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International Council for the<br>C.M.1993/F:9<br>Exploration of the Sea

# REPORT OF THE SECOND SPECIAL MEETING ON ICHTHYOPHONUS IN HERRING 

Aberdeen, Scotland, 21-22 January 1993

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# SECOND SPECIAL MEETING ON ICHTHYOPHONUS IN HERRING 

SOAFD Marine Laoratory, Aberdeen, Scotland

21-22 January 1993

## 1 INTRODUCTION

### 1.1 Terms of Reference

The meeting was convened to meet Resolution 2:46 of the 80th ICES Statutory Meeting which stated: "A second Special Meeting on Ichthyophonus (Convener: Dr A McVicar, UK) will be held in Aberdeen, Scotland, UK from 21-22 January 1993 to update and analyse information on the prevalence and impact of Ichthyophonus in different herring and other fish stocks and to estimate the extent of mortality on herring stocks."

### 1.2 Participation and Agenda

The draft agenda was adopted with slight amendments (Appendix 1). Dr D Bruno and Mr D Bucke were selected as rapporteurs.

Eighteen delegates attended the meeting including members of the Working Group on Pathology and Diseases of Marine Organisms, the Working Group on Stock Assessment of Pelagic Fish Species and other scientists involved in Ichthyophonus research (Appendix 2). Dr Carl Sindermann, USA, was a specially invited participant having pioneered many aspects of the research investigations on Ichthyophonus epizootics in herring.

## 2 DISTRIBUTION AND PREVALENCE OF ICHTHYOPHONUS IN HERRING

### 2.1 Historic Information

Objective: To assess previous information on Ichthyophonus infection in herring, to put the current epizootic into context and to determine if the epidemiological pattern occurring was similar to that in previous outbreaks.
a) Western North Atlantic. In North American waters there have been five previously recorded epizootics of Ichthyophonus in herring stocks, the last occurring in the Gulf of St Lawrence in 1955. A summary paper of available information was tabled by C Sindermann. Infection occurred in all life stages, first becoming apparent in the juveniles of a population but soon after being found in adults. Mortalities were observed in the Gulf of St Lawrence outbreaks but not off Maine, possibly reflecting the shallower water in the former area. Sindermann has concluded that Ichthyophonus was the most serious disease of herring in the western North Atlantic.
b) Eastern North Atlantic/North Sea/Baltic. Prior to July 1991, there is no scientific evidence that epizootics of Ichthyophonus had occurred in herring in European waters. Thus, although it was being concluded that the current Ichthyophonus outbreaks had probably been developing undetected for some years it was not possible to associate the large fluctuations in European herring stocks which have been noted with outbreaks of this disease. Ichthyophonus has a long history of occurrence in European waters, having been recognised in haddock, plaice and other fish stocks since the beginning
of this century. With the evidence of high levels of Ichthyophonus infection in haddock and lethal epidemics in plaice particularly in an area north of Scotland, it could be concluded that occurrence of the disease agent in an area was not sufficient to cause an epidemic in herring.

Conclusion: Considerable similarity was noted in the sequence of events leading to the earlier Ichthyophonus epizootics in North American waters to those occurring in European waters in 1991; evidence of dead fish on the sea surface and bottom and dead fish washing up onto beaches. Some differences were apparent in the epizootics in the two areas notably in the occurrence of infection initially in young fish in American waters.

### 2.2 Identification and Taxonomy

Objective: To obtain information on whether the same disease was affecting herring on different sides of the Atlantic and in different species of fish in the same area.

Without knowledge on whether a single species of Ichthyophonus was affecting herring in different localities and different species of fish in affected areas, the full interpretation of the epidemiological data from herring and of sources of infection in other organisms is filled with difficulties. From his studies in Canadian waters, T Rand presented information on the life-cycle of Ichthyophonus. It was emphasised that, although the life cycle seemed relatively simple, there were substantial differences in the developmental patterns, spore size, and morphology of the fungus in different fish species, suggesting that different forms, or even species, of the organism could be possible. For example, the dimension of the spores normally varied between 8-20 $\mu \mathrm{m}$, but in some species were found to range up to $32 \mu \mathrm{~m}$. Caution was advocated in interpreting morphology as it was also considered possible that appearance could be significantly affected by the fish species the fungus was infecting. This was supported by observations in culture where there have been several developmental patterns of the organism observed, possibly reflecting different growth patterns on different media. However, it has been shown that Ichthyophonus from herring is capable of affecting other species of fish (eg plaice, common dab) and there is good information from the literature that it shows low host specificity.

