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REPORT OF THE ICES WORKSHOP ON THE USE OF PATHOLOGY IN STUDIES OF THE EFFECTS OF CONTAMINANTS

Dublin, Ireland, 21-22 April 1986

edited by

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A Workshop on the use of pathology in studies on the effects of contaminants was held in Dublin, Ireland, 21-22 April, 1986, with Dr. J. Thulin as chairman.

The Workshop participants were welcomed to the Department of Tourism, Fisheries and Forestry for Ireland by Dr. F.A. Gibson, Director of the Fisheries Research Centre, Dublin.

Introductory comments were made by the Chairman, who also outlined the terms of reference of the workshop which were:

a) critically review published and unpublished studies or case histories of pathology in relation to pollution in order to identify which approaches have been or could be used successfully in identification and quantification of changes related to pollution or in establishing cause-effect relations;

b) consider problems of sampling, statistical design and interpretation of results of pathology studies in relation to pollution;

c) investigate the possibilities to use pathology in programmes to monitor the effects of contaminants;

d) make proposals for further activities in this field within ICES.

Rapporteurs were selected, Dr. A. McVicar, Dr. J. McArdle for the first day and Dr. J. Stewart and Dr. B. Hill for the second day.

The previously prepared agenda (Annex 1) was accepted without addition and the members of the Workshop (Annex 2) introduced with a short synopsis of their principal work interests.

Review papers.

Dr. V. Dethlefsen presented an introductory review paper entitled "Studies on pathology in relation to pollution - present status" a preliminary copy of which was circulated to members and will be finalised for the report (Annex 3).

The object was to review available information and to make comment based on the author's own data. Groups for and against a connection between diseases and pollution agreed on the principals suggested by Sindermann (1983, 1984): principally that stress can provoke fish diseases. Various pathologies and disease of marine fish have shown a correlation between them, but should not be taken as proof of a cause - effect relationship, because the evidence is circumstantial. For European ICES area an increasing number of studies is available with the existence of correlations between pollution and disease but the conclusions were weak in most cases due to lack of associated chemical analysis. Systematic prolonged studies by FRG and the Netherlands have indicated a correlation. In Danish investigations consistently increased disease levels were found in polluted areas. In German investigations metals such as cadmium and lead in dab liver show highest levels in the Dogger Bank area, where there is also a high frequency of disease which may indicate an abnormal situation. In the dumping area for vastes from TiO - production in the centre of the German Bight increased chromium concentrations were found in external tissues of dab and a correlation existed between chromium contents of the fish and the site of their papilloma.

During questions it was indicated that high tissues levels of metals may be found without apparent disease. There is a requirement for data on fish population in such studies to ensure homogeneity of samples. The importance of age - disease relationships was stressed. It was considered that there are difficulties in using disease as a direct and straightforward index of damage by pollution but it was mainly the concept of using fish disease as a tool to monitor pollution that the Workshop was particularly applying itself to.

Dr. H. Möller presented a paper entitled "Problems of sampling, statistical design and interpretation of results in fish pathology/pollution studies" a preliminary copy of which was circulated to members and will be finalised for the report (Annex 4).

It was considered that the controversy between different hypothesis on the effects of pollutants on fish health was principally due to interpretation of results. After an introductory comment on the nature of pollution it was concluded that scientists working on associated topics could be divided into 2 groups: pure scientists who raise working hypotheses then seek arguments for and against them, or advocates of nature or of groups responsible for waste disposal who seek proof in one direction. It is essential that ecologists do not decide policy and must present results only. For cause-effect correlations it was suggested that a gradient in disease prevalence must be correlated with a gradient in pollution and that pseudocorrelations are excluded. Examples of fish disease and pollution studies on the Elbe Estuary were considered and the importance of 13 parameters influencing disease levels stressed. Understanding of the epidemiology of a fish disease requires not only studies in pathology and pollution but ichthyology, fisheries biology, hydrography, population dynamics of food stocks etc. Because of the major influence of natural factors on them some diseases were considered not to be useful for pollution monitoring studies. However, certain fish parasites and liver histopathology may offer a number of advantages.

During questions it was suggested that increased levels of disease in the estuary could be linked with higher tissue levels of pollutants in that area. However, increased heavy metals in the area may be linked with decreased liver weight associated with starvation. Another interpretation presented was that the high disease levels in the Elbe Estuary were abnormal or a deviation from the expected pattern. Discussion on the merits of using estuary, offshore and polluted areas concluded that changes in disease levels are due to a complex interaction of a wide range of factors which are difficult to bring together. With the knowledge that waste will be produced it was considered

important to pose the question whether these should be disposed of on land or sea. Finally it was noted to be important to establish the correct sensitivity of end-points in pollution measurements in relation to trials and observations on relationships with fish disease.

Participants experiences on pollution related diseases - short communications

1. Twomey, Ireland. A neoplasm, classified as a sarcoma has been studied in common cockles in Cork Harbour. The origin of the condition is not known. A seasonal trend was apparent in the first year of sampling with peaks in June and November but this was not apparent in the second year. The condition which was widespread within Cork Harbour, was present west of Cork but was absent east of Cork. Transmission was achieved by injection of neoplastic cells but neoplasms did not develop within 6 months of transferring healthy cockles into affected areas.

2. McArdle, Ireland. Disease surveys have been under way from 1971 in Dublin Bay and control areas considering several disease conditions. The recording system used enabled the location of different diseases on the fish body to be accurately plotted. Ulcers and fin rot mostly occur in the caudal area, Lymphocystis dorsally and towards the tail. The importance of repeat sampling seasonally and over a long period of time was stressed. Disease levels were highest in Dublin Bay and in flounder. Difficulty was experienced in relating disease levels to pollution.

   Dethlefsen presented results strongly supporting the requirement to carefully consider seasonal and interannual fluctuations in fish disease: epidermal hyperplasia varied from 1-2% to 6-7% prevalence depending on season. Evidence was presented for interannual variations in disease levels but it was considered that the time span of sampling was too short to discern trends.

Evidence for Lymphocystis regression was presented during questions of 6-7 months in experimental infection in dab and during spawning migrations in flounder. The possibility that the virus could persist without external signs was considered and the relationship between development and healing of lesions with temperature was stressed.

3. Peterson, Ireland. From December 1983 onwards 327 fish representing 15 species initially then 22 species were examined from Cork Harbour for 4 disease conditions: fin rot, petechial haemorrhage, ulcers and fin rot. Data was collected on age, sex, season and on chemical load. A statistical difference was detected between samples taken inside Cork Harbour and 5-6 miles outside, particularly with pigment abnormalities.

   In response to a question on the possible regression of Lymphocystis Peterson reported this was being tested by experiment.

4. Bylund, Finland. No correlation was established between levels of fish disease and titanium dioxide dumping sites. Fin damage and liver pathological changes in perch associated with pulp industries were found similar to that recorded by Swedish workers. Widely fluctuating levels of Lymphocystis in herring were found off the south coast of Finland but eye lesions in the same species were related to the method of capture, particularly excessive exposure to U V light.

5. Mellergaard, Denmark. Although considered in the Anton Dohrn Workshop it was necessary to re-emphasise the importance of sampling methods and statistical design in fish disease research programmes. Virus particles have been found in all samples of hyperplasia and papilloma examined.

   During the following discussion it was pointed out that experiments to transmit hyperplasia/papilloma by co-habitation, rubbing, scari-

fying were negative after 3 months duration. A correlation was found by Vethaak between papilloma and Lymphocystis in dab indicating a similar mechanism operating for both.

6. Egidius, Norway. In outer Oslo Fjord livers of cod showed significantly poor condition and an investigative study is in its early stages. Pollution in relation to fish farming was mainly an inshore problem. Research is in progress on the subject in Norway and ICES has set up a working group (correspondence) to determine the extent of the problem.

7. McVicar, Scotland. Four years survey data of various dab and haddock diseases in the northern North Sea showed no obvious trends but it is too early for meaningful comment. A study by D. Groman, Aberdeen, on a haemolytic anaemia condition in the estuary of the River Don indicated a probable pollutant cause. The rationale behind the development of a research programme into the causative agent was described. A similar condition has been observed by Dethlefsen in Baltic cod.

8. Hill, England. An internal working group has been set up in England to consider problems in developing a fish disease sampling programme in relation to pollution. Experience showed that dedicated disease cruises were necessary but in all aspects a compromise would have to be sought between facilities available and a meaningful scientific approach. No indication was found of increased diseae levels in polluted areas. Fish sampling areas should be chosen by consultation with fish stock assessment groups.

During questions it was noted that any statements on the existence or lack of correlation of fish disease generally with pollution should be qualified by a statement on the present state of knowledge on fish disease.

9. Dethlefsen, FRG. Considering a computer model with seven grossly detectable external diseases of dab, two different zones could be detected in the southern North Sea. Large scale pollution effects

must be considered in such studies. Some correlation was found between lysozymal activity and increasing disease. There was almost no seasonal variation in condition factor in dab in the Dogger Bank area unlike other areas and evidence for starvation possibly a result of non-selective feeding habits.

10. De Clerc, Belgium. A fish disease survey off the Belgian coast will terminate in 1986. Although diferences in the levels of cortesol and vasotoxin are good indicators of stress it is not known if these lead to disease.

11. Stewart, Canada, introduced the Canadian Technical Report of fisheries and Aquatic Sciences 1424. Although various abnormalitites and diseases were extensively studied and considered in relation to pollution/chemistry studies there were no indications of changes in prevalence away from natural background levels. It is important to consider that pollutants such as PCBs do not necessarily concentrate at the point of pollution.

12. Van Banning, Netherlands. In recording disease levels in wild fish populations on stock assessment surveys the following criteria had been set up and used; a) continuous recording for a long period (at least five years), b) recordings must be selected for high and low condition seasons (for Atlantic waters this means twice a year, early spring and autumn), c) recordings must be made in a wide area, covering several populations and/or environmental situations, d) data are to be plotted per disease and per fish species in a geographical grid system (e.g. ICES system), e) because of the existence of natural variations of prevalences a simple widescale classification must be used, f) choice of internal and external macroscopically visible diseases must be made to be integratable with standard handling.

    A specific pollution-related study was carried out in cooperation with Belgian workers on eel in the Westerscheldt area which included chemical data from sediment, water and fish tissues of diseased and

healthy fish. This study showed no direct relationship between contaminant level and the presence of disease but a possible indirect relationship. Bacteria occurred in the blood of eels in polluted areas.

During discussion the requirement to establish disease distribution maps and chemical distribution maps and to use specifically trained and interested staff was emphasised.

