FOREWORD

Marine contamination by petroleum, whether by natural seepage or by spills from ships at sea, by accidents in harbour or at offshore installations or by atmospheric or terrigenous input is by no means a new or rare phenomenon. In recent years however, the problems have been highlighted not only by the increased utilisation and marine transport of oil but also by a number of spectacular accidents which have raised questions about possible effects on the ecosystem. A number of detailed studies have been carried out in an attempt to answer these questions. The demands for such knowledge have been further increased by the various questions raised as a result of expansion of offshore exploration and exploitation for oil, particularly in environments hostile to these operations, in regions as far apart as the northern North Sea and the coast of Alaska.

Consequently, diverse aspects of the problem are being studied in several parts of the world by chemists and biologists who are often asking the same questions but using different approaches and sometimes producing conflicting views. Against this background, it seemed timely therefore to bring together a group of scientists from university, industry and government, actively engaged in such work, to examine and discuss common problems relevant to petroleum hydrocarbon contamination of the marine ecosystem and so a Workshop was sponsored by the International Council for the Exploration of the Sea, and held in Scotland at Aberdeen in September 1975.

The Workshop considered methodology, occurrence and fate in the environment, and effects on the ecosystem of petroleum hydrocarbons in the sea. Most of the papers presented and updated where necessary, are brought together in the present volume together with an edited version of the recorded discussion that followed each session. Of necessity, the reportage of the discussion is very brief although the proportion of time available for discussion compared favourably with that set aside for formal presentation of the papers. In preparing the discussion reports, the editors were assisted in particular by Dr R. Hardy, Dr R. Johnston, Mr P. R. Mackie and Dr I. C. White, and by comments from several contributors.

No attempt was made to produce specific recommendations but a study of the papers in this volume does give a clear indication of several lines of research which must be followed up before an adequate understanding can be reached of the effects of petroleum in the sea and it is evident that widespread monitoring operations will be fully effective only when the basis of our knowledge has been thus extended.

A list of participants to the workshop may be found in Appendix I.

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THE EFFECTS OF OIL ON SEABIRDS

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Seabirds are not only the most obvious victims of oil pollution but perhaps the only group where oil pollution may pose a survival threat. The external effect of oil in destroying plumage waterproofing is detailed and the state of knowledge of the direct and indirect internal effects of crude and fuel oils reviewed, inevitably in rather general terms. The need for more precise information on the behaviour of oil slicks and oil/dispersant mixtures and films at sea is emphasised in relation to potential techniques and strategies for minimising the high risk to seabirds (whether at breeding colonies or on wintering grounds) from oil slicks.

INTRODUCTION

Although I wish principally to summarise what is known about the more specific external and internal effects of petroleum hydrocarbons on birds it is perhaps useful first briefly to put this in perspective by considering in more general terms the effect of oil on seabirds. A detailed treatment with full bibliography appears in Croxall (1975).

As might be expected the seabird species most frequently in contact with oil are those that congregate in flocks both at breeding colonies and in wintering grounds and especially those which frequent offshore waters and/or migrate near busy shipping lanes. Thus in the northern hemisphere auks Alcidae and certain diving seaduck (chiefly eider *Somateria mollissima,* scoter *Melanitta* spp. and long-tailed duck *Clangula hyemalis*) are principally affected. In the southern hemisphere penguins Spheniscidae and cormorants and shags *Phalacrocorax* spp. are the main victims.

It seems that most chronic oil pollution and large-scale oiling incidents occur in winter and early spring (see Table 66), presumably when environmental conditions are at their most difficult both for birds (ensuring that the effects of oiling are particularly severe) and shipping operations.

The scale of the annual mortality is very difficult to assess even within the British Isles and using the data resulting from the regular beached birds surveys organised by the Royal Society for Protection of Birds (see Table 67). There is particular need for caution in interpreting figures representing the number of birds found per km. Nevertheless the data do demonstrate that auks are the main casualties in British waters, and that there may have been a reduction in casualties (or fewer birds to remain at risk) following the widespread adoption of load-on-top measures in the early 1960's. There is, however, no indication of a trend to reduction in the number of birds killed over more recent years and an estimate of an annual European mortality of 150 000–450 000 birds is very unlikely to be pessimistic.