Taxonomically, Ichthyophonus does not fit into any known order of fungi.
Conclusions: It was concluded that Ichthyophonus is recognised as a fungus with at least two different forms (possibly different species). However, it was agreed that the morphology of Ichthyophonus in herring was identical in the east and west North Atlantic and that the same organism is found in other fish species in an affected area. More research effort is needed on the specific identification and taxonomy of this organism(s).

### 2.3 Current Data on the 1991-92 Epizootic

Objective: To update information on the distribution and prevalence of Ichthyophonus in herring in European waters.

The first records of infected herring from the current European epizootic were made in the North Sea and Kattegat in July 1991 and this was the subject of the ICES Special Meeting on Ichthyophonus held in Lysekil, Sweden in November 1991 where standardised sampling and diagnostic methods were established. These methods provide the raw data which the current meeting was convened to consider. Working papers reporting studies on the prevalence of Ichthyophonus were tabled by several countries (Denmark, Scotland, Norway, Sweden, Germany, The Netherlands, England and Wales), with written reports from Iceland and Finland. It was suggested that these be submitted as an ICES Co-operative Research Report (see Section 7). All data confirmed that the disease is present at highest prevalence in the northern North Sea east of Shetland up to $64^{\circ} \mathrm{N}$, inshore along the Norwegian coast, and in the Skagerrak, Kattegat and western Baltic. The
prevalence in the North Sea decreased southwards to $56^{\circ} \mathrm{N}$ and was only occasionally recorded in the southern North Sea. A single herring with Ichthyophonus was reported from Icelandic waters. Ichthyophonus has not been detected in herring west of the UK and Ireland.

A note of caution was introduced on the use of disease prevalence data from trawling surveys as this method of fishing was considered to be excessively selective for catching infected fish and hence less selective for healthy fish.

Conclusions: The focus of the epizootic is in the northern North Sea, extending along the Norwegian coast to the Skagerrak, Kattegat and western Baltic.

### 2.4 Possible Causes and Sources of the Epizootic

Objectives: To consider if underlying causative mechanisms of the epizootic could be identified and to determine if common factors could be detected in North American and European waters.

Physical factors. It was suggested that the start of the epizootic could have been linked to recent hydrographic changes in the northern North Sea. Some evidence (which was not considered to be incontestable) has indicated that high nutrient upwellings occurred in the summers of 1989 and 1990 and, as a consequence, it was suggested that herring migration routes may have changed to follow these new upwellings. Such changes could have significance to Ichthyophonus. Supporting this, it has been noted that the pattern of other parasitic diseases of fish has changed in relation to changes in hydrographic features, e.g., larval tapeworms in mackerel in the mid 1970s in relation to changes in water currents west of UK and Ireland.

Biological factors. Epidemics of infectious disease are often associated with high host density but it was the opinion of the meeting that no obvious direct correlation could be made between the current epizootic and the state of European herring stocks.

As blue whiting were abundant in the area where herring were affected and had a migration pattern coincident with the occurrence of infected herring, it was suggested they might act as a reservoir for this disease. In the absence of evidence of whether or not blue whiting is extensively infected with Ichthyophonus this was indicated as a topic worthy of further study.

As one possible explanation for the absence of the disease in the southern North Sea stocks, it was suggested that herring feeding north of the Shetland Isles during the summer either died or were too sick to migrate southwards if they became infected. In contrast, the North American experience showed that herring became infected when they moved inshore and that it appeared that hydrographic changes were not relevant.

To date there is only evidence for the oral route of infection and experimental infection has only been successful when fish were fed doses of 105 spores/g fish or greater. Consequently, in the open sea, large numbers of spores would have to be available to individual fish for infection to occur. In the Kattegat all sizes of herring, including 0 -group, were found to be infected and Ichthyophonus spores possibly drifted with the larvae so providing a source of infection at an early stage. Small fish may feed on the relatively large spores mistaking them for food particles. Alternatively, there may be some form of intermediate host (eg krill, plankton) which could be a reservoir of infection. It was thought possible that juvenile herring could be a significant source of infection for adult herring in the Kattegat, but these two groups do not normally mix and have different feeding areas. Attempts should be made to look for a common carrier between herring populations as they were distinct groups with no, or very little, mixing.