13. Vethaak, Netherlands. During a detailed study off the coast of the Netherlands in the period 1983-85, the state of health of flounder, plaice and dab was assessed considering external and internal conditions. Taking into account natural factors influencing variation in disease levels it was concluded with the data available that the observed disease rates in flounder might possibly be linked with marine water pollution. To confirm this hypothesis a follow-up study has been started in 1985 relating possible pollution-related diseases to body burdens of contaminants and the bacterial loading of the environment. It was considered particularly important to select reference areas to be as similar as possible to test areas.

14. Lindesjöö, Sweden. Fin erosion in perch in the Baltic was shown to be associated with effluent from a pulp mill, decreasing levels of the condition being found with decreasing effluent gradient. Fin erosion were also experimentally induced by holding fish in effluent. Chlorine bleaching in the pulp mill process was possibly implicated. The condition was absent from the vicinity of pulp mills not using chlorine bleaching.

15. Thulin, Sweden. The fish species Zoarces viviparous (viviparous blenny) was identified as a suitable biological indicator of pollution. Ready availability and the viviparous habit weald enable teratogenic studies to be easily carried out or for studies on new born fish to be made.

The use of fish pathology in programmes to monitor the effects of marine contaminants.

Dr. A. McVicar presented a paper with the above-mentioned title and a written version of his presentation will be finalised for the report (Annex 5).

It was considered that fish pathology is potentially useful in monitoring pollution as it measures relatively subtle and often non lethal changes in the structure of organisms and should be more sensitive than direct mortality effects. It is a biologically based index, sensitive to changes which can be measured in a natural population and its biological significance possibily more easily assessed than direct chemical measurements. However, pathological studies must be closely integrated with other pollution index studies, biotic and abiotic. A pathological condition is the end result of a long sequence of events within tissues and cells, hence lacks some sensitivity, it lacks specificity because of the limited ways in which tissue can respond, often lacks durability in the absence of causitive agents because of the powers of regeneration of fish tissue and, by often showing pathogenicity, can be difficult to measure because of differential mortality in the fish population. No specifically pathognomic lesions to pollutants have been identified in the North Sea and consequently pathology will probably be most useful as an indirect index. It has been shown that lowered environmental quality results in increased disease but because of the strong influence of natural factors on disease levels and lack of knowledge of fish disease it has not yet been possible to clearly establish links between pollution and disease in wild populations. Absolute levels of fish disease may be of less significance than trends over a period of time.

A firm data base is required for pathology - pollution studies with particular emphasis on principals of epidemiology, mortality effects and the relationship between prevalence and incidence of infection. Accurate standardisation of data from different sources is essential.

Resources, particularly cost and manpower availability, impose severe constraints on a study programme, and although methods employing commercial catches, joint cruises etc. have been attempted, the preferred approach has been to use specifically disease cruises staffed by specialists. Because of the extent of influence of natural phenomena on fish disease levels and the impracticality of performing frequent and extensive disease cruises a system is preferred in which the effect of natural variation is reduced as far as possible by standardising sampling in terms of season, fish species, area, disease studied etc. Similarly because disease often occurs at low prevalence levels extensive sampling of few conditions in large numbers of fish is preferred to intensive studies of a few fish. Appropriate diseases for study are those easily and accurately diagnosed and which preferably have a known aetiology and information available on their natural variation. Knowledge of fish stocks is essential to allow sampling homogenicity, or relatively static groups or species of fish used.

Following the presentation by McVicar the discussion focused in the main on the question of whether it was possible to use the presence of pathological conditions or infectious diseases amongst aquatic animals as a biologically based index sensitive to change that could be measured readily. A reasonable summary of the views impinging on this question is that while it is too early to link pollution and disease (clear definitive evidence is lacking) there is insufficient evidence to discount the relationship entirely. If progress is to be made towards providing the answer it will be necessary to extend our basic knowledge of disease, develop an extensive data base over a wide geographic area with time (possibly 5 years) and look for major departures from the baseline or "normal situation".

The views expressed by individual members of the Workshop ranged over a wide area. One member seemed to capture the majority view of the group by stating that initially he was pessimistic about being able to find evidence or support for the link between pollution and disease in aquatic animal populations. After hearing the national reports, however, he was encouraged and concluded that the support and evidence

for a link was stronger than it had been previously and that there is merit in continuing work aimed at settling the question of whether the link exists.

This member went on to state that the investigation of pathology of fish in certain areas was not enough to decide whether deviations encountered are related to pollution since the question was obviously far more complex than had been believed previously. He advocated additional measures or tests be undertaken such as:

(1) Scope for growth in mussels
(2) Physiological studies in invertebrates
(3) Hydroid test

i.e. techniques that impinge on other indicator species and are not confined to a single target species. It is better to have a range of techniques and species rather than attempt to base results on a single measure to answer such a complex question.

Choices of species

Dab, Limanda limanda, was chosen for much work in the North Sea because it is readily available and because it has a high disease prevalence. It was not recommended, however, as the only animal but rather as an example of the reasoning leading to the choice of species.

The major criteria for the animal or animals chosen should be its (their) availability, the list of phenomena or abnormalities associated with them, coupled with the basic understanding of their biology and movements; sessile or limited range species were considered preferable (e.g. invertebrates, flounder, etc). It was not considered necessary for all scientists to use the same species, but, it was considered essential that approaches and methodology be standardised to permit comparisons of data.

Area considerations

Since there are no ready means whereby disease studies can be used as evidence of pollution it was considered that the best strategic approach would be to build up baseline disease data as a measure of biological information on populations thereby providing the means to determine departures from or return to "normality".

Repeated references were made to the desirability of developing 5 year time series based upon 30 minute squares over the ICES area as has been done by P. van Banning. It was recognised that this is a mammoth undertaking; several members of the Workshop doubted whether it would be possible to carry it out.

Several suggestions were made in the discussion which followed:

(1) Would it be possible to divide the areas such as the North Sea, Baltic etc with separate portions being done by individual nations using agreed standard methods?

(2) Carry out wide ranging 5 year baseline studies first and then with the assistance of appropriate statistical analyses devise a plan whereby sampling would be reduced to a minimum to maintain adequate coverage of stocks in relation to hydrography and migration.

(3) Discrete stocks of animals should be considered the basic unit rather than geographical area. It might be possible to choose particular stocks as indicators for particular species. This will require close cooperation and collaboration with population dynamicists especially in the planning stages.

(4) International calibration of methods is a prime requisite for all disease surveys.

Cruise type

It was agreed by all those involved in collecting disease data on cruises that the ideal situation was to have cruises dedicated to disease survey work. The worst option was to have the work done as an

added requirement for untrained observers engaged primarily or almost exclusively in stock assessment work. Unless at least a portion of the cruise was dedicated to the disease objective it has not been possible to obtain more than minimal fullfilment of the objectives. Many reasons were given such as an understandable absorption with the primary work of the cruise, lack of training, the cruise not being directed to areas of disease interest, lack of time etc. A hierarchy of cruise types was developed and is given below in descending order of desirability and effectiveness.

(1) Dedicated solely to disease studies and involving only trained observers.

(2) Combined cruises with split objectives e.g. disease and pollution or disease and stock assessment. A portion of the cruise would be dedicated to the objectives of each party.

(3) As an add-on to stock assessment cruises.

Target organ

It was agreed that the primary targets should be those where the gross pathology would be easiest to detect and accurately diagnosed. Internal examination should be limited thus the list should contain:

(1) Skin

(2) Fins

(3) Gills

(4) Liver

The need was seen as in the Sea-Going Workshop in Disease (Anton Dohrn) to have a wide range of data from which it would be appropriate following analysis to select the best targets. Nothing has happened since that cruise to alter this approach.

Parasites

Since parasites at one stage or another in their life cycles might be more sensitive to pollution than are their hosts the possibility of using these as indices was discussed.

It was agreed that there were sufficient features such as known appearances or disappearances of particular parasites from a fish stock, modifications to reproductive capacities etc which make the proposals attractive. It was recognised that the development of a biological data base adequate to permit interpretation of the results would be lengthy and difficult, but no more so than for diseases generally and possibly far less.

Conclusions

(1) The links between pollution and disease, although tenuous, are considered to be more evident now than previously.

(2) There is a growing recognition that the problem of linking pollution with disease is much more complex than hitherto considered.

(3) There is a growing appreciation that even if the link is proved disease events (abnormalities) probably cannot be used as direct indices of pollution, but rather as the basis for general statements on the quality of the environment.

(4) There is a need for extensive data bases i.e. readily observable conditions measured annually (two seasons) over a 5 year period and plotted on 30 minute squares (ICES statistical grid).

(5) Once data bases have been built for the broad areas statistically designed plans of a lesser magnitude must be devised to monitor the situation.

(6) Specific pollution related studies should be encouraged in contaminated areas with suitable indicator species for the area(s) concerned.

(7) The main aim should be the use of change in disease prevalence rates as an indicator of the health of the environment.

(8) The interpretation should be based on stocks rather than solely on geographical areas.

(9) Internationally agreed and calibrated standard methods should be applied.

(10) Work should be concentrated on juveniles and adults since studies on larval forms in general are unlikely to yield useful results.

(11) Certain results could be followed-up in experimental studies.

(12) Dedicated cruises for disease studies using trained observers are best, followed by combined cruises also using trained observers.

(13) It was noted that with one exception all participants of the Workshop were drawn from the disease field. It must be emphasized that if fish studies are to be designed to gauge the link between pollution and disease then pollution must form a major part of the considerations. Thus pollution and population experts must be involved in the planning process and any subsequent evaluations and interpretations.

Based upon the foregoing it was considered profitable to continue work on disease studies in relation to pollution, but with a changed perspective to capitalise on and exploit the knowledge gained in recent studies and the new concepts developed as a result. The following recommendations are put forward to reflect the conclusions arrived at by the Workshop.

Recommendations

It is recommended that:

(1)A. Knowledge of background levels of disease should be obtained from long term (e.g. 5 + years) baseline studies on a regular basis over broad areas using dedicated or combined cruises staffed by trained observers using internationally agreed approaches and methodology.

a) The interpretation of results should be based upon stocks rather than solely on geographic areas.

b) Target organs should include skin, gills, fins and liver.

B. Specific investigations on diseases in relation to pollution should be carried out in hot spot and reference areas.

(2) A Second Sea-Going Workshop should be convened under the auspices of the WGPDMO in 1987 or 1988 to discuss and calibrate improvements to methodology developed since the First Sea-Going Workshop, 1984, and member countries are requested to provide ship-time.

(3) Arising from the present stage of knowledge of fish parasites the potential for the use of ectoparasites as indicators of environmental changes, including pollution effects, should be explored as a promising new approach.

Workshop on the use of pathology in studies of the effects of contaminants.