The significance of such mortality is also far from clear, chiefly because of the difficulties in obtaining reliable population estimates of the species mainly involved. This is notably so for auks, the group most affected by oil pollution. This is particularly serious as rapid numerical recovery is made impossible by the demographic structure of their population, resulting in

Table 66. Seasonal distribution of British oiled, beached Guillemots, 1970–71 and 1973–74

	Beach surveyed (km)		No. Corpses		% Annual total*	
	1970–71	1973–74	1970–71	1973–74	1970–71	1973–74
Jan.	163	1 858	94	595	23·0	37·5
Feb.	91	2 248	37	111	15·9	5·8
Mar.	121	1 808	50	102	16·3	6·6
Apr.	168	453	42	20	10·0	5·2
May	44	227	11	27	10·0	13·9
Jun.	48	141	2	3	1·5	2·5
Jul.	48	240	2	10	1·5	4·8
Aug.	39	218	0	6	0	3·2
Sep.	53	1 488	7	25	5·1	2·0
Oct.	53	1 128	6	43	4·4	4·5
Nov.	55	1 866	4	107	2·8	6·7
Dec.	59	790	14	49	9·5	7·3

* Corrected for length of beach surveyed.

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Table 67. Annual totals, British oiled beached seabirds

	1951–52	1952–53	1966–67	1968–69	1969–70	1970–71	1971–72	1972–73	1973–74
Divers/grebes	235	161	71	89	78	60	66	78	25
Gannet	–	–	60	9	46	29	40	53	47
Cormorant/shag	–	–	59	21	72	24	58	72	133
Wildfowl	–	–	229	80	772	105	99	104	271
Waders	–	–	73	16	194	18	18	14	249
Gulls/terns	4 700	331	1 141	353	290	303	462	311	529
Auk	1 065	402	1 417	1 878	2 646	620	1 315	837	1 559
Other	101	105	30	43	60	29	42	62	144
Total	6 722	1 408	3 080	2 511	4 158	1 188	2 100	1 489	2 957
Beach surveyed (km)	c220	c175	c600	2 839	4 009	4 605	9 826	11 942	12 517
Oiled birds/km	c30·6	c8·1	c5·1	0·89	1·04	0·26	0·21	0·13	0·24

particular from the delayed sexual maturity, small clutches and considerable natural loss of eggs and northern hemisphere seabird groups currently at risk from oil pollution.

Thus there is convincing, if circumstantial, evidence that oil was implicated at least in the decline of southern European auk colonies, particularly of puffins *Fratercula arctica* in the Scilly Isles (Cramp et al, 1974; Parslow, 1967, 1967a), common guillemots *Uria aalge* at Ailsa Craig (Fisher and Lockley, 1954), razorbill *Alca torda* on the Newfoundland coast (Giles and Livingston, 1960) and in the 90 % reduction in long-tailed duck migrating through Finland (Lemmetyinen, 1966). There is virtually direct evidence from the Sept Isles Brittany seabird colony (Monnat, 1969) that the Torrey Canyon mortality reduced the breeding pairs of guillemots by 81 %, razorbill by 89 % and puffin by 84 %, with gulls and gannet *Sula bassana* unaffected.

Auks are also faced with other environmental pressures, such as the loss of breeding sites to puffin through erosion, the presence of chemical pesticide residues (e.g. Parslow and Jefferies, 1973) and perhaps in some areas changes in the abundance and distribution of their main fish foods. Lack of ability to respond rapidly to this combination of threats means that the survival of small auk colonies (many in Britain) and those on the margin of a species range (chiefly S and SW Britain) is severely imperilled.

DIRECT EFFECTS OF OIL

External oil rapidly destroys the waterproof nature of the birds plumage while virtually simultaneously oil is ingested in an attempt to preen the contaminant off the feathers. Many of the subsequent internal conditions undoubtedly result from the effects of both external and internal oil but it is convenient here to try to distinguish them.

EXTERNAL OIL

External oil disrupts the regular precise arrangement of the fine structure of the feathers and allows water to penetrate the plumage. The resultant rapid loss of insulation necessitates use of energy reserves to maintain body temperature and it has been calculated (Hartung, 1967; McEwan and Koelink, 1973) that a heavily oiled bird uses these about twice as fast as unoiled birds. Once fat reserves have been metabolised (usually within 48—72 hours) the muscular energy reserves are mobilised. As oiling effectively prevents efficient feeding these reserves cannot be replaced and atrophy of the pectoral muscle and severe emaciation develop rapidly and chilling and starvation produce symptoms of physiological stress.