Although both cod and haddock may contain a large number of spores, the infection was contained within a granuloma and it is unlikely that these would be a significant source of spores in the water. Other species
of fish such as plaice, which show less ability to resist infection, however, may act as a production focus for spores in an area.

Conclusions: No underlying cause of the current epizootic of Ichthyophonus in European herring was identified. The possibility of a link to recent hydrographic changes remains a possibility.

### 2.5 Analysis of Sources of Variance

Objectives: To identify sources of variance in data on Ichthyophonus and to suggest solutions or improvements.

Accurate data on the prevalence of Ichthyophonus infection is essential to provide a sound basis for calculations of effects of the disease. The meeting recognised that there were considerable difficulties in obtaining accurate data on disease prevalence in wild fish populations and that particular problems existed for Ichthyophonus in herring. These included:

Herring stock density. Where stock densities were high, sampling did not reveal as many infected fish as where stocks were less dense and spread out, suggesting that infected fish were stragglers from the main shoals.

Different shoals may show different levels of infection and as these pass through an area the prevalence of infection observed would vary. Seasonal variations were identified as being particularly important.

The fishing gear used to catch herring could also alter disease prevalence. A comparison of different types of gear demonstrated that a higher prevalence of diseased fish was found in pair trawl samples and that lower prevalence levels were found in gill net samples followed by purse seine. This may be correlated with a possible difference in behaviour of affected herring and in their ability to escape or avoid different types of nets.

## Conclusions:

a) Disease sampling from commercial catches caught by purse seine probably provides the most accurate estimate of prevalence of Ichthyophonus, although it may still underestimate the true prevalence by catching healthy normal shoaling fish.
b) Data from an area should be considered seasonally.
c) In an effort to reduce further the variation and effort in sampling beyond that suggested by the Lysekil meeting the following modifications to suggested procedures were proposed:

As the prevalence of disease, recorded by visual examination of the heart and visceral organs at the same time as the maturation assessment was made was only slightly less than combined visual and microscopic examination visual examination only would give sufficiently accurate data. Some concerns were expressed about the ability of observers to detect infection visually in the hearts of small fish and it was recommended that the combined examination be used for fish less than 20 cm .

For survey work, the sample should be stratified by length, i.e., 20-24, 25-29 and $>30 \mathrm{~cm}$, except in the Baltic where stocks were smaller in length, (i.e., 15-19, 20-24 and $>\mathbf{2 5} \mathbf{~ c m}$ ).

## 3 PATHOGENICITY AND DISEASE DYNAMICS

Objectives: to obtain information on the effect of Ichthyophonus on herring, particularly on survival.

### 3.1 Histopathology and Immunology

Variations in the extent of host response were demonstrated with a series of histological slides showing different life-cycle stages of the fungus and different rates of infection in individual herring hearts. A cellular defence response was clearly being activated but there was little evidence of containment and destruction of the infection in any herring. This contrasted with information on the disease in cod, haddock, mackerel and dab (including material from a collection made by Johnstone sampled at the beginning of the century) where a strong cellular response apparently normally prevented proliferation and spread of the infection throughout the body. This evidence would suggest that herring have little resistance to Ichthyophonus and the general conclusion can be made that the disease is probably normally lethal. Information presented on the obviously emaciated state of infected herring supported this conclusion.

It was reported that there was very little known about the immune response of fish to Ichthyophonus and although antibody to Ichthyophonus could be shown in species such as plaice, protection to the host was not demonstrated. The antibody response in herring to Ichthyophonus is unknown.

### 3.2 Information Derived from Experiments

Laboratory experiments on the sequential development and pathogenicity of the disease may not reflect the natural situation. Important factors such as predation are absent and the experimental mortality rate may be slower than in the wild where it could be expected that moribund fish would be rapidly removed by predators. Nevertheless, experiments can give some information about the infection and mortality rates. Sindermann stated that experimental exposure of two year old Gulf of Maine herring to fungal material from the St Lawrence epizootic in 1955 resulted in an infection rate of one acute to two chronic. Mortalities from acute infections occurred in one year old herring within 15-30 days, whilst deaths from chronic infections were spread over six months. At the termination of the experiment, the survivors revealed few infected. The conclusion was that most herring die of Ichthyophonus and the infection dose needed to cause infection was 105 spores/g of fish material. At the Marine Laboratory in Aberdeen and in Denmark, some experimental work including herring-challenge had recently commenced, but the results were so far inconclusive. A further challenge had been made using plaice and dab, but again the results were inconclusive. These trials will continue.