AGENDA

Monday 21 April, 1986

9^{30} Opening and welcome
Aim of Workshop
Rapporteurs
Changes in Agenda

Introductionary reviews:
a. Studies of pathology in relation to pollution. Present Status.
Coffee break
b. Problems of sampling, statistical design and interpretation of results in pathology/pollution studies.
General discussion
Lunch

13^{30} Short communications (max. 15 min.) of the participants own experiences regarding the above-mentioned minutes.
General discussion.

Tuesday 22 April, 1986

9^{30} The use of pathology in programmes to monitor the effects of contaminants.
Species, age and target organs of fish.
Coffee break
Generalization in between results from different areas.
Lunch

13^{30} General discussion
Further activities and Recommendations.

ANNEX 2

Participants at the ICES Workshop on the use of Pathology in studies of the effects of contaminants. Dublin, Ireland, 21-22 April, 1986.

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ANNEX 3

Workshop on the use of Pathology in Studies of the Effects of Contaminants

Studies of pathology in relation to pollution
- Present status -

Volkert Dethlefsen

I. Introduction

Members of the ICES community interested in diseases of marine fishes belong to two different groups. First those who are in favour of a connection betweeen diseases and pollution, and second those who are against. Both groups agree to the basic principles which are repeatedly and convincingly outlined by Sindermann (1983 and 1984): Stress can provoke fish diseases (infectious and non-infectious). Stress is the sum of all physiological responses by which an organism tries to maintain or re-establish a normal metabolism in the face of a physiological or chemical force.

This basic principle includes that genesis of disease is multifactorial. It involves environmental stress, facultative pathogens resistance of host and latent infections. Epizootics can be triggered by a complex interplay of pathogen environment and host population.

In his 1984 paper Sindermann lists eight different biological and ten physiological chemical factors which either singly or combinedly may act on fish populations resulting in a stress which can be the cause for a disease. Disease therefore has to be understood to be an unspecific response toward all kinds of stress.

In the literature there are some undisputed examples for a relationship between pollutants and diseases in certain areas. Amongst these are the occurrence of ulcerated fish in connection with the Amoco Cadiz-wreckage (Balouet and Baudin-Laurencin, 1980), fin rot in flat fish of the New Bight (Murchelano and Ziskowski, 1979), fin erosion and liver conditions in fishes from three coastal regions of the United States (Sherwood, 1982), black

spot disease of crustacea of the New York Bight (Young and Pearce, 1975), black gill in crustaceans of the New York Bight (Sawyer et al., 1983), the ulcus syndrome in cod Gadus morhua in Danish coastal waters (Jensen, 1983).
The most advanced study to detect cause effect relationships between pollution and fish diseases are done in the Pudget Sound by Malins and his co-workers. They conclude that statistically significant correlations between chemicals in sediments and hepatic neoplasms in bottom dwelling fish appeared to be indicative of a general cause and effect relationship. However, such an association should not be interpreted as de facto evidence of specific cause effect (Malins et al., 1981; 1983; 1984).

II. Disease studies in the ICES area.
The debate within ICES mainly centered on diseases of fishes in the North Sea, where studies by Möller (1979; 1981), Dethlefsen (1978; 1984), Dethlefsen and Watermann (1980; 1982), Dethlefsen et al. (1984) provided information on high rates of external diseases on dab (Limanda limanda). The diseases described were lymphocystis, ulcerations, epidermal papilloma and hyperplasia and others. They were described to occur in relatively high frequencies and it was the statement of Dethlefsen and Watermann (1980) that high disease rates within the centre of the German Bight might be related to pollution, especially to wastes from titaniumdioxide production, which were dumped into an area which was characterized by high disease rates.
Following these initial papers studies on occurrence and abundance of diseases of marine fishes have been carried out in the Thames estuary in the 1980s by Bucke and others (1983a). Wootten et al. (1982) investigated disease conditions of fish in Scottish waters. McArdle et al. (1982) investigated disease frequencies in Irish waters. Authors of the last three papers considered their material too preliminary to finally state whether pollution could influence the occurrence of diseases of the fishes investigated.
But there are interesting remarks in these papers, for example there is a statement in the paper of Wootten et al. (1982) where

highest prevalence rates of dab afflicted with papilloma occurred in two hauls in the outer Firth of Forth at a sewage sludge dumping ground and in the Firth of Murray. Both areas are considered to be polluted. In the paper by McArdle at al. (1982) it was found that higher disease prevalences occurred at sites inshore. High prevalences of three diseases of dab were found at a station which is known to be highly contaminated with copper. Also in the study of fish diseases in the outher Thames estuary (Bucke et al., 1983a) disease rates especially of flounder were quite high. Fin rot for example reached to a maximum of 13.5 %. Nevertheless, the conclusion of the authors is that they have no evidence that the incidence of fish diseases is affected in the United Kingdom most important dumping area for U.K. sludge. The major shorthcomings of these studies is that they are not based on repeated sampling and that they neglect seasonal and annual variability which can be quite high. So that the conclusions of these papers that no correlation was found to exist between increased disease rates of dab for example, and the pollution of the respective areas is not sound enough. Systematic studies have been carried out by Danish, Dutch and German workers and the results of these groups will briefly be highlighted in the following part of the paper.

1. Danish investigations

Mellergaard and Nielsen started their investigations in May 1983 and performed two cruises per year. They concentrated on diseases of flat fishes. Their results are to be found in ICES papers (Mellergaard and Nielsen, 1984a; 1984b; 1985a; 1985b). Their main emphasis is on regional distribution of external diseases and some parasites and on the impact of the diseases on growth and condition of the fish. They find a relatively high variability of disease prevalences in the various areas covered. Highest frequencies were found for lymphocystis on dab followed by epidermal papilloma and hyperplasia. Areas with higher disease frequencies were consistently found to be located in the German Bight and in the 1984 and 1985 papers the following sentences

can be found: the increased frequencies in these areas are possibly due to the effect of environmental stress (1984) and the impact of pollution on fish in the German Bight may result in less resistance against infections and therefore aggravate the course of the disease dealing with epidermal ulcerations (1985).

2. Dutch investigations

Van Banning et al. (1984) studied the contamination of eels (Anguilla anguilla) from Dutch coastal waters in relation to diseases. No direct relationship between diseases and contamination was detected but a possible indirect effect of pollution high rates of bacterial infection of blood of eels (80 %) from the polluted areas were contrasted by low rates (4 %) from the reference area.

This finding is interpreted as possible indication for an indirect effect of pollution.

Vethaak (1985) investigated 15,000 fish in 1983 and 1984 for the occurrence of tumours, pseudotumours, ulcers, fin rot, skeletal deformities and disorders of internal organs. The stations were located at the Dutch coast and were selected based on knowledge concerning the degree of the pollution. Disease prevalences from areas with lesser degree of pollution were compared with areas of higher pollution. Flounder was the fish most frequently afflicted with internal/external disorders. Internal diseases ranged to 5.8 % and external to 18.7 %. The results of this survey show that the increased occurrences of lymphocystis, ulcers and fin rot on flounder around the coast of the Netherlands may be related to pollution. The occurrence of flounders with tumours and liver nodules in the coastal zone may also support this. This conclusion is also drawn despite the fact that no chemical data have been obtained during the study of Vethaak. In the case of dab and plaice the observed disorders and their regional distribution gave no reason for suspecting a link with marine pollution.

3. German investigations

Two groups carried out studies in the southern North Sea.

A. Möller

Möller carried out studies in February 1977, 1978 and August 1978 and 1980 (Möller, 1979; 1981). In his first three cruises he investigated diseases of flatfish in the southern North Sea and in August 1980 he concentrated on German and Danish coastal waters. He found relatively high disease rates of dab afflicted with lymphocystis, ulcerations and epidermal papillomas. His conclusions in 1979 were that malnutrition is supposed to induce relatively high rates of ulcers and lymphocystis in dab in the central North Sea. Water pollution possibly favours the increase of ulcer disease in cod from certain localities in the Baltic Sea, of cauliflower disease in Elbe eels, and of fin rot in dab from the German Bight dumping area. Effects of anthropogen pollution, if any, on fish diseases in the open North Sea are masked by more significant natural conditions. In the context of his studies no chemical invetigations were done and no evaluation of pollution situations in the areas investigated is presented.

B.1. Dethlefsen

These studies were started in 1977 and are still under way. More than 200,000 dab (Limanda limanda) were investigated amongst other fish species for the occurrence of external deviations. Some of the papers published in the context of these investigations are Dethlefsen et al. (1984) reporting on sources of variance in data from fish disease surveys; results of histological studies of disease phenomena in dab and cod are given by Watermann (1982), Watermann and Dethlefsen (1982), Watermann (1984); epidemiology of pseudobranchial tumours of cod in the North Sea by Watermann et al. (1982); Watermann and Dethlefsen (1985) provide information on epidermal hyperplasia and degenerative changes in skin of gadoid fishes in the North Sea; Dethlefsen (1980) gives more general information on occurrence of diseases of cod, dab, plaice and flounder; information on diseases of dab are further given by Dethlefsen

(1984) and Dethlefsen (1985a); Wolthaus (1984) investigated seasonal fluctuations of disease frequencies of dab in the German Bight based on 180,000 dab; Dethlefsen and Huschenbeth (1986) provide information on organochlorine contamination of dab in the southern North Sea; Dethlefsen and Knust (1986) describe the influence of x-cell in gills of dab on condition and growth of the fishes; Dethlefsen (1985) reviews investigations of effects of dumping of wastes from titaniumdioxide production in the German Bight on the contamination of biota and diseases of dab.
In their epidemiological studies Dethlefsen and his co-workers considered amongst others the biological factors food, migration, net injuries, and chemical factors like concentrations of iron and manganese in seawater, of various heavy metals in sediments and of residues in biological material including organochlorine substances and chromium.

B.2. Findings in the dumping area for wastes from titaniumdioxide production.

Dethlefsen and Watermann (1980) found increased incidences of dab afflicted with epidermal papilloma in the dumping area for wastes from titaniumdioxide production in the German Bight. Their conclusion that a correlation might exist between wastes and the diseases is supported by additional material given by Dethlefsen (1985), where the data were re-analyzed using a statistical procedure which considers different lengths and sexes of fishes in the area. In this study it was shown that increased concentrations of heavy metals in the water column and in sediments of the dumping area were clearly measurable and also the contamination of dermal tissue of dab with chromium which is one of the main heavy metal compounds of the wastes was increased in fish from the dumping area as compared to reference stations. There was a positive correlation between contamination of livers of dab with chromium and the size of epidermal papilloma of fish from the respective stations. His findings are interpreted to be circumstantial evidence for a correlation between diseases and wastes in the dumping area.