Effects usually ascribed to stress include:

a) reduction in white blood corpuscles, reducing resistance to infection;
b) disruption of fluids and electrolyte balance leading to dehydration;
c) disturbance of endocrine balance, usually associated with enlarged adrenals;
d) fatty degeneration of the liver, reducing ability to break down toxins;
e) kidney changes, restricting elimination of waste products.

INTERNAL OIL

With birds oiled naturally it is very difficult to distinguish between the effects of stress and those of ingested oil. Dosage of healthy birds with oil offers the best chance of evaluating the direct effects of ingested oil but it is impossible totally to exclude effects produced by stress resulting from the experimentation.

Although there is substantial anecdotal evidence of the internal effect of oil the only critical study along the above lines is that of Hartung and Hunt (1966).

Isotope studies (Hartung, 1963) had already demonstrated that moderately to heavily oiled ducks (weight 0·7 kg) preen 50 % of polluting oil off their plumage in 8 days and will ingest up to 7 g oil, in the process of which 1·5 g may be swallowed in the first day. Thus artificial dosage levels of 2—3 g/kg can be considered reasonable approximations to normal pollution conditions.

A variety of oils (light fuel, diesel, lubricating, cutting and mineral) were administered by stomach tube at varying concentrations (mainly 1—4 ml/kg) to healthy ducks and a number of analyses performed at autopsy to test for damage to the function of the pancreas (plasma lipase levels), liver (plasma glutamic oxalacetic transaminase levels), kidney (non-protein nitrogen concentration) and nerve impulse transmission (plasma cholinesterase levels). Unmedicated control birds were similarly tested.

The results, of which Table 68 is a summary, showed that all oils commonly caused lipid pneumonia and extreme, even haemorrhagic, inflammation of the lungs with 2 ml/kg dosage. Fuel and diesel oil produced severe irritation of the digestive tract (enteritis) with 1 ml/kg dosage, lubricating oils only a slight irritation (judged by the level of hyperemia of the digestive wall) and mineral oils not at all. The occurrence of diarrhoea and bile pigments in faeces was common to all oils. 2 ml/kg diesel oil produced a significant degree of fatty infiltration and liver degeneration after only 24 hours but lubricating oils had no marked effect in this time. All oils produced a reduction in pancreatic zymogen granules (taken as an index of the quantity of stored enzyme precursors). Lubricating and cutting oils caused only minor kidney changes but fuel and diesel oils gave rise to typical symptoms of toxic nephrosis; changes in non-protein nitrogen gave similar findings. Adrenal enlargement (hyperplasia of cortical tissue, medullary layer largely unaffected) was caused by 1 ml/kg of any oil, the changes being very similar to those described for Selye's general stress adaptation syndrome. Diesel and cutting oils significantly depressed plasma cholinesterase levels and uncoordination, ataxia and tremors were noted with higher doses, suggesting that the higher levels of organic phosphate in these oils inhibited acetyl cholinesterase activity and caused the observed changes in motor activity.

These findings are closely paralleled by the results of post-mortem examinations of naturally oiled birds (e.g. Beer, 1968; 1968a; Clark and Kennedy, 1968; Hartung and Hunt, 1966; Snyder et al, 1973), where enteritis, nephrosis, liver disfunction, adrenal enlargement and lipid pneumonia are the commonest ailments reported.

It should be noted that the oils used in the above experiments are not fatal (even, except for cutting oil, at doses of up to 20 ml/kg) to otherwise healthy birds. If administered to birds under stress (e.g. crowded together at sub-zero temperatures), however, the LD50 for cutting oil is 1 ml/kg and for fuel and diesel oil 3—4 ml/kg.

Table 68. Effect of dosage of 2–3 ml/kg of various oils in certain bird organ systems (summarising Hartung and Hunt, 1966)

Oil	Fuel	Diesel	Cutting	Lubricating	Mineral
Effect on:					
Lungs	+ +	+ +	+ +	+ +	+ +
Adrenals	+ +	+ +	+ +	+ +	+ +
Pancreas	+ +	+ +	+ +	+ +	+ +
Kidney	+ + +	+ + +	+	+	?
Gastro-intestinal tract	+ + +	+ + +	?	+	0
Nervous system	?	+	+ +	?	?
Liver	?	+ +	?	0	?

+ + + = strong reaction, + + = significant reaction, + = weak reaction, 0 = no reaction, ? = no information.