Conclusions: Current evidence indicates that Ichthyophonus is lethal to herring. Two phases of the disease were recognised in experiments; acute when mortality occurred in 15-30 days and chronic when mortalities were spread up to six months.

### 3.3 Health, Economic and Trade Concerns

Objectives: To evaluate if there are potential human health implications from Ichthyophonus and to consider available information on effects on herring as a product.

There is no indication from the available scientific or medical literature of any pathogenic effects or infection in humans exposed to Ichthyophonus. Experiments currently being conducted in Denmark are addressing this question directly. When rats were exposed to the same dose of spores as prove infective to fish, there was no evidence of infection, with the body tissues appearing normal after six months. Reports that Ichthyophonus had been observed in gulls and some crustaceans require verification of the species involved through direct comparison with Ichthyophonus from fish.

It has been found that infected herring were unsuitable for processing, as they fell apart and blocked machinery. They tended to fall from kippering racks, and also have an offensive smell. Transfer of infection from affected fish fillets to unaffected fillets by contact was indicated in some cases. The disease condition
in other fish species is well known to some fish processing sectors, e.g., infected haddock are known as "greasers" in the North East Scotland industry.

There was no survival of the fungal spores after processing through the normal pickling processes with vinegar and salt medium ( pH 3.0 ). Killed spores were recognised by the condensed cytoplasm, large pores and the breaking up of spores. However, Ichthyophonus is known to survive temperatures of $60^{\circ} \mathrm{C}$ for at least 30 minutes and some spores are believed to survive freezing; this information has implications for the use of affected fish as a source of fish food, e.g., for farmed fish.

Conclusions: There is no information on Ichthyophonus to suggest any associated human health problems. When infection prevalence levels are high, there are recognised significant problems to fish processors.

## 4 HERRING STOCK ASSESSMENT

## Objectives: By combining data sets from fish disease specialists and herring stock assessment specialists attempt to delineate affected stocks and to calculate the impact of the disease.

### 4.1 Delineation of Affected Herring Stocks

Background information on herring stocks was presented. It is generally accepted that there are several separate populations of herring in the North Sea, although inevitably there is some mixing of stocks. An estimation of stock size by acoustic surveys in 1991 revealed 1.3-1.4 million tonnes in the North Sea and, from earlier records, there is information that the population had been at this level for some years. From the disease data presented the epizootic of Ichthyophonus is occurring in the Norwegian spring spawning stock, part of the northern North Sea stock, the western Baltic spring spawning stock and possibly the Iceland autumn spawners.

### 4.2 Historical Data During and After Epidemics in North American Waters

Sindermann presented a summary of data from epizootics in the Gulf of Maine and Gulf of St Lawrence with reference to published data from pre and post epidemic periods. It was indicated that for the five year period following the start of heavy mortalities in the Gulf of St Lawrence, average herring landings decreased by nearly $40 \%$ with no change in fishing effort. Spring spawning stocks were particularly affected. However, caution was advocated because of insufficient knowledge of the size of the herring stocks and their fluctuations.

### 4.3 Performance of Present Herring Stocks

## North Sea herring

Assessment of herring stocks was based on annual catch data, acoustic surveys, larval surveys and recruitment surveys. For 1991, there was no change in abundance and the prediction for 1992 was that there was no trend of changes in the stock. The current 1.3 million tonne stock was not large and, by historic terms, it should currently be at 2 million tonnes. It was concluded that there was no evidence for a population change resulting from Ichthyophonus disease. Although ICES and ACFM are concerned about Ichthyophonus in herring, at present they state that there is no need for specific management advice to be given as the stock was well above the minimum acceptable range of 800,000 tons. It is possible that the spawning stock may decline because of fishing mortalities and recruitment may be less than expected in future years.