In a final study Dethlefsen et al. (1986) concluded that biological factors, like migration, spawning, food, condition and population density cannot be responsible for regional differences of incidences of epidermal papilloma of dab in the German Bight.
For ulcerations and lymphocystis it was found that net injuries or impact of fishing gear could have an influence triggering these diseases in certain areas of the North Sea. For ulcerations it could not be excluded that differences in the migration of healthy and diseased fishes existed. Biological factors, like spawning, condition, food and population density, were not found to be responsible for regional differences in disease frequencies.
Another study in the German Bight was done by Stork (1983) on ulcerated cod (Gadus morhua). Comparison was done on two groups of otherwise identical cod (one group healthy, the other one with obvious external signs of ulcerations) with regard to their residues of PCB. It was found that cod afflicted with external ulcerations contained higher levels of PCB than their healthy cohorts.

III. Conclusions

It is evident that the fraction of scientists within ICES who see correlations between the occurrence of diseases of fishes in the North Sea and pollution is increasing (Dethlefsen and co-worker, Vethaak and Mellergaard and Nielsen). Workers who cannot see the correlation between pollution and diseases in their studies are, with one exception, living on the other side of the North Sea (Bucke, Wootten, McArdle and their co-workers). The exception of course is Möller of Germany.

Chemical factors in relation to the epidemiological studies were only investigated by Dethlefsen and his group. In the other studies only epidemiological data are provided and reference is made to the assumed degree of pollution of the areas covered. So the basis for rejection or exception of a connection between water pollution and fish diseases is relatively weak. But also the evidence provided by Dethlefsen is circumstantial, not allowing to draw conclusions on cause effect relationships. It is further striking, that those investigators who performed prolonged systematic studies with repeated samplings including high numbers of fishes investigated were more positive to a correlation between diseases and pollution than those who did single surveys, for example the British colleagues. It is needless to say that the representativeness of single surveys is rather limited taking into account the high seasonal and annual variability of the incidences of the diseases under study.

One of the astonishing and hitherto unexplained fact was that diseased fishes are not restricted to occur close to the coast which would have been expected following the traditional thinking that pollution is high in coastal onshore areas and is decreasing with increasing distance from the coast.

So the fact that high disease rates of dab, for example in the Dogger Bank area, which is located in the central southern North Sea with maximum distance from estuarine inputs is taken as an argument against the hypothesis that diseases are linked to pollution. It is assumed that fishes occurring in these

areas should be less contaminated than their onshore or estuarine cohorts.
We therefore investigated organochlorine contamination and heavy metals in livers of dab in the southern North Sea and found that the contamination of livers of dab with lead and cadmium was highest in fishes from the Dogger Bank area as compared with fishes from the southern North Sea. This is an astonishing result which should be taken into consideration.
Also the contamination of offshore fishes with organochlorines was sometimes higher than that of onshore specimens.
Regarding the contamination of sediments, with heavy metals for example, it was found that stations far away from estuarine inputs were characterized by higher heavy metal concentrations than the onshore stations. There are further examples in the literature showing that the assumption, that pollution is restricted to coastal areas, is wrong. Of course, these results do only indicate a coincidence of the occurrence of higher disease rates of fishes and areas of high contamination a cause effect relationship is not proven by these findings. One of the requirements often heard, for example in statements of the ACMP of ICES, is that experiments should be carried out to prove the potential of various contaminants to produce the diseases under study. Reference is made to an extensive literature which is partly summarized by Meyers and Hendriks (1982), where present knowledge on experimental studies to produce diseases using various toxicants is given.

So there seems to be no need for further experimental work to demonstrate the potential for various substances to produce diseases.
Additional experimental evidence would not help to enlighten a highly complex interactive situation in the field.
Most of the statements on causes of diseases of marine fishes heared in the past were simplifications, both of those who were

in favour of a correlation between pollution and diseases, one of those who rejected this connection. It is at present agreed that high disease frequencies are the result of a complex interplay of natural and anthropogenic factors, biotic and abiotic, where pollution plays a certain role. Since pollution has the potential to impose stress on organisms and since a certain degree of likelyhood exists that pollution might be causative the monitoring of fish diseases is one task for the future and the reduction of the pollution another.

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ANNEX 4

PROBLEMS OF SAMPLING, STATISTICAL DESIGN AND INTERPRETATION OF RESULTS IN FISH PATHOLOGY/POLLUTION STUDIES

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Recent studies in pathology have led to controversial hypotheses concerning the effects of pollutants on the health status of marine life, especially on fish health. To my opinion this is first of all not a methodological problem due to methods of sampling or of statistical design, but a problem of the interpretation of results. The main problem in this context seems to be a rather basic one and therefore I will start with a more detailed introduction to the subject.

Pollution in its basic sense is the disposal of anthropogenic wastes. The production and the disposal of wastes is a natural phenomenon in any species. It inevitably leads to a change in the ecosytem and consequently affects its inhabitants, some of them in a positive, but most of them in a negative way. In this respect, man differs in some important points from other highly developed vertebrates: (1) He may produce more wastes than necessary for his survival and welfare, (2) he is able to realize this, and (3) he knows about most of the consequences.

The marine ecologist automatically becomes involved in pollution problems. Man, as being an intelligent species, has realized two important facts: It is extremely favourable for his health to dispose his wastes far away from his

living places, and, this will happen automatically, when he throws them into a river which, obeying to natural laws, brings them out of his reach. More favourably, most rivers empty into the sea, offering a large potential of dilution.

In this respect it is interesting to note that in the English as well as in the French language proverbs can be constructed like "The solution of pollution is dilution" or "La solution de la pollution c'est la dilution". The same is impossible in Netherlands, German or any Scandinavian language. I am not sure if this has any deeper meaning, probably it is a mere accident.

Problems caused by marine pollution and aspects of environmental protection of the sea have gained increased interest among scientists as well as in the public since the mid of the 1970s. However, the intensity of this discussion is quite different in different parts of the world. It is interesting to note that among the ten leading fishing nations (Japan, USSR, China, USA, Chile, Norway, India, Korea, Thailand, Indonesia) the USA are the only country, where marine pollution problems are a major subject of present ecological studies. Another regional focus of recent marine pollution studies is the North Sea, but there the pollution problem is jugded quite differently by different countries, in the official governmental opinions as well as in the public opinions.

This unclear situation is typical for a young field of an ecological science. It is due to scientists at the moment being unable to make clear and incontestable statements. However, one point is incontestable: The present state of pollution of an area is a fact, as it is the composition of the seawater or the age structure of a fish population. If two different descriptions of this fact are given, there are only two valid explanations: either one is right and the other one is wrong, or both are wrong.

However, the interpretation of the effects of this fact is open to scientific discussion. The quality of this interpretation will depend on the knowledge and the honesty of the scientists.

Honesty of a scientist in this respect means two things. One is the use and open presentation of adequate sampling methods, of reliable laboratory

procedures, and of adequate statistical treatment of data. The second point is a more personal one. It concerns the ideal position of the scientist. Every scientist working on pollution problems clearly should identify his position: Does he want to be a pure scientist whose ambition is exclusively the understanding of ecological processes being influenced by man? Is he an advocate of nature by trying to find arguments to reduce waste disposal? Or is he an advocate of a group that is responsible for waste disposal? In our society it is not regarded dishonest to act as an advocate, but this position is different from that of a pure scientist.

The task of an advocate is to find arguments that support only one party in a conflict situation. In contrast to him, the pure scientist has to identify and describe the problem, for example by building up a working hypothesis, and then to study arguments that support and contradict this hypothesis. A scientist who classifies himself as a pure scientist and who does not include contradicting arguments into his considerations, is dishonest. A number of examples could be cited of authors who are experts in selective and one-sided citations of conform literature only.

An ecologist who acts as an advocate of nature automatically makes himself an opponent of an advocate of economic interests as nearly any kind of nature protection needs money. This will bring him into a weak position as the arguments of ecologists and economists always are of different quality. The economist usually will be able to calculate with figures, to bring reproducable results and to demonstrate social and economic consequences of his findings. His results can be recalculated and checked. The ecologist on the other hand hardly can present any more than hypotheses as an ecosystem usually is too complex to be quantified to a reliable degree. His statements are largely influenced by his experience and his understanding of ecological processes. Consequently, his statements cannot be recalculated and only future will show if they are right or wrong.

To believe in someone's experience and "ecological understanding" is a venture. If an ecologist on this basis expects a politician to trust his hypotheses, he first of all has to assure that he acts as a pure scientist and not as an advocate of any party.

Most persons who have studied an ecological science in conflict situations tend to act as advocates of nature. This behaviour is understandable, but it is not very helpful as it usually leads to an intensification of the conflict. In lack of knowledge of political economics the average ecologist is not able to answer questions like: "How much nature conservation do we need?" or "How much pollution can we accept?" On the other hand, the average economist is in the same situation due to a lack of ecological understanding. The answers to these two questions can only be given by the politician who represents the community as a whole and who will be made responsible for the consequences of his decision. A wise decision, however, does not only need a wise and honest politician, it primarily needs sufficient and honest information. The duty of the economist as well as of the ecologist in this context is the preparation of such information.

It is not a fault of the ecologist if his complicated study subject does not allow more than the presentation of a hypothesis. This is the professional risk of the politician who has to build his decisions partly on these hypotheses.

I draw two conclusions from these thoughts:
(1) Man produces wastes. These wastes have to be disposed. This disposal in general is detrimental for the environment.
(2) The ecologist is not the right person to decide whether a pollution policy has to be changed or not. His task is to demonstrate the present state of pollution effects, to hypothesize what will happen if this policy will be changed, and to bring these findings to the attention of the politician.

What does this mean for the marine ecologist studying pathology/pollution problems?

The sea is one place where human wastes are disposed and, consequently, where the environment is damaged. One aspect of this damage is the negative influence on fish health. It is known from a large number of experiments that organic as well as inorganic substances may induce or trigger the outbreak of various diseases of fish health in captivity. The presence of heavy metals or pesticides in the water may increase the deformity rate

of hatching larvae (BENGTSSON 1975, COUCH & al. 1979, OZOH 1979, WEIS & WEIS 1976), the presence of copper is suspected to induce vibriosis (ROEDSAETHER & al. 1977), the uptake of aflatoxin-contaminated food leads to the formation of liver carcinomas (GHITTINO 1976, TAKASHIMA 1976), and crude oil may induce fin rot (GILES & al. 1978, MINCHEW & YARBROUGH 1977) and tissue abnormalities in various organs (GARDNER 1975, McCAIN & al. 1978, SOLANGI & OVERSTREET 1982). Results from fish-carcinogen tests in the United States recently were reviewed by COUCH & HARSHBARGER (1985). There is no reason to doubt that similar effects will occur if fish in nature are exposed to pollution levels similar to those used is the experimental studies.

