

Chapter (non-refereed)

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MERSEY ESTUARY BIRD MORTALITIES

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ABSTRACT

Between late summer and early winter in the years 1979-1982, birds died on the Mersey estuary with high levels of alkyl lead compounds in their tissues. The behavioural and morphological changes seen in these polluted birds in the wild were also seen in birds dosed with alkyl lead compounds in the laboratory. Furthermore, tissue levels of alkyl lead compounds in affected laboratory birds were similar to those found in affected wild birds. We concluded that the majority of birds that were found dead on the Mersey in this period were killed by the alkyl lead. A monitoring programme to determine the levels of alkyl lead in birds now living on the estuary has shown that many birds contained sufficient alkyl lead to cause sub-lethal effects and to impair their chances of survival. Further mortalities may occur should environmental factors, linked with the hydrodynamics of the estuary area, combine to cause an increase in the birds' exposure to alkyl lead compounds.

INTRODUCTION

The Mersey estuary in north-west England is heavily industrialised and receives effluent from these industries and sewage water from the towns and cities in the area. The estuary supports a large number of birds, mainly overwintering waders, wildfowl and gulls. At least 2500 birds were found dead on the estuary in 1979, and smaller mortalities occurred in 1980, 1981 and 1982. High levels of alkyl lead compounds were found in the birds. Earlier reports have described the incidents between 1979 and 1981 (Head *et al.* 1980; Osborn & Bull 1982; Bull *et al.* 1983). Details of experimental studies examining the toxicity of alkyl lead compounds to birds have also been published (Osborn *et al.* 1983). These experimental studies supported the view that the majority of the birds found dead on the estuary in recent years were killed by alkyl lead compounds.

This report outlines the main findings of the incident-related work on birds, gives data on the levels of alkyl lead in birds shot and netted on the estuary for monitoring purposes, and briefly discusses possible and actual effects of these levels on the birds.

METHODS

Methods have been described in Bull *et al.* (1983) and Osborn *et al.* (1983).

RESULTS

As described by Bull *et al.* (1983), mortalities on the Mersey estuary involving large or notable numbers of birds have occurred between late summer and early winter.

Many species have been involved. In 1979, 1300 of the birds found dead were dunlin (*Calidris alpina*) along with several hundred gulls (mainly black-headed gulls (*Larus ridibundus*)). In 1980, mortalities were mainly gulls, *L. ridibundus* again. In 1981, mortalities were spread amongst several species and in 1982 herons (*Ardea cinerea*) were prominent. There have been wildfowl casualties in all years (eg teal (*Anas crecca*) and mallard (*Anas platyrhynchos*)).

Affected birds on the estuary exhibited unco-ordinated movements and a head tremor. Some seemed unable to feed properly. Post-mortem examinations of dead birds showed they had discoloured intestines, brilliant green bile and discoloured livers. Analyses for toxic chemicals showed that the only measurable chemical detected in significant quantities was an alkyl lead compound. This was most probably trimethyl lead. Tissue levels of alkyl lead were generally >10 mg/kg wet wt in the livers of dead birds. In shot and netted birds, some contained >5 mg/kg wet wt and a high proportion contained >1 mg/kg. Many of the shot and netted birds had some of the abnormal morphological features seen in dead birds. Osborn *et al.* (1983) showed that birds that died when dosed with alkyl lead compounds exhibited very similar behavioural and morphological features to those seen in sick and dying birds on the Mersey. Birds given sub-lethal doses of alkyl lead compounds showed loss of condition and had the morphological features found in live birds shot and netted on the Mersey. In addition, the experimental work showed that dosing with alkyl lead compounds led to dose-related enlargement of the gall bladder and dose-related bone marrow activation.

Tissue levels of alkyl lead in dosed birds were similar to those observed in wild birds in which similar effects had been seen.

It was concluded that the experimental work supported the view that alkyl lead compounds had poisoned birds on the Mersey estuary and that the majority of the bird deaths recorded on the Mersey between 1979 and 1982 were caused by the birds eating prey contaminated with these compounds.

Attempts have been made to collect birds from the estuary at intervals so that (i) the amounts of alkyl lead in their tissues could be measured and (ii) they could be examined to see if they showed any of the abnormal morphological features experimentally associated with sub-lethal tissue levels of alkyl lead. Attention has been concentrated on 2 species, teal and dunlin.

