fatty livers and enlargement of the adrenal glands when fed to ducks in single doses as low as 1 ml/kg; effects were more common at dose levels about 2 ml/kg. The pancreas degenerated in those ducks fed cutting oil and diesel oil. Diesel oil and fuel oil produced toxic nephrosis of the kidneys.

Coastal and pelagic species of birds compensate for osmotic water loss when they are exposed to salt water by an increase in the rate of water uptake by the small intestine and by an increase of the excretions from the paired nasal glands (Bradley and Holmes 1972; Crocker and Holmes 1971a). The absorption of water and ions at an increased rate is essential to trigger the development of the nasal gland (Crocker and Holmes 1971b). If the absorption of ingested seawater is impaired, the nasal glands will also be impaired and the bird will become dehydrated.

Pekin ducklings (*Anas platyrhynchos*) given a single oral dose of 0.2 ml of crude oil at the same time that their drinking water was replaced by 60 percent seawater did not develop the characteristic increases in the uptake of water and sodium ions observed in control ducklings (Crocker, Cronshaw, and Holmes 1974). In addition, the increase in water uptake by the intestine that developed during prolonged exposure to salt water was eliminated after a single dose of Santa Barbara crude oil. The water soluble extracts of crude oils from San Joaquin Valley, California and Paradox Basin, Utah also inhibited water and ion uptake by the intestine (Crocker, Cronshaw, and Holmes 1975). It seems probable that dehydration may cause the death of many oil contaminated birds.

The effects of oil on aquatic birds is the subject of current research at the U. S. Fish and Wildlife Service's Patuxent Wildlife Research Center, Laurel, Maryland. Much of the previous work on this subject has dealt with the rehabilitation of oiled birds with very little attention given to the effects of chronic low level oil pollution or to the less easily observed effects of oil spills. Birds may be affected by oil directly, through feather oiling, by exposure of eggs to oiled feathers, and by ingestion of oil. They may be affected indirectly through changes in habitat and food supply and by exposure to oil through the food chain.

The overall objective of our work is to evaluate the effect of sublethal levels of oil to birds through correlated physiological, toxicological, and ecological investigations. Specific objectives include the evaluation of the effects of ingested oil on survival and reproduction, the effects of oil films on viability of eggs, the assessment of the prevalence and degree of oil ingestion by wild birds, the joint effects of oil and toxic chemicals on survival and reproduction, and the relationship between tissue levels of oil products and physiological and ecological damage. Without this knowledge it would not only be impossible to predict the impact of oil pollution on aquatic bird populations but would leave us without a firm footing on which to base future recommendations.

Oil ingestion should have its most dramatic effects during the critical period when ducklings are growing rapidly. Mallard ducklings that were reared for 8 weeks on a diet containing up to 50,000 ppm of South Louisiana crude oil (SLC) survived as well as controls (Szaro, unpublished data). The ducklings that received this high dosage, however, weighed an average of 200 g less than the controls (Table 1). Their livers were double normal size whereas their spleens

Table 1. Body and organ weights of ducklings reared for 8 weeks on a diet containing South Louisiana crude oil.

Treatment	Mean body[a] weight (g)	Liver[b] Mean weight (g)	Liver[b] Mean percentage of body weight	Spleen[b] Mean weight (g)	Spleen[b] Mean percentage of body weight
Control	1118.5	34.5	3.2	0.87	.074
250 ppm	1126.5	33.0	3.0	0.74	.067
2500 ppm	1108.5	31.5	2.9	0.64	.058
25,000 ppm	1069.1	51.4[c]	4.8	0.39[c]	.037
50,000 ppm	913.4[c]	69.7[c]	7.4	0.30[c]	.032

[a] $n = 50$.
[b] $n = 10$.
[c] $P < 0.05$

were less than half normal size. The enlargement of the liver indicates increased liver function since the liver is the site of removal of the toxic compounds present in oil. Moreover, feather development was retarded in the birds given high dosage levels.