It makes no sense for a pathologist to ask the question whether the introduction of a pollutant into the sea is harmful for the health of some of its inhabitants or not. Certainly it is - this statement needs no scientific activity. The important question to answer is whether the pathologist is able to identify pollution as a cause of the outbreak of a disease or of an increased disease prevalence in a natural population.

The proof for this cause-and-effect correlation has to be brought in two steps: The first step is to demonstrate a gradient in disease prevalence being correlated with a gradient in pollution. This gradient may be demonstrated regionally by comparing areas with different levels of pollution or temporarily, surveying one area where the pollution level changes as time goes by. The second step is by far more difficult than the first one: It is the exclusion of pseudocorrelations.

Some examples from recent studies in fish pathology from the Elbe estuary demonstrate how easily one might be misled by such pseudocorrelations. The Elbe River is heavily polluted by various kinds of chlorinated hydrocarbons, mercury, and communal wastes, regularly leading to oxygen deficiency. Due to the location of the main emittants and due to dilution processes, the concentration of most toxicants is decreasing from the Hamburg-Stade area towards the North Sea (Fig. 1). If a measurable impact of pollutants on fish health exists, one would expect increasing disease prevalences from the North Sea towards Hamburg. In some instances this seem to be the case:

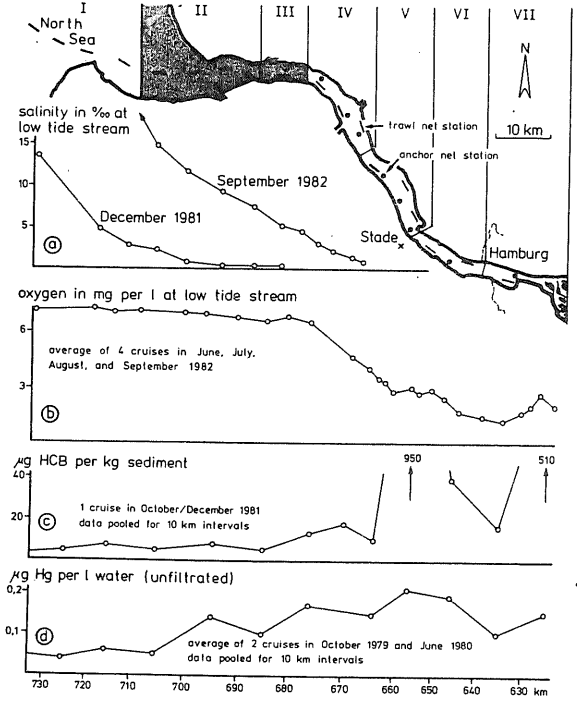

Fig. 1. Regional fluctuations of selected hydrographical parameters, pollutants, and of various data on fish biology from the lower Elbe River.

	I	II	III	IV	V	VI	VII
DISEASE RATES IN % (e)							
SMELT 14 cm Feb - Nov 1982							
pharyngeal granuloma	5.7	6.9	6.8	6.6	6.5	6.0	5.0
spawning papillomatosis	0.2	5.1	6.5	3.5	4.2	3.0	4.2
deformities of operculum	1.0	1.0	0.9	0.9	1.4	1.4	1.2
deformities of lower jaw	0.8	1.4	1.7	1.7	1.8	1.9	1.5
FLOUNDER 18 cm Sep 1981 - Sep 1982							
skin ulceration	1.2	2.4	1.5	0.4	0.3	0	0
lymphocystis	1.2	2.2	2.3	1.6	0.3	0	0.5
fin rot	0.5	0.7	0.4	0.3	0.3	0	0.3
bleaching syndrome	1.0	3.1	0.6	0	0	0	0
EEL 21 - 25 cm May - Sep 1982							
cauliflower tumor	-	12.3	14.2	13.4	12.2	10.6	9.8
CONDITION FACTOR (f) only healthy fish examined							
maximum value = 100							
SMELT	93	90	91	97	100	-	-
FLOUNDER	85	84	84	88	89	92	100
EEL	-	94	97	100	99	99	93
WEIGHT OF STOMACH CONTENT (g)							
maximum value = 100							
SMELT	100	63	48	78	63	66	-
FLOUNDER	66	40	33	← 50 →		← 100 →	
EEL	100	57	46	81	46	41	16
HEAVY METALS IN FLOUNDER (h)							
in mg per kg liver dry weight, average of size groups 15-17 and 20-25 cm							
cadmium	0.12	-	0.24	-	-	0.17	
lead	0.16	-	0.19	-	-	0.11	
copper	21.2	-	27.5	-	-	20.4	
mercury	0.33	-	0.97	-	-	0.55	
POPULATION DENSITY same months as in (e) (i)							
maximum value = 100							
SMELT 12-21 cm, anchor net stations	-	100	52	90	77	26	27
FLOUNDER 12-25 cm, trawl stations	22	31	100	23	24	27	38
EEL 11-70 cm, anchor net stations	-	26	40	31	86	100	33

(1) The prevalence of diseases in flounder increases together with the increasing pollution load of the water from the open North Sea towards the inner Elbe estuary, suggesting a causal correlation. However, as the pollution level increases further upstream, the disease prevalences drop almost to zero (Fig. 1e). Tidal fluctuations in salinity are thought to be the dominent causative factor. They prevent the establishment of larger benthos communities, thus leading to starvation of benthos-feeders (Fig. 1f). A higher susceptibility to pathogens is the consequence.

(2) The highest residues of cadmium, lead, copper, and mercury in flounder liver are found in the central estuary, where the highest prevalences of skin ulcerations, lymphocystis, fin rot, and bleaching syndrome are registered. At first glance a cause-and-effect relationship seems to be existing, but at closer look it is more likely that these two facts only have the same reason without being directly related: Starvation on one side is supposed to be the cause of increased susceptibility to pathogens, on the other side it leads to a reduction of reserve substances in the liver and, consequently, an overall increase of heavy metals per unit liver weight (Fig. 1h).

(3) In the summer of 1982, half of the cod population from the Elbe estuary showed externally visible skeletal deformities, much more than ever registered in any other German coastal area. When studying the local cod population for a longer period, it became evident that this high prevalence obviously is not an effect of pollution but a result of affected cod not joining the migration of the healthy members of the population (Table 1).

(4) Mainly during early summer, flounder with hypertrophied gill lamellae (see Plate 85 in MÖLLER & ANDERS 1986) are found in and close to Hamburg harbour. These lesions could be interpreted as being pollution-induced, however, in reality they are attachment sites of parasitic copepods that have dropped off the gills of the fish after immigration into the freshwater region of the river in spring (GERCKEN 1982, MÖLLER 1984b).

These are only four examples from my own working group. I am sure that additional cases from other areas could be found as well. The identification of cause-and-effect correlations and their separation from such pseudocor-

Table 1. Occurrence of externally visible skeletal anomalies in cod (7-20 cm) from the Elbe estuary over a 12-month-period.

month	healthy fish caught per hour	deformed fish caught per hour	percentage of deformed fish
Sep 1981	621.9	2.2	0.4
Oct	422.5	1.6	0.4
Nov	400.7	1.5	0.4
Dec	206.5	1.4	0.7
Feb 1982	170.5	2.6	1.4
Mar	196.8	1.5	0.8
Apr	211.9	2.6	1.2
May	166.0	2.0	1.2
Jun	3.1	2.8	47.5
Jul	0.6	0.7	53.8
Aug	0.8	0.3	27.3
Sep	41.7	0.5	1.2

relations is a central problem in pathology/pollution studies. In our case it is identical with the separation between anthropogenic and natural parameters causing regionally or seasonally increased disease prevalences.

The solution of this problem is not a matter of sampling methods nor of statistical design, nor of data treatment. It primarily is a matter of ecological interpretation. This problem only can be solved if sufficient information on the host, the parasite, and the environment is available.

Table 2. Parameters that are proven or are suspected to influence disease prevalences in free-living fish.

stock-specific parameters	environmental parameters
a spawning habits	a environmental conditions during the egg and larval phases
b population density	b salinity conditions during the adult phase
c fish migration	c temperature conditions during the adult phase
d fish length	d selection pressure
	e nutritional condition
	f parasites
	g food
	h fisheries
	i pollutants

For to identify what kind of additional information will be necessary, we have to know what environmental conditions may cause increased disease prevalences. In Table 2 thirteen conditions are compiled. Four of them are host-specific parameters, the others are environmental parameters. In the following chapter some examples will be demonstrated:

HOST-SPECIFIC PARAMETERS

(a) Spawning habits

Spawning can be regarded as a kind of stress, which lowers the resistance of fish to infective agents. For example, relatively high prevalences of lymphocystis were found in Baltic and Elbe flounder during the weeks following the spawning season (ANDERS 1984, VITINSH & BARANOVA 1976). Spawning papillomatosis in Elbe smelt also occurs only during the spawning season early each year (MÖLLER 1984a). WOLTHAUS (1984) found the highest prevalence of epidermal papillomas in North Sea dab during spawning season from March to May in an area which is considered to be a major spawning ground of the species. In freshwater, infection of spawning salmonids with Saprolegnia and of various species with Ciliophora is common (MEYER 1970, ROBERTS 1978). Spawning stress, together with parasite infection, has supposedly caused mass mortalities of American smelt in northeastern American lakes (HALEY 1954, SCARBOROUGH & WEIDNER 1979). Large spawning mortalities without involvement of disease are common e.g. in capelin from north Atlantic waters.

(b) Population density

Infective agents are more easily transmitted at high than at low population densities. Accordingly, accumulation of fish on spawning or feeding grounds, or for any other reason, may lead to higher disease prevalences. In this respect, an indirect effect of pollution on fish disease should be taken into account: Raised temperatures in the vicinity of power plants and organic "pollution" frequently lead to increased benthic production, thus attracting high numbers of benthos feeders (ANGER 1975, SPIGARELLI 1975, MÖLLER 1978).

WATERMANN & al. (1982) found the distribution pattern of pseudobranchial lesions in cod from the German Bight to be positively correlated with the population density of the host. Differences in population density might explain why as many as 8.7 % of cod in the central German Bight were affected by skin ulcers in August 1980, whilst the prevalence was only 0.1 % in coastal waters; about 1,000 cod per unit haul were caught in the first area and only 38 in the second (MÖLLER 1981). The probably virus-induced spinning disease of menhaden only leads to mass mortalities during the season when the fish accumulate in large schools in coastal waters.