I know of only two other relevant experimental investigations. Holmes (1975) and Crocker et al (1974) demonstrated that nasal gland action (maintaining osmotic balance of birds living in hyperosmotic environments) is normally stimulated by reabsorption of hypertonic fluid from the gastro-intestinal tract. Single oral doses of 0·2 ml crude oil inhibits mucosal water and sodium transport and the effect of the dose persists for 4 days. The resulting dehydration may also be a contributory mortality factor.

Following a large scale oil pollution incident involving Bunker C fuel oil adult ducks and young chicks were fed samples and the results compared with the analyses of ducks, auks and grebes Podicipedidae killed in the incident (Snyder et al, 1973). Pathological findings on birds naturally and artificially ingesting oil agreed closely and were substantially similar to those already described.

In addition tissues were analysed for $C_{15}+$ hydrocarbon fractions. This fraction was recovered from the liver of chicks dosed with 1·5 ml oil at 2 day intervals for a week but only from the intestine (and not from liver, kidney, brain and fat) of duck given 10 ml doses on each of two days and killed 48 hours after commencing the experiment. This suggests that this hydrocarbon fraction may not reach past the digestive tract until fat reserves are exhausted. In tests on 8 naturally killed birds, 3 gave negative results for the $C_{15}+$ fraction, but this was recovered from all tissues tested (liver, kidney, brain, fat, heart muscle) of one bird and from at least the liver of the remainder, often in substantial quantities, varying between 1·25 mg/g and 9·1 mg/g wet weight. This perhaps suffices to demonstrate that hydrocarbon uptake does occur but that individual variation may be substantial and that more critical experimentation is desirable and I understand this to be at present in progress in the U.S.A.

It should be quite clear that internal and external oiling effects will rapidly prove fatal to most birds from a normal oiling incident, without remedial treatment. Oil ingestion clearly plays an important part in causing observed disorders but it still remains to assess how much is due to induction of physiological stress and how much to toxic effects and in the latter case to identify the components of oils principally responsible for observed symptoms. Only then will it be possible to understand more precisely the mechanisms by which the toxic effects operate.

I should like to conclude by briefly mentioning a very different aspect of the relationship between seabirds and petroleum hydrocarbons which has important implications for control and conservation measures alike.

Whereas the effect of chronic small-scale oil pollution can usually only be minimised by technological improvements, vigilance at danger points and in high risk areas, and perhaps sometimes legislation, with a large oil slick steps can also be taken to reduce its impact on marine life.

From the point of view of seabirds removal of the slick following its containment by booms would be ideal but this is not feasible in rough weather and even under better conditions it might often be difficult to get enough equipment to the scene quickly enough for an efficient operation. More often the slick will be chemically dispersed. Here again reduction of seabird casualties depends on rapid action and it appears that care must also be taken that a quantity of dispersant appropriate to the volume of oil is used otherwise it seems that incomplete dispersion may occur — and a dispersant-oil film is just as effective at destroying plumage waterproofing as oil alone.

In winter, even with the best planning and organisation, there will always be difficulties in taking appropriate action but for all seabird wintering grounds near oil terminals and developments there should be an adequate supply of slick dispersing equipment and fully tested operational contingency plans.

It is clearly very difficult to make any special arrangements to deal with incidents involving seabirds on their spring and autumn migrations but the major threat is posed by the prospect of oil slicks near important breeding colonies, an increasing risk with the continuing oil development off the Scottish east coast. Any disaster will inevitably affect not only adult birds but also that year's production of offspring. Even lightly oiled birds will transfer oil to eggs they are incubating, usually resulting in hatching failures (Gross, 1950; Hartung, 1965; Kadlec and Drury, 1968; Rittingshaus, 1956). Also many juvenile birds are particularly vulnerable once they have left the breeding colony but before they can fly properly and adult auks are also flightless for a period during their moult, which immediately follows breeding (Birkhead and Taylor, in press).

It is even more important to take rapid appropriate action with a slick near a breeding colony and even though the use of audible and visible stimuli (Crummett, 1973) may be helpful in keeping birds away from a slick, and should be investigated further, it is essential to have accurate information on the speed of movement of slicks under various weather conditions and on how quickly action can be taken to disperse the slick comprehensively and efficiently.

There is a basic need for the establishment of agreed areas of high risk of oil pollution to breeding and wintering seabirds and ensuring that for these areas every facility for minimising casualties is available close at hand and can be used with the least delay.

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