A study has been initiated to examine the sublethal effects of the chronic ingestion of petroleum hydrocarbons in liver function in the mallard duck. These ducks are being fed a reconstituted aromatic mixture containing representative aromatic hydrocarbons found in South Louisiana crude oil at 400 and 4000 ppm. Liver function is being determined by the indocyanine green clearance technique. This technique is an accurate reproducible method of measuring removal of the dye by the liver. After 3 months and 5 months on the diet, the 4000 ppm group showed significant changes in liver function as evidenced by an increase in the disappearance rate of the dye. No changes were seen in plasma enzymes, hemoglobin, or total blood protein. The data suggest that the ingestion of high levels of aromatic hydrocarbons results in increased liver function due, probably, to cell enlargement. No cellular damage has yet been demonstrated (Patton, unpublished data).

Food chain studies are also being conducted to study the possible effects of natural uptake of petroleum hydrocarbons on aquatic birds. The presence of petroleum hydrocarbons has been demonstrated in several marine organisms ranging from sedentary diatoms and phytoplankton to zooplankton and oysters (Thompson and Eglinton 1976, Blumer, Gillard, and Chase 1971; Corner et al. 1976; Anderson 1975). However, investigations on the passage of petroleum hydrocarbons to the higher trophic levels are virtually nonexistent. Crayfish (*Procambrus* spp.) have proven a suitable food item for waterfowl and are currently being exposed to a water soluble fraction of oil. This mixture will be radioactively labeled to trace its bioaccumulation. These labeled crayfish will then be fed to adult mallards.*

Crude oils also contain high concentrations of metals. The Committee on Biologic Effects of Atmospheric Pollutants (1974) has reported that different types of crude oils contain as high as 1400 ppm vanadium. This crude oil component was found to significantly alter lipid metabolism in mallard hens (White and Dieter, unpublished data). Normal cholesterol concentrations in blood of nonlaying hens averaged 11.9 mg/l compared to 3.8 mg/l in laying hens. However, in laying hens

*Tarshis 1976: personal communication.

fed 100 ppm vanadyl sulfate, the average cholesterol concentration was not different than that in nonlaying hens, averaging 9.1 mg/l. Similar alterations in lipid metabolism occurred in a pilot study of mallard hens fed 10,000 ppm crude oil.

Reproductive Effects of Oil

Oil may affect aquatic birds by decreasing their reproductive potential. Mallard and pekin ducks fed 2 g/kg body weight of a relatively nontoxic lubricating oil stopped laying eggs for 2 weeks (Hartung 1965). Mallards fed a diet containing 25,000 ppm SLC laid significantly ($P \leq 0.05$) fewer eggs than controls. The ducks fed diets containing oil laid an average of 11.0 eggs whereas the control birds laid an average of 24.6 eggs during a 30-day period. Mallards fed diets containing 10,000 ppm paraffins and a 2500 ppm oil diet did not lay significantly fewer eggs than the controls (Coon, unpublished data).

Nesting seabirds would be particularly vulnerable to petroleum pollution because many of the species, particularly the alcids, have a low reproductive potential and are heavily concentrated on the nesting grounds (Vermeer and Vermeer 1975; Vermeer 1976). Oil could present a serious hazard to these birds through egg contamination. Female Sandwich terns (*Sterna sandvicensis*) and other shorebirds contaminated with oil that had been washed ashore have been observed returning to their nests and transferring oil to their eggs (Rittinghaus 1956). These eggs failed to hatch even after 50 days of incubation. Gull and cormorant populations have been successfully controlled by spraying an oil and formalin mixture on their eggs (Gross 1950). Diesel fuel sprayed on 57 pheasant (*Phasianus colchicus*) eggs decreased their hatchability to zero; 44 percent of the 57 control eggs hatched (Kopischke 1972). Very small quantities of oil coated on mallard eggs was sufficient to reduce their hatchability by 68 percent (Hartung 1965). These experimental studies however, failed to account for possible deaths due to the toxic nature of the oil rather than to the blockage of air transfer through the shell.

Small drops of either crude or refined oil applied to artificially incubated mallard eggs produced significant mortality (Table 2) (Szaro, Albers, and Coon, unpublished data). The hatchability of common eider eggs was reduced from 96 percent to 69 percent with the application of 20 μ 1 of No. 2 fuel oil (Szaro and Albers, in press). It was evident that embryonic mortality was caused by the toxic nature of the oil rather than by the blockage of normal gas exchange, because eggs treated

Table 2. Hatching success of mallard eggs treated with oil.