(c) Fish migration

High disease prevalences in certain localities may be due to the fact that diseased fish show abnormal migration behaviour. Heavy infection with certain ectoparasites is supposed to have a similar effect (SPROSTON & HARTLEY 1941, GUTHRIE & KROGER 1974).

During the summer of 1982 the prevalence of deformed cod amounted to 54 % in the Elbe estuary, whilst from September to May it was below 1.5 %. An analysis of the population revealed that deformed cod did not participate in the seasonal emigration of the species from the estuary to the open sea, thus leading to an increased prevalence in summer. A similar abnormal migratory behaviour was supposedly the reason for the very high deformation rate of herring in the German Bight during the summer of 1980 (MÖLLER & ANDERS 1986).

(d) Fish length

The highest prevalences of most fish diseases occur in specific length (= age) groups. Fin rot seems to be a notable exception (Fig. 2). The prevalences of skin ulcers in gadids and flatfishes, and of spawning papillomatosis in European smelt, increase with increasing fish length. The frequencies of pseudobranchial lesions in gadids, lymphocystis in European flatfishes, epidermal X-cell lesions in Pacific flatfishes, papillomas in dab, and cauliflower disease in eel increase up to a certain fish length and

decrease thereafter (ANGELL & al. 1975, STICH & al. 1976, MÖLLER 1981, REIERSEN & FUGELLI 1984). Differing results may be obtained, if a population under study does not include the very old age groups, as for lymphocystis in Fig. 2.

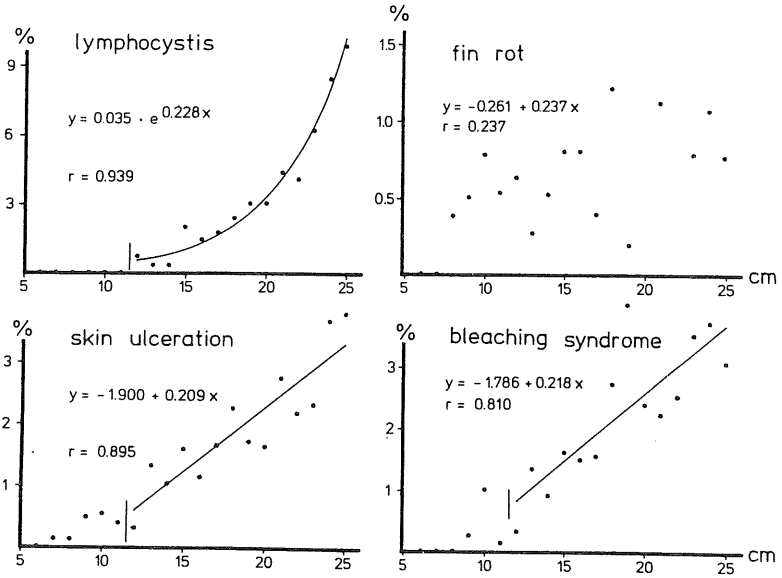

Fig. 2. Relationships between host length and disease prevalences in flounder from the Elbe estuary (region II and III in Fig. 1), September 1981 to September 1982.

In order to detect differences in the regional impact of environmental factors on disease prevalences, preferably fish of similar length groups should be compared. A simple method to correct length-dependent variations in disease rates is described by MÖLLER (1981).

ENVIRONMENTAL PARAMETERS

(a) Environmental conditions during the egg and the larval phase

Extremes in salinity and temperature lead to relatively high malformation prevalences in larval populations, as demonstrated experimentally by BATTLE (1929), GABRIEL (1944), HUBBS (1959), KOO & JOHNSTON (1978), and MOTTLEY (1937). It is assumed that the same effect occurs in fish fry that develop in the outer ranges of the spawning area, thus leading to relatively high deformation rates in adult fish in these regions.

This hypothesis is supported by observations of FEDERLEY (1909), KÄNDLER (1932), and LUNDBECK (1928), who found increasing malformation rates in flatfish and cod from the western Baltic Sea towards the limits of their distribution in the eastern Baltic Sea. An uncommon spawning of cod in the Sea of Riga outside its usual spawning grounds in 1935 is thought to have been the source of high deformation rates of adult cod seen in this area some years later (BERZINS 1943).

(b) Salinity conditions during the adult phase

During the summer of 1980, the highest prevalences of flounder diseases in the Weser River (southeast German Bight) were found in the central estuary, where high tidal fluctuations in salinity are common. Disease rates were considerably lower in the more stable freshwater area upstream and in the nearly marine area downstream (MÖLLER 1981).

A similar but more pronounced situation was described for lymphocystis, skin ulcers, fin rot, and bleaching syndrome of Elbe flounder. Individual disease prevalences in the transition area were more than twice as high than in the outer estuary and at least three times higher than in the freshwater region. Similar observations in other fish were made for diseases supposedly caused by viruses or microorganisms such as cauliflower disease of eel and spawning papillomatosis of smelt, but not for skeletal deformities (Fig. 1e).

(c) Temperature conditions during the adult phase

The frequency of nearly all fish diseases is influenced by the water temperature. However, under natural conditions, this relation is frequently masked by the effects of other parameters. In the sea, temperature-dependent disease rates will fluctuate during the course of the year. In the warmer months, increasing prevalences of skin ulcers in cod (Table 3) and cauliflower disease in eel have been demonstrated from boreal waters. Additional examples are provided by diseases observed in aquaculture (MEYER 1970, ROBERTS 1978, REICHENBACH-KLINKE 1980).

a

month	number examined	disease prevalence in %
Jan	6,270	0.2
Feb	8,301	0.5
Mar	2,923	0
Apr	8,783	1.5
May	648	0
Jun	8,719	3.3
Jul	8,454	3.5
Aug	11,534	4.0
Sep	1,031	1.4
Oct	2,488	0.3
Nov	0	
Dec	10,034	0.3

b

year	number examined	disease prevalence in %
1971	2,222	2.2
1972	4,023	0.5
1973	32	
1974	2,729	0
1975	6,856	3.9
1976	20,472	0.9
1977	19,316	1.6
1978	12,925	2.7

c

length in cm	number examined	disease prevalence in %
11-15	3,687	0
16-20	8,111	0.4
21-25	6,432	1.2
26-30	5,782	1.7
31-35	5,720	2.0
36-40	3,330	1.9
41-50	3,919	1.9
51+	789	4.7

Table 3.
Seasonal (a), yearly (b), and length-dependent fluctuations (c) in the prevalence of ulcer disease in cod from Kiel Bight.

RODGERS & BURKE (1981) described high prevalences of ulcerated mullet Mugil cephalus in Queensland estuaries following rapidly changing water temperature and salinity. Unusually low temperatures are presumed to have initiated skin abrasion in North Sea common sole during extremely cold winter periods (RAUCK 1969). BURRESON & ZWERNER (1984) reported a

high prevalence of the blood parasite Trypanoplasma bullocki in juvenile summer flounder in coastal waters of Virginia. Parasite-induced mortalities were related to water temperature, with the highest rates occurring in the coldest water bodies.

(d) Selection pressure

Disease is a negative selection factor for its host. In areas where predation is low and sufficient food is available, diseased animals have greater chances of survival than under conditions of high predation and food scarcity. This effect can be demonstrated best for fish in aquaculture.

Low selection pressure is suspected to have been the cause of relatively high disease prevalence in Bairdiella icistus in the Salton Sea (WALKER 1961). A small number of breeding fish were introduced to this saltwater lake in 1952. Due to the absence of predators and the availability of sufficient food, explosive population growth took place during the following year. One year later, when competition had increased and the older generation was to some extent acting cannibalistic, the disease rate dropped considerably.

(e) Nutritional condition

A negative correlation between prevalence of certain diseases and the nutritional condition of dab was described from the southeastern North Sea (Table 4). A similar correlation was established for nutritional condition and various external diseases of flounder from the Elbe. From the central estuary towards the city of Hamburg, the average nutritional condition factor increased from 1.06 to 1.25 whilst the total disease prevalence dropped from 8.4 to 0.8 % (Fig. 1e, f).

A low condition factor is usually caused by post-spawning conditions or lack of food. The possible impact of secondary factors seems evident: exclusion of predators may lead to overpopulation, resulting in a shortage of food. The same effect may occur if coastwardly migrating fish are con-

centrated in funnel-shaped estuaries. On the other hand, food resources may suffer from pollution, effects of coastal engineering, extreme ice conditions, heavy storms, and fluctuations in salinity.

Table 4. Condition factors of healthy dab (17-19 cm) and disease prevalences of dab (10+ cm) from stations in the southeastern North Sea in summer 1980.

station	condition factor	skin ulcer	lymphocystis	epidermal papilloma	fin rot
47	1.158	0.20	0.05	0	0.05
50	1.136	1.69	0.45	0	0
49	1.124	1.01	0.38	0	0.21
44	1.123	0.76	0	0	0
51	1.121	0.60	0.40	0	0
48	1.118	1.25	0.16	0	0.25
52	1.117	1.08	0.30	0	0.27
39	1.109	0.79	0.20	0	0
53	1.098	2.64	0.72	0	0.45
38	1.094	0.60	0	0	0
46	1.088	1.71	0	0	0.32
66	1.068	3.34	1.24	0.15	0.36
57	1.055	1.87	0.13	0.11	0
65	1.049	3.62	0.14	0	0
61	1.049	3.04	0.79	0.24	0.57
63	1.048	1.05	0.50	0.03	0.25
60	1.035	3.59	1.09	0.10	3.05
56	1.027	4.05	0.14	0.38	0
59	1.021	2.45	2.18	0	0.27
63	1.015	1.05	0.50	0.03	0.25
64	0.995	3.82	0.54	0.36	0.05
correlation coefficient cond.factor/disease		0.690	0.464	0.620	0.251

(f) Parasites

In natural waters, parasite-induced mortalities are mainly due to the effects of viruses or microorganisms, e.g. as supposed for the infectious pancreatic necrosis (IPN) virus in menhaden (STEPHENS & al. 1980), the bacterium Vibrio anguillarum in eel (MEYER 1933), the fungus Ichthyophonus hoferi in herring and plaice (SINDERMANN 1958, McVICAR 1979), and the protozoan Glugea hertwigi in American smelt (SCARBOROUGH & WEIDNER 1979). High mortalities directly caused by pluricellular zooparasites have been reported only occasionally (Table 5). However, certain species reduce the condition and fitness of the hosts, thereby favouring secondary infections by microbial agents.

Table 5. Some parasitic infections that led to serious damage in free-living marine fish.

parasite species	host fish	area	main effect	source
Ichthyophonus hoferi	Clupea harengus	Gulf of St. Lawrence	mortality	FISH (1934), SINDERMANN (1958)
Ichthyophonus hoferi	Gadus morhua	western Baltic Sea	emaciation	MÜLLER (1974)
Ichthyophonus hoferi	Pleuronectes platessa	north of Scotland	emaciation	McVICAR (1980)
Trypanoplasma bullocki	Paralichthys dentatus	Chesapeake Bay	mortality	BURRESON & ZWERNER (1984)
Eimeria sp.	Micromesistius poutassou	north of Scotland	emaciation	MacKENZIE (1981)
Eimeria sardinae	Sardina pilchardus	Lisbon	castration	PINTO & al. (1961)
Glugea hertwigi	Osmerus mordax	northeastern America	mortality	HALEY (1953)
Glugea stephani	Parophrys vetula	Oregon coastal waters	emaciation	OLSON (1976)
Pleistophora typicalis	Osmerus eperlanus	Elbe estuary	emaciation	see Plate 45
Benedenia monticelli	Liza carinata	Gulf of Suez	mortality	PAPERNA & al. (1984)
Pseudanthocotyloides sp.	Engraulis japonica	southern Japan	mortality	YAMAMOTO & al. (1984)
Poecilancistrium caryophyllum	Cynoscion nebulosus	west of Florida	emaciation	COLLINS & al. (1984)
Pseudoterranova larvae	Osmerus eperlanus	Elbe estuary	emaciation	KLATT (1985)
Lernaeenicus radiatus	Brevoortia tyrannus	off North Carolina	emaciation	VOORHEES & SCHWARTZ (1979)
Peroderma cylindricum	Sardina pilchardus	Tunisia	emaciation	HEDI KTARI & ABDELMOULEH (1980)
Lernaeocera branchialis	Gadidae	southeastern North Sea	emaciation	MANN (1952), MÖLLER (1983)
Lernaeocera branchialis	Melanogrammus aeglefinus	northern North Sea	emaciation reduced fecundity	KABATA (1958) HISLOP & SHANKS (1981)
Lernaeocera larvae	Platichthys flesus	Elbe estuary	emaciation	WICHOWSKI (1983)
Lernaeocera minuta	Pomatoschistus minutus	southeastern North Sea	emaciation	MANN (1965)
Paragnathia formica	Mugilidae	western Europe, northeastern Africa	emaciation	MENEZES (1984)
Cardiodectes medusaeus	Stenobrachius leucopsaurus	California	castration	MOSER & TAYLOR (1978)
Lironeca ovalis	Morone americanus	Delaware River estuary	reduced growth	SADZIKOWSKI & WALLACE (1974)
Emetha audouini	Spicara smaris	Adriatic Sea	emaciation	RADUJKOVIC (1982)

Serious problems may occur when a local fish fauna is exposed to parasites it is not accustomed to. For example, the introduction of the monogenean gill parasite Nitzschia sturionis on Aral Sea sturgeon fry that were transferred from the Caspian to the Aral Sea led to heavy mortalities in the local sturgeon population with disastrous consequences for the fisheries (LUTTA 1941). Certain ectoparasites are known to transmit blood parasites which may be fatal to the host (BURRESON & ZWERNER 1984) or are supposed to transmit pathogenic bacteria (GRIMES & al. 1985).

(h) Food

The role of food in fish pathology stands in a close context with the roles of nutritional condition, of endoparasites that are acquired by eating their intermediate hosts, and of certain pollutants (e.g. aflatoxins, algal toxins) that are ingested with the food.

A surprising observation recently has been made by ANDERS (in prep.) during studying the etiology of tumor-like lesions in the mouth cavity of smelt from the North Sea coast. These pearl-like silver-white nodules reach up to 2 mm in diameter. In a late state they may include virus-like particles as well as fungi. However, initially they are induced by spines from the legs of amphipods that break off in the mouth cavity of the fish (Plates 15, 114-116 in MÖLLER & ANDERS 1986).

(h) Fisheries

Although the proof is still missing, it can be assumed that fishing activities influence the prevalence of certain diseases. Skin damage due to nets and jaw damage due to hooks favour the settlement of pathogenic microorganisms. It is speculated that bleaching syndrome preferably develops in discarded fish with a ruptured integument.

Seriously diseased fish are more susceptible to active fishing gear, thus probably leading to an overestimation of the disease prevalence in the fish population under observation. As another consequence, the disease preva-

Table 6. Mass mortalities of marine fish due to anthropogenic pollutants.

area	time	author	species
Germany: Trave estuary	Mar 1927	EBERLE (1929)	Clupea harengus
Denmark: west off Limfjord	summer 1964	BOETIUS (1968)	Clupea harengus
Netherlands: Scheveningen	Mar 1965	ROSKAM (1965)	several
Canada: Newfoundland, Placentia Bay	1969	IANGAARD (1970)	several
Sweden: Bärsebäck	May 1985	THULIN (1985)	Belone belone (gas bubble disease)

Table 7. Compilation of some surveys where pollution has been discussed as a source on increased disease prevalences in coastal fish by the authors.

lesion	host species	area of survey	source
chromosomal abnormalities	Scomber scombrus (egg)	New York Bight	LONGWELL & HUGHES (1980)
skeletal deformities	Gadus morhua	Elbe estuary	WUNDER (1971)
hepatomas	Microgadus tomcod	Hudson River estuary	SMITH & al. (1979)
hepatomas	Myxine glutinosa	Gullmarfjord	FALKMER & al. (1976)
hepatomas	Pseudopleuronectes americanus	Boston Harbor	MURCHELANO & WOLKE (1985)
lesions of internal organs, incl. hepatomas	Parophrys vetula Platichthys stellatus	Puget Sound	McCAIN & al. (1977b, 1982)
mesenchymal tumors	Mugil cephalus	Gulf of Mexico	EDWARDS & OVERSTREET (1976)
epidermal papillomas	Limanda limanda	German Bight	DETHLEFSEN (1980)
oral papillomas	Micropogonias undulatus	southern California	RUSSELL & KOTIN (1957)
cauliflower tumor	Anguilla anguilla	Elbe River	PETERS (1981)
lymphocystis	Platichthys flesus	Oslo Fjord	REIERSEN & FUGELLI (1984)
gill hyperplasia and others	Pleuronectes platessa	Brittany	HAENSLY & al. (1982)
muscle necrosis	Mugil cephalus	Brittany	BALOUET & BAUDIN LAURENCIN (1983)
skin ulcers	Gadus morhua	Belt Sea	JENSEN (1983)
fin rot	Microstomus pacificus	southern California	MEARNS & SHERWOOD (1977)
fin rot	Pseudopleuronectes americanus	New York Bight	MURCHELANO & ZISKOWSKI (1982)
disruption of ovarian cycle	Pleuronectes platessa	Brittany	STOTT & al. (1983)

lence in the exploited population will decrease. However, the opposite result will be obtained when fishermen reject diseased fish (e.g. flatfish with lymphocystis, eel with cauliflower tumor) back to the sea.

(i) Pollutants

The introduction of pollutants has frequently been the source of massive mortalities of fish in streams, lakes, and occasionally, in coastal areas as well (Table 6).

The establishment of statistically significant relationships between the degree of water pollution and the occurrence of fish diseases is difficult and its interpretation is even more complicated. Several authors have claimed to have found evidence or support for such a relationship (Table 7). However, in several cases other authors, when working in the same locality, with the same fish species, or even with the same set of data, came to contradictory conclusions.

These thirteen examples make evident, that the understanding of the epidemiology of a fish disease does not only need studies in pathology and pollution, but in a large field of ecology, including ichthyology, fisheries biology, hydrography, population dynamics of food stocks, and so on. An overlooking of this demand has led to much confusion, e.g. by the introduction of the term "pollution-associated" disease. This term has been mis-used by a number of authors to describe any kind of disease that was registered in a polluted habitat, regardless whether the disease was also present in fish from non-polluted waters.

How to go on?

I doubt that it will be useful to work out a sort of manual entitled "How to study pollution impact on fish health" for the use in natural waters. I also doubt that it is very useful to work with disease frequency forms as presented in the pathology working group of the ICES during recent years. These forms will cause confusion rather than clearing-up unless

they are more specified. The details of specification depend on the kind of lesion and on the host species. The problem is that we do not know what kind of data are required to use a certain disease as an indicator unless we know the principals of the host's and the pathogen's biology. Usually it will not be possible to collect from the beginning all data that later probably will be needed for an adequate analysis of the disease epidemiology. The explanation of seasonal or regional fluctuations in a disease prevalence usually will need several years of research. During this time the sampling strategy as well as the laboratory work has to be developed and adjusted according to progressing knowledge. This development does not need a rigid manual - it needs ideas and discussions.

About ten years ago the working hypothesis has been erected that fish diseases might be used as indicators of marine pollution effects. In a number of countries this idea has raised considerable interest of governmental institutions. In other words: It was relatively easy to obtain money for studying this problem. It is not surprising that as a consequence many scientists first of all tried to find arguments that support this working hypothesis. In this context many useless discussions have been caused by persons who have mixed two different things. One is the fact that pollution is harmful to fish. The second one is the question whether this harm is so serious that resulting fish diseases may be used as indicators of pollution effects.

To my opinion the time has come to critically consider whether the study of fish diseases should be further applied to monitoring pollution effects or not.

Results from two of the most intensive fish disease surveys, carried out in heavily polluted areas, seem to provide a negative answer. During a five-year- study in the New York Bight, fin rot of winter flounder was the only pollution-associated finfish disease noted (MURCHELANO 1982). During three out of five years it occurred at higher prevalences in the Apex-dumping-area than in the less polluted Sandy-Hook- and Raritan-Bay area. However, despite unchanged dumping loads, the disease prevalence decreased sharply during the period under study (MURCHELANO & ZISKOWSKI 1982). During the survey in the Elbe estuary, the impacts of

various natural parameters on fish disease prevalences could be demonstrated, but not that of pollution. In both areas overfishing and destruction of spawning grounds played more significant roles than the presence of pollutants (MÖLLER 1984b, SINDERMANN & al. 1982).

If it is not possible to demonstrate an effect of pollution on fish health in these two places, the same hardly will be possible in places along the open shore or in the open sea.

In the New York Bight and in the Elbe estuary as well as in most other areas that have been surveyed, pathology/pollution studies were focussed mainly on macroscopically visible external lesions, many of them being induced by pathogenic viruses or microorganisms. These studies have provided scientifically useful information on the complicated relationship between fish, disease and environment. However, few if any examples give evidence that an increased disease prevalence is caused by pollution. I conclude that in the near future these disease types will not be successfully used as pollution indicators.

One might discuss whether other types of lesions will be more suitable, for example the presence of histopathological conditions or of parasites or changes in blood or tissue fluid composition.