Figure 1 shows alkyl lead levels in Mersey teal livers between 1980 and 1982. Although there may be some evidence of a decline in levels (we believe alkyl lead effluent levels on the estuary have fallen), some birds still contain high enough levels to cause some sub-lethal effect. Seventeen teal collected in February and November 1982 have been studied

in particular detail to look not only for morphological abnormalities, but also for evidence of the loss of condition that alkyl lead compounds brought about in laboratory birds. No evidence of any loss of condition was found in these teal, but the majority exhibited some morphological abnormality in that they had enlarged gall bladders and green-stained livers and intestines. Teal affected in this way had a mean level of alkyl lead in their liver greater than that found in the unaffected birds (Figure 1).

For various reasons, fewer dunlin samples have been obtained. Figure 1 also shows the liver levels of alkyl lead found in dunlin netted on the Mersey. Overall, these show little real sign of any decline in the level in live birds since the time of the 1979 incident, although when the August 1980 results became available it appeared a decline had occurred. Furthermore, morphological changes in dunlin were more severe than those in teal. Studies of dunlin body condition have yet to be made.

DISCUSSION

There are a number of features of these mortality incidents that can only properly be considered when studies currently in hand at the North West Water Authority laboratories, and elsewhere, are completed. These features include an explanation of why mortalities were not seen in earlier years and why they tend to occur in the period of the year when they do. They may all be explained in terms of the hydrodynamics of the estuary and associated waterways.

However, so far as the birds are concerned, many still have levels of alkyl lead in their tissues that are sufficient to cause morphological changes. Also, mean levels in teal and dunlin are not yet below the 0.5 mg/kg wet wt which had been suggested (Bull *et al.* 1983; Osborn *et al.* 1983) as the maximum level above which levels should not rise if the chance of a substantial further mortality was to be avoided with a fair degree of certainty.

It seems that large numbers of birds on the estuary (particularly dunlin and other waders) are still at risk from alkyl lead compounds and that, given an unfortunate combination of environmental factors linked with the hydrodynamics of the estuary area, another substantial bird mortality could occur.

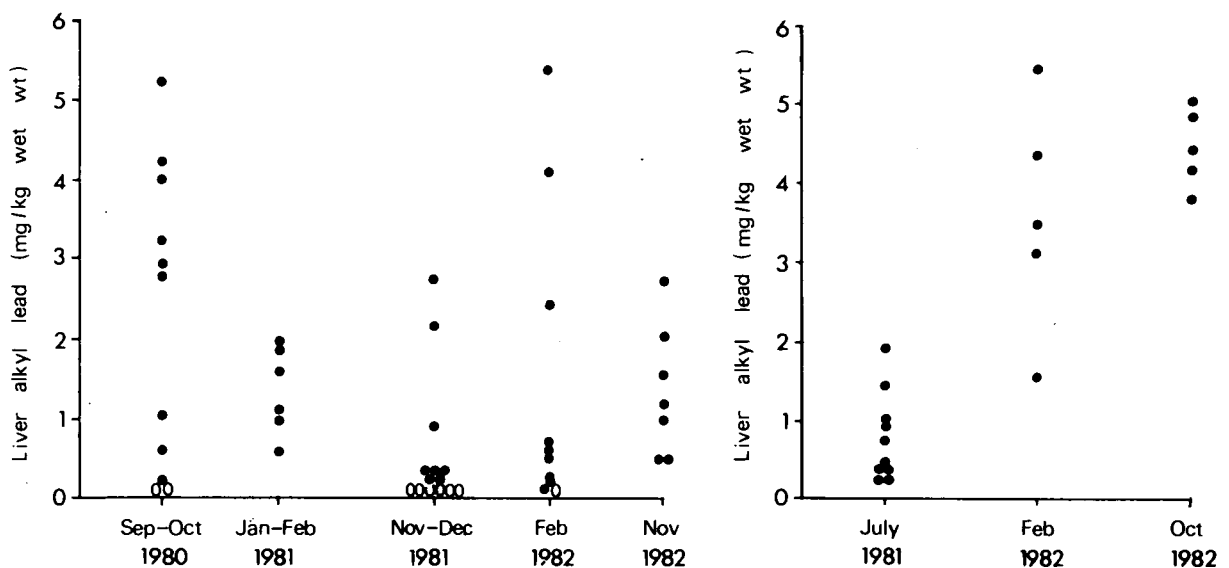
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Figure 1 Concentrations of alkyl lead in livers of live-caught teal (left) and dunlin (right)



Notes:

1. More birds must be analysed before statistical tests can be done or conclusions about trends in the data can be drawn.
2. 0 = <0.1 mg/kg.
3. Many of these birds showed internal signs of being affected by alkyl lead (Osborn *et al.* 1983). In 17 teal examined in February and November 1982, alkyl lead levels in livers of the 10 affected animals were 1.7 mg/kg compared to 1.0 mg/kg for unaffected birds. The respective geometric means were 1.1 and 0.4 mg/kg.
4. Some data were added after the workshop.