Treatment	South Louisiana crude oil (% hatch)	Kuwait crude oil (% hatch)	No. 2 fuel oil (% hatch)
Control[a]	92	92	88
1 μ 1	62[b]	72[b]	64[b]
5 μ 1	2[b]	24[b]	18[b]
10 μ 1	2[b]	16[b]	10[b]
20 μ 1	0[b]	6[b]	0[b]

[a] $n = 50$.
[b] $P < 0.05$.

Table 3. Hatching success of mallard eggs treated with 5 microliters of Southern Louisiana crude oil or No. 2 fuel oil at different stages of development.

Age of embryo at treatment (days)	South Louisiana crude oil (% hatch)	No. 2 fuel oil (% hatch)
Control[a]	100	80
2	0[b]	13[b]
6	3[b]	33[b]
10	8[b]	68[b]
14	78[b]	83
18	88[b]	80
22	95	93

[a] $n = 50$.
[b] $P < 0.05$.

with propylene glycol in even greater amounts exhibited normal hatching success. Albers (in press) showed that 50 μ 1 of propylene glycol covered approximately the same surface area of a mallard egg as 10 μ 1 of No. 2 fuel oil. The hatchability of those mallard eggs treated with propylene glycol was 80 percent as compared to 88 percent for the control. In later studies, propylene glycol had no effect on the hatchability of either mallard or common eider eggs (Szaro, Albers, and Coon, unpublished data; Szaro and Albers, in press). The hatching weights of the ducklings from oiled and control eggs were not significantly different ($P \leq 0.05$) in any of these studies.

The survival of oil treated eider embryos depends on age at treatment (Szaro and Albers, in press). Embryos that died after treatment, with 20 μ 1 of No. 2 fuel oil averaged 4.3 days of age at treatment, whereas surviving embryos were treated at an average age of 16.1 days. Albers (unpublished data) found that the hatchability of mallard eggs treated with petroleum increased as the age of the embryo at treatment increased (Table 3).

Conclusions

The chronic effects of oil pollution on aquatic birds are not well known. Our preliminary studies indicate that oil ingestion is probably not a major cause of seabird mortality. Oil ingestion may affect the physiological and reproductive condition of seabirds. Moreover, a substantial number of seabird eggs may be destroyed each year by oil contamination. Such laboratory studies now need to be extended to the field. Further laboratory studies are needed to measure the accumulation and persistence of oil in tissues and to interpret the significance of oil residues in tissues.

Acknowledgments

I thank the following people for generously allowing me to present their findings: Peter Albers, Andre Belisle, Nancy Coon, Michael Dieter, Martha Gay, John Patton, and Barry Tarshis.

Literature Cited

Albers, P. H. In press. Effects of external applications of fuel oil on hatchability of mallard eggs. *In* D. A. Wolfe, ed. Proceedings of the Symposium on Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. November 1976, Seattle, EPA/NOAA.

Anderson, R. D. 1975. Petroleum hydrocarbons and oyster resources of Galveston Bay, Texas. Pages 541–548 *in* Proceedings of the Joint Conference on Prevention and Control of Oil Spills. March 1975, San Francisco, American Petroleum Institute.

Blumer, M., R. R. L. Gillard and T. Chase. 1971. Hydrocarbons of marine phytoplankton. Mar. Biol. 8: 183–189.

Bourne, W. R. P. 1968. Oil pollution and bird populations. Pages 99–121 *in* J. D. Carthy and D. R. Arthur, eds. The biological effects of oil pollution on littoral communities. Field Stud. 2 (Suppl.)

______, J. D. Parrack, and G. R. Potts. 1967. Birds killed in the Torrey Canyon disaster. Nature (London) 215: 1123–1125.

Bradley, E. L. and W. N. Holmes. 1972. The role of the nasal glands in the survival of ducks (*Anas platyrhynchos*) exposed to hypertonic drinking water. Can. J. Zool. 50: 611–617.

Clark, R. B. 1973. Impact of chronic and acute oil pollution of seabirds. Pages 619–634 *in* Background papers for a workshop on inputs, fates, and effects of petroleum in the marine environment. Ocean Affairs Board, Nat. Acad. Sci., Washington, D. C.

Committee on Biological Effects of Atmospheric Pollutants. 1974. Pages 24–31 *in* Medical and biologic effects of environmental pollutants. Nat. Acad. Sci., Washington, D. C.