The use of fish parasites as pollution indicator is a completely new subject. Surprisingly few steps have until now been undertaken to study the impact of aquatic pollutants on parasites (for review see MÖLLER, in prep.). I assume that this subject will attain considerable more attention in the near future. In regard to the value as environmental indicators, pluricellular parasites offer a number of advantages when compared with pathogenic microorganisms: their biology in general is better known, many of them can be easier detected and determined than disease signs and their causes, and their establishment in a fish as well as their loss take place within a very short time, while the development of disease signs usually takes several days. The composition of an endoparasitic fauna gives a lot of additional information on the biology of the host as well as of the state of food stocks in its environment. And, probably most valuable, ectoparasites are more susceptible to a number of pollutants than fish are. Some of these toxicants are used as antiparasiticides in aquaculture practice.

At present, the histopathological evaluation of liver presently seems to be the most promising way to demonstrate effects of pollution on fish in natural waters. High levels of PCB were supposed to be a probable cause of high prevalences of hepatomas in Atlantic tomcod in the Hudson River estuary, New York (SMITH & al. 1979) and in California sole in the Puget Sound, Seattle (PIERCE & al. 1978). McCAIN & al. (1982) continued the studies in the latter area. They differentiated fourteen principal types of liver lesions, four of which were clearly neoplastic. Although the direct cause of the neoplasms remained unknown, the relatively high prevalences in polluted areas compared to less urbanized sites makes a cause-and-effect relationship between chemicals in the sediment and tumor formation likely (MALINS & al. 1985). Liver cancer was also epizootic in winter flounder collected near a sewage outfall in Boston Harbor, Massachusetts (MURCHELANO & WOLKE 1985).

However, the liver is the most complicated organ of the fish and histopathology is one of the most complicated subjects of biology. Such studies need extremely laborious sampling and processing procedures and well-experienced scientists. This combination will prevent the use of histopathology during routine examinations in most countries.

We should carefully consider what will be the need for future research. The statement that a certain pathological condition is induced by pollution first of all is of academic interest. It shows that we are able to prove what everybody knows: that pollution has a negative effect on our environment. If we are able to find such a prove or not, will depend largely on the quality and the amount of work we can spend. It does not automatically mean that this condition will be a practicable indicator of pollution effects.

What we need is a condition that has the character of an alarm bell. It has to develop rapidly as the pollution situation happens, it has to be clearly related to this pollution effect, and it has to be easily detected to allow routine monitoring programs. It will be difficult to find such a condition in the field of histopathology and pathology of fishes.

For references see:
MÖLLER, H.; ANDERS, K.; 1986: Diseases and parasites of marine fishes. Kiel: Möller, 365 pp. (Parts of the text, figures, and tables in this paper are taken from the same book)

ANNEX 5

ICES "Workshop on the use of Pathology in studies of the effects of Contaminants", Dublin, Ireland 21-22 April, 1986.

The use of fish pathology in programmes to monitor marine contaminants.

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1. Introduction

Criticism of direct chemical measurement of contaminants has often centred on the problems of relating levels found to the effects or dangers in biological terms. It is clear that the ecosystem in general, or individual components of it, may be able to tolerate very high levels of one type of contaminant, but only traces of another and without experimental evidence it is not possible to assess significance. Such evidence, particularly for mixes of compounds is often not available. In addition, because living organisms have evolved a range of tolerance limits to individual components of the environment, only a spectacular departure from natural background levels may result in death or migration and lead to change in the biotic community structure which can be measured. In terms of pollution assessment and control, employment of such changes could be considered to lack sensitivity so that any resulting deleterious effects are detected too late with damage being severe or long lasting. Consequently techniques capable of detecting more subtle, non lethal changes in the structure of marine organisms have been sought. The ICES workshop at Beaufort in 1979 identified approximately 50 techniques which could be appropriate to biological monitoring of marine pollution.The variety of physiological and biochemical parameters in fish considered to be potentially useful all suffer to some extent from their dependence on often sophisticated and time-consuming laboratory based techniques.The use of pathology in pollution monitoring studies was considered attractive as morphological changes would be involved, many of which could be detected in the field and consequently much larger samples of fish screened.The present paper is based principally on personal experience and should be considered complementary to the extensive reviews of the subject by Dethlefsen and by Moller in this meeting. Some of the advantages and disadvantages of using fish pathology as an indicator of marine pollution are considered.

2. Objectives of the study.

Without an initial clear concept of the main objectives of pursuing a study it is easily possible to utilise methods or to investigate diseases/pathologies which are totally inappropriate. At the start of any study it is important to determine why pathology is being investigated and to clearly distinguish applications in monitoring levels of pollution (or quality of the environment generally) from studies on the deleterious effects of pollution.

(a) Monitoring studies. Based on the principle that disease levels

are influenced by environmental quality the objective is to obtain a biologically based index which is sensitive to changes in the environment, which can be measured in the natural population and which persists for some time. In practice this should restrict studies to conditions showing low pathogenicity otherwise affected individuals are selectively removed from the population.

(b) Effects studies. Because of the requirement to concentrate on pathological conditions which impair survival of the individual or of the population (for example by reducing reproductive capacity) appropriate conditions for such studies are difficult to investigate in natural populations as they will have a low prevalence. As it is necessary not only to establish a relationship between the pathological condition and pollution, but also to demonstrate deleterious effects it will probably be necessary, at some stage during such investigations, to complement field data with experimental studies.

Because of incomplete knowledge of the aetiology and epidemiology of most diseases and pathological conditions, often mixed objectives of a study and practical limitations it is not always possible to closely match study aims with an ideal disease condition.

Clearly pathology studies in relation to pollution should not be considered in isolation and must be closely integrated with other pollution index studies both in the biotic (eg benthic community, fish population structure,enzyme analysis) and abiotic (eg chemical analysis) fields. To date there has been a scarcity of such co operative investigations on a meaningful scale.

3. Limitations in the use of pathololgy as a monitoring tool.

Consideration of some of the main shortcomings in using pathology as a tool in pollution effects monitoring may forewarn investigators of some of the pitfalls which could be encountered in interpretation of data. Although more responsive to changes in the surrounding environment than loss of species or populations from an area, pathology does also lack some sensitivity as most obvious conditions useful for monitoring are the end state of often a long sequence of events and changes within the tissues and cells of the organism.To help improve sensitivity, studies could advance beyond simple gross or light microscope observations and consider, at the expense of simplicity,the additional use of electron microscopy, serology and enzyme studies. Because of the limited number of ways in which cells or tissues can respond to surrounding abnormalities, similar or identical pathological changes may be the result of a wide range of environmental or biological phenomena ie. pathology does lack specificity. For example, gill hyperplasia may be the result of irritation by water borne particulate material, chemical insult, external parasitic infection eg Trichodina or internal conditions eg X cells, blood fluke eggs.

In comparison to responses in higher animals the pathological response in fish tissue is remarkable in that generally there appears to be an absence of long-lasting scar tissue after removal of the insult or the cessation of antigenic stimulation. This largely reflects the powers of regeneration of fish tissues, but a consequence is that there is no durable index of pathology.In some circumstances this could be an advantage as the pathology would only be detectable at the time and source of insult but in most cases would be a disadvantage as often an overview of the long term pollution status of an area is being sought.

Pathology, by its very definition, indicates abnormal changes in the tissues of an organism and frequently these changes have deleterious effects. If efficiency of an organism is significantly impaired, in terms of swimming ability, behaviour,camouflage or nutritional status , then affected individuals will be selectively removed from the population. Low prevalence levels of a disease in a population do not necessarily mean a rare disease, but possibly that it is highly pathogenic.Consequently, conditions with high pathogenicity will be difficult to measure in wild fish populations.

4. Potential of using pathology as a tool in measuring pollution.

A large number of anomalies and diseases have been recorded in fish from the North Sea for at least 100 years and current studies have shown that many disease conditions can occur in both 'polluted' and 'clean' localities (note reviews by Dethlefsen and Moller). Present knowledge on the cause and effects of many of these fish diseases is limited but the possibility of using some as indicators of pollution in the North Sea exists in two respects.

(a) Directly. The occurrence of lesions arising as a direct consequence of contact with a single pollutant or group of pollutants may be useful in defining areas of contamination. It is known, particularly from experimental toxicity studies on marine and freshwater fish, that even low concentrations of some pollutants can induce pathologies. However, as indicated, many closely similar pathological conditions may result from insult by a wide range of disparate phenomena. No lesion specifically pathognomic to pollutants has yet been identified in North Sea fish populations, those currently being recorded having unknown or natural aetiologies.

(b) Indirectly. In captive fish populations, eg in fish farms, the susceptibility of fish to naturally occurring disease is significantly increased with decreasing environmental quality (arising from bad husbandry practices or natural events). As indicated by Dethlefsen and Moller in this workshop, investigations into the concept of using abnormally high levels of disease as indicators of pollution have been in progress for several years, principally in European and North American waters. With particular reference to the North Sea it is clear that the range of diseases present, their natural background levels, causes and natural variabilities are insufficiently understood at present to establish links between pollution and differing levels of any one disease. Much of the variability recorded is due to unknown reasons or can be explained in terms of geographical differences , changes associated with the fish host (migrations, spawning, nutrition,age) and other biotic and abiotic features of the natural environment. It is necessary to extend the basic biological data of fish disease and to determine normal base-line levels of disease over a long period before attempting to discern any trends away from these which may be linked with pollution. Because of the major influences of natural phenomena on the levels of fish diseases it is considered that absolute levels of disease within a defined area may be of less significance than trends in disease over a period of time, particularly if there are concurrent changes in the pollution status of the area as measured by other indices. It would be logical, in view of the present state of knowledge, to concentrate on areas of changing pollution status (eg planned new dump sites, abandoned dump sites, areas with new effluent outfalls) in order to establish if any good relationship exists between pathology and pollution.

5. Basic requirements and constraints of a pathology/pollution monitoring programme.

The following comments on requirements and constraints in pathology monitoring studies are largely based on experiences in the Marine Laboratory, Aberdeen by various research workers who have examined fish disease conditions since approximately 1910.

(a) Data base. It is only relatively recently that attempts have been made to consider possible relationships between pollution and disease.However,earlier studies provide the necessary breadth and depth of knowledge and practical experience of the problems of investigating fish diseases. The beginning of an extensive data base was provided by H.C.Williamson who published a report on diseases and abnormalities of fish in the North Sea off Aberdeen in 1913. In the early to mid 1960s Z.Kabata and colleagues carried out a very broadly based study considering a large number of fish species, a large number of specimens and over a wide area to determine what conditions (mainly parasitological) were present. Unfortunately the full results of this survey were not published. Later, H.H.Williams and colleagues