## Norwegian spring spawning herring

The herring stocks in northern Norway reached over 10 million tonnes in the 1950s and 1960s and since 1970 the stock had been low ( 0.05 million tonnes). There was a good year class in these stocks in 1983 and since 1988 this has been the main contributor to the spawning stock. The minimum target of 2.5 million tonnes spawning stock is now within reach. There is no evidence of Ichthyophonus in young herring in the Barents Sea, only in the larger fish. It was suggested that there had been a fairly recent change in migration and behaviour pattern of Norwegian herring, since almost all the Norwegian herring population overwintered in one fjord. Because of the high stocking density in this fjord, herring may be under stress with oxygen deficiencies. The change in population habit started in 1988, and the fat content in these fish was still high until 1991 when it decreased by $5 \%$ in 1992. It was considered reasonable to believe that within a 5-10 year period a spawning stock of 5-7 million tonnes was possible. There was no indication from stock assessment studies that this population was being seriously affected by the Ichthyophonus infection. The possibility was noted of a compensatory effect of predation turning away from uninfected parts of the population to infected fish, which could reduce the overall impact of the disease on the total population. The difficulty in measuring such an effect was recognised.

### 4.4 Estimation of Mortality Rates

A preliminary attempt to calculate mortality in herring populations in Divisions IVa, IVb and VIa due to Ichthyophonus was prepared by J Simmonds.

Information on the extent of the infection and the resulting rates of mortality is limited. However, given some assumptions, it is possible to obtain a very approximate estimate of mortality rates for the disease itself.

The numbers of herring in the population that will die due to the disease may be estimated from:

$$
\begin{equation*}
\text { Annual Mortality Rate }=\frac{\mathrm{PR} * 365 * \mathrm{DM}}{\mathrm{LE} * \mathrm{DP}} \tag{1}
\end{equation*}
$$

## in which

1) the observed prevalence $(\mathrm{PR})=$ the fraction of the total population that are found to be infected from samples.
2) the detected infected proportion (DP) = the fraction of the infected fish in the sample that have been detected by the examination method.
3) the disease mortality $(\mathrm{DM})=$ the proportion of infected fish that will die from the infection.
4) the life expectancy $(\mathrm{LE})=$ the average duration of the illness (days) for those fish that are expected to die following infection.

The ICES coordinated herring acoustic survey was used to estimate the prevalence of infected herring (Simmonds et al., in press). During the survey, samples of fish were inspected for Ichthyophonus infection macroscopically. The numbers infected and the location of samples are shown in Figure 1. This figure also shows the simple area averages, clustering samples together on an equal basis for each fish. The samples have also been combined using linear interpolation for unsampled squares. Both the unweighted and weighted method were used for squares without data. The distribution of infection rates for the weighted method is shown in Figure 2. The total numbers infected are shown in Figure 3 and tabulated along with the numbers
uninfected and the percentage infection rates by ICES area in Table 1. The three columns show the different methods for averaging the samples; they are not significantly different. The right hand column is probably the best. In this study the Baltic spring spawning herring are included with the North Sea autumn spawning fish as there was no information on the proportions of infected fish from each population.

The estimated values for the parameters in equation (1) are

| Detection proportion | 1 |
| :--- | :---: |
| Mean life expectancy | 105 days |
| Disease mortality | 1 |
| IVa and b proportion infected | 0.045 |
| VIa proportion infected | 0.00 |
| Mortality rate for Sub-area IV | $16 \%$ |
| Mortality rate for Division VIa | $0 \%$ |

## Assumptions

- The infection rate is uniform over the year.
- The infected fish are not subject to other preferential mortality.
- The infected fish are correctly represented in the samples.
- The life expectancy for the diseased fish is correctly known
- All infected fish in a sample are detectable.

If these assumptions are incorrect the errors may be considerable.
a) Effects of errors in the assumption of uniform infection rates

If the source of infection is external to the population, infection will occur only when the source and population are coincident in space and time. If the infection is transferred through ingestion the infection may only occur when feeding rates are high enough. Under these circumstances the relationship will no longer follow equation (1) given above as the numbers of infected fish will depend on the different rates of infection through the year, the life expectancy-versus-time relationship and the point of observation.

If the point of observation occurs immediately after a short infection period the numbers infected will represent the entire infected proportion for the year, and equation (1) will cause an overestimate of the annual mortality rate. If ingestion is the major source of infection then it is likely that infection occurs mostly between April and October. By July, the observation point, the infection process will be almost fully underway giving an infection rate that is not critical to precise timing. The duration of infection will then be shorter than 365 days assumed. Using this assumption of uniform infection rate and a mean life expectancy of 105 days will give an overestimation rate of approximately two. As the most probable cause of infection is ingestion, significantly greater overestimation of mortality seems unlikely. If the observation point is well after the period of infection then substantial numbers of fish may have already died before being detected; thus, the mortality will be underestimated. For mortality to be underestimated by a factor of two the infection period would have to be short (significantly less than six months) and the peak infection would have to be more than 3.5 months prior to the observation point in July. This requires infection for a short period in winter.
b) Effects of predation on mortality errors

If there is no preferential predation on infected fish then the mortality is correctly estimated and all the mortality is excess mortality in addition to the normal annual natural mortality.

If the infected fish are subject to additional preferential mortality the life expectancy will be shorter than the 105 days. Using a figure of 105 days will cause an underestimation of the numbers of infected fish and of the mortality. However, some of the natural mortality normally experienced by the stock will almost certainly have transferred to the infected fish, compensating to some extent for the underestimation. Assuming $50 \%$ of the infected fish were lost to predation with uniform probability during the period of illness, the effective life expectancy would decrease from 105 to 78 days. Thus the mortality should be increased by $1 / 3$. However, if all of that mortality replaced natural predator mortality the effect would be to decrease the excess mortality to $50 \%$ of the total ( $66 \%$ of the predicted figure). The higher the proportion of ill fish that are taken in replacement of natural mortality, the lower is the impact of the disease related mortality. In the extreme case where all ill fish are predated as replacement there is no excess mortality.

It is impossible to put an upper bound on the effects of predator mortality. If predator mortality on ill fish is additional to the natural mortality, this will cause an underestimation of the infection mortality and this mortality should be added to the overall annual mortality of the stock.
c) Effects of errors in the mean life expectancy due to the disease

If the mean life expectancy caused by the illness itself has been incorrectly estimated, this will affect the calculated mortality. If life expectancy increases by $10 \%$ then mortality will be overestimated by $10 \%$. The estimate of mean life expectancy of 105 days is based on aquarium experiments on 2,000 one year old herring (Sinderman and Chenoweth). Stress on aquarium fish may be greater thus underestimating life expectancy. The infected fish in the North Sea stocks are often older fish and here the progress of the disease may be different. Accordingly it seems more likely that the life expectancy will be underestimated, and the mortality overestimated.

## d) Effects of errors in the detection of illness in samples

The numbers of diseased fish are estimated using macroscopic examination of the heart. Estimates of disease mortality at $100 \%$ are based on data from the same aquarium experiments described above and microscopic examination of tissue from samples of wild fish. These estimates seem fairly realistic but any errors in this factor will result in overestimation of the excess mortality. Errors in this parameter are expected to be small.

## e) Effects of errors in the estimates of the proportion of infected fish

Correct estimates of the proportion of infected fish in the stock are crucial to the estimation process. Herring is migratory and the spatial distribution is non-uniform. In addition the distribution of the disease is nonstationary in a statistical sense. It is necessary to obtain estimates of both abundance and proportion for the whole stock in order to establish the overall prevalence. The most obvious method for this is the use of acoustic survey data which provides estimates of abundance and coincident trawl data on the proportion of infected fish. The overall abundance does not affect the estimates of mortality but could be scaled by the results of the complete herring assessment procedure if required. The survey provides relative density figures to combine with temporally and spatially coincident estimates of infected proportions allowing calculation of total prevalence.

Studies of fishing sampling on herring suggest non-uniform mixing of infected and uninfected fish, and preferential sampling for some fishing gears. Comparison of demersal trawl and acoustic surveys suggest that areas of zero occurrence coincide. The highest proportions of infected fish occur in demersal trawl samples or in low density concentrations. The lowest proportions, but not exclusively so, were from dense aggregations using trawl or purse seine. The problems of determining the correct proportion for the areas where diseased fish are present are considerable. Determination of this factor probably gives rise to the largest source of error. In general it is easier to catch diseased fish so the prevalence may be overestimated. However, if the spatial distribution of diseased and uninfected fish is different, for example if diseased fish migrate slowly, or not at all, they may be underestimated because they are located in unsurveyed areas at the time of the observation.

The major sources of error are: the non-stationary spatial distribution of diseased and uninfected fish, the excess predator mortality on diseased fish over and above the normal rates of natural mortality, and the preferential sampling of diseased fish. The first two of these errors may tend to cause underestimation of mortality, the third overestimation.

## Conclusions:

Data from herring stock assessment studies have not revealed changes which could be attributed to abnormal mortality levels due to Ichthyophonus infection.

By combining the prevalence data of the disease from Dutch, Scottish and Norwegian sources for Division IVa, provisional estimates of the numbers of herring affected and possible impact could be made. It was noted that up to $4.5 \%$ of the herring (which could amount to 500 million fish) were estimated to be infected. As the disease is fatal for herring, the percentage of infected spring spawners could be important in Division IVa.

## 5 OTHER RESERVOIRS OF INFECTION

Objectives: From a consideration of information on other hosts for Ichthyophonus is it possible to determine if any of these may be acting as reservoir or transmission hosts important to herring?

Of seven other fish species (cod, whiting, plaice, dab, flounder, mackerel and sprat) examined in the Skagerrak and Kattegat, Ichthyophonus was recorded at prevalence levels of $1.2 \%$ in flounder and $6.6 \%$ in sprat. Only disintegrating spores where found in cod. No examination for Ichthyophonus of herring present in the stomach contents of predatory fish had taken place and it was considered that this should be encouraged. In the northern North Sea the disease has been previously recorded in haddock, dab and plaice. Low specificity of Ichthyophonus is indicated from the worldwide literature and the occurrence of a host record may only indicate that appropriate studies have been performed on that fish. Differences in the degree of containment of infection may prove to be important in determining whether a particular host species may be an important transmission agent of the disease or not. Insufficient information is available from most hosts for conclusions to be reached.

There are also reports in the literature of Ichthyophonus spores from Calanus sp. In view of the importance of this species as a food organism for herring, the meeting considered that research should be applied to challenge Calanus sp. and krill to establish whether these animals are successful hosts for Ichthyophonus.
Conclusions: No other host was recognised as important for transmission of the disease to herring but information available was limited.

## CONCLUSION

a) Currently, the following seriously infected groups of herring are identified:
i) Norwegian spring spawners
ii) Part of the northern North Sea stock
iii) SW Baltic stock

Other stocks, e.g., southern North Sea, south west Iceland are known to harbour infection at lower levels.
b) Epizootics of Ichthyophonus may decline quickly as experienced in the USA and therefore data should be gathered quickly if we are to learn from this epizootic. It was considered that monthly sampling should be carried out to establish the route of infection.
c) Although there is limited experience, it was concluded that there was currently no known risk to humans
d) Although the mortality rate following infection is known to be high in some areas, at the present time there is no evidence from the catch data that herring stocks in the North Sea were being seriously affected by Ichthyophonus infection.
e) An increased range of fish species and vertebrates should be examined from field and experimentally infected groups to determine possible reservoirs and sources of infection.
f) An increased effort should be directed towards surveying commercial catches. Although these may underestimate true prevalence, they probably provide the best index of disease prevalence. The current work on acoustic surveys should be continued. A stock assessment programme should be encouraged particularly in countries that currently do not participate. The current monitoring programme should be extended to include juvenile fish.
g) There should be an emphasis on increased experimental work to investigate the pathogenicity of the disease under experimental circumstances. Extrapolation of these data to mortality rates in the wild should be treated with caution.
h) The effect of freezing on spore viability should be further studied because of its commercial implications.

## 7 RECOMMENDATIONS

a) Stocks currently affected by an Ichthyophonus epizootic at significant levels are the Norwegian Spring spawning stock, part of the Northern North Sea stock and the South Western Baltic stock. Although pathogenicity of Ichthyophonus in herring is thought to be high no changes in stocks which could be attributable to the disease could be detected from stock assessment data. No recommendation can be made from the present data whether disease-induced mortality should be taken into account in management of affected herring stocks.
b) Working papers should be prepared and sent to A H McVicar, Aberdeen by 1 April 1993 for publication as an ICES Cooperative Research Report. They should be prepared in Word Perfect using Times Roman font 12ppt.
c) There should be continued cooperation between the pathology and stock assessment groups to determine Ichthyophonus-induced mortality rates in European herring. A further meeting should be held in late 1994 in Bergen, Norway, chaired by A H McVicar (Scotland) to consider data then available and to update recommendations.

## 8 REFERENCES

Simmonds, E.J., Dommasnes, A., Aglen, A., Corten, A. and Reid, D.G. 1993, in press. 1992 ICES coordinated acoustic survey of ICES Divisions IVa, IVb and VIa. ICES C.M. 1993/H...

## APPENDIX 1

## SPECIAL MEETING ON ICHTHYOPONUS IN HERRING

## Agenda

1. Opening of the meeting and background information.
2. Adoption of the agenda, selection of rapporteurs.
3. Distribution and prevalence of Ichthyophonus - data from different countries and sampling methods.
i) Historic information from east and west North Atlantic.
ii) Identification.
iii) Current data.
iv) Analysis of sources of variation.
4. Pathogenicity and disease dynamics
i) Histopathology and immunology.
ii) Experimental information.
iii) Economic and trade aspects.
5. Herring stock assessment data
i) Delineation of affected stocks from population and seasonal data.
ii) Historical data preceding, during and post epidemics (North American data, acoustic data, fish data).
iii) Performance of present herring stocks in affected and non affected areas.
6. Information on Ichthyophonus in other fish stocks.
7. Is there a measurable effect on fish stocks from Ichthyophonus infection which should be taken into account in fisheries management?
8. General conclusions and recommendations.
9. Closing of meeting.

## APPENDIX 2

## SPECIAL MEETING ON ICHTHYOPHONUS IN HERRING

## List of Participants

Banning, Paul van
Bruno, David
Bucke, David
Costa, Graca
Hagström, Olle
Hjeltnes, Brit
Lang, Thomas

McVicar, Alasdair (Chairman)
Mellergaard, Stig
Patterson, Kenneth
Rahimian, Hassan
Rand, Tom
Scullion, Francis
Simmonds, John
Sindermann, Carl
Sparggard, Bettina
Thulin, Jan
Toresen, Reidar

RIVO, Netherlands
SOAFD, Marine Laboratory, Scotland, MAFF, Fish Diseases Laboratory, England SOAFD, Marine Laboratory, Scotland

Institute of Marine Research, Sweden
Institute of Marine Research, Norway
Bundesforschunganstalt für Fischerei, Germany

SOAFD, Marine Laboratory, Scotland
DIFMAR, Fish Disease Laboratory, Denmark
SOAFD, Marine Laboratory, Scotland
Göteborg University, Sweden
Saint Mary's University, Canada
Fisheries Research Centre, Ireland
SOAFD, Marine Laboratory, Scotland
Oxford Laboratory, USA
DIFMAR, Fish Disease Laboratory, Denmark
Institute of Marine Research, Sweden
Institute of Marine Research, Norway

Table 1 Estimated numbers (millions) of herring from the acoustic survey in June-July 1992, showing numbers infected, numbers uninfected and the percentage infection (Simmonds et al. in press).

|  | Method of averaging samples |  |  |
| :--- | ---: | ---: | ---: |
| Area | Boxes weighted by <br> total numbers | Unweighted samples; <br> linear interpolation | Weighted samples; <br> linear interpolation |
| Numbers Infected |  |  |  |
| IVa | 532.80 | 521.57 |  |
| IVb | 3.94 | 5.23 | 524.16 |
| VIa | 0.00 | 1.05 | 2.53 |
| Total | 536.73 | 527.85 | 1.85 |
|  | Numbers Uninfected | 528.53 |  |
| IVa | 10069.26 | 1008.49 | 10077.90 |
| IVb | 1308.37 | 1307.08 | 1309.78 |
| VIa | 1927.49 | 1926.43 | 1925.64 |
| Total | 13305.12 | 13314.00 | 13313.32 |
| Total |  |  | 13841.85 |
| population | 13841.85 | 13841.85 |  |

Percentage Infection

| IVa | 5.29 | 5.17 | 5.20 |
| :--- | :--- | :--- | :--- |
| IVb | 0.30 | 0.40 | 0.19 |
| VIa | 0.00 | 0.05 | 0.10 |
| Total | 4.03 | 3.96 | 3.97 |



Figure 1 Samples values for Ichthyophonus infection and simple area averages from the combined servey (all herring) (Simmonds et al. 1993, in press).
119
9
7
5
3
1E
3
5
7
13
62
612

## Infection Rate

 (proportion) using weighted means and linear interpolation60
59
58
57



54

5

| 0.00 | 0.00 | 0.00 | 0.02 | 0.01 | 0.00 | 0.00 | 0.03 | 0.03 |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- | :--- | :--- |
| 0.09 |  |  |  |  |  |  |  |  |



Figure 2 Infection rate (fraction) by linear interpolation from the samples shown in Figure 1 (Simmonds et al. 1993, in press).


Figure 3 Numbers of infected herring (millions) from the combined survey (all herring). (Simmonds et al. 1993, in press).


[^0]:    *General Secretary
    ICES
    Palægade 2-4
    DK-1261 Copenhagen K
    DENMARK