Corner, E. D. S., R. P. Harris, C. C. Kilvington and S. C. M. O'Hara. 1976. Petroleum compounds in the marine food web: short-term experiments on the fate of naphthalene in *Calanus*. J. Mar. Biol. Assoc. U. K. 56: 121–133.

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: 165–177.

______. 1975. The effect of several crude oils and some petroleum distillation fractions on intestinal absorption in ducklings (*Anas platyrhynchos*). Environ. Physiol. Biochem. 5: 92–106.

Crocker, A. D. and W. N. Holmes. 1971a. Intestinal absorption in ducklings (*Anas platyrhynchos*) maintained on freshwater and hypertonic saline. Comp. Biochem. Physiol: 40A: 203–211.

______. 1971b. Intestinal absorption in the duck (*Anas platyrhynchos*) maintained on freshwater and hypertonic saline. J. Endocrinol. 49: 18–29.

Erickson, R. C. 1963. Oil pollution and migratory birds. Atlantic Natur. 18: 5–14.

Frost, P. G. H., W. R. Siegfried, and J. Cooper. 1976. Conservation of the Jackass Penguin (*Spheniscus demersus* (L.)). Biol. Conserv. 9: 79–100.

Gross, A. O. 1950. The herring gull-cormorant control project. Proceedings of the International Ornithological Congress 10: 532–536.

Hartung R. 1963. Ingestion of oil by waterfowl. Papers of the Michigan Academy of Sciences, Arts and Letters 48: 49–55.

______. 1964. Some effects of oils on waterfowl. Ph.D. Thesis. University of Michigan. Ann Arbor, 190 p.

______. 1965. Some effects of oiling on reproduction of ducks. J. Wildl. Manage. 29: 872–874.

______. 1967. Energy metabolism in oil-covered ducks. J. Wildl. Manage. 31: 798–804.

______ and G. S. Hunt. 1966. Toxicity of some oils to waterfowl. J. Wildl. Manage. 30: 564–570.

Hunt, G. S. 1961. Waterfowl losses on the lower Detroit river due to oil pollution. Great Lakes Res. Div., Inst. Sci. and Tech., Univ. Michigan 7: 10–26.

Kopischke, E. D. 1972. The effect of 2,4-D and diesel fuel on egg hatchability. J. Wildl. Manage. 36: 1353–1355.

McEwan, E. H. and A. F. C. Koelink. 1973. The heat production of oiled mallards and scaup. Can. J. Zool. 51: 27–31.

Rittinghaus, H. 1956. On the indirect spread of oil pollution in a seabird sanctuary [in German]. Ornithologische Mitteilungen 8: 43–46.

Smith, P. C. and J. S. Bleakney. 1968. Observations on oil pollution and wintering purple sandpipers in Nova Scotia. Can. Field-Natur. 83: 19–20.

Snyder, S. B., J. G. Fox and D. A. Soave. 1973. Mortalities in waterfowl following bunker C fuel exposure. Division of Laboratory Animal Medicine, Stanford Medical Center, Stanford, 27 p.

Storrs, P. N. 1973. Petroleum inputs to the marine environment. Pages 50–58 *in* Background papers for a workshop on inputs, fates, and effects of petroleum in the marine environment. Ocean Affairs Board, Nat. Acad. Sci., Washington, D. C.

Szaro, R. C. and P. H. Albers. In press. Effects of external applications of No. 2 fuel oil on common eider eggs. *In* D. A. Wolfe, ed. Proceedings of the Symposium of Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. November, 1976, Seattle, EPA/NOAA.

Thompson, S. and G. Eglinton. 1976. The presence of pollutants hydrocarbons in estuarine epipelic diatom populations. Estuarine Coastal Mar. Sci. 4: 417–425.

Vermeer, K. 1976. Colonial auks and eiders as potential indicators of oil pollution. Mar. Pollut. Bull. 7: 165–167.

——— and G. G. Anweiler. 1975. Oil threat to aquatic birds along the Yukon coast. Wilson Bull. 87: 467–480.

Vermeer, K. and R. Vermeer. 1975. Oil threat to birds on the Canadian west coast. Can. Field-Natur. 89: 278–298.

Wilson, E. B., and J. M. Hunt, ed. 1975. Petroleum in the marine environment. Ocean Affairs Board, Nat. Acad. Sci., Washington, D. C. 107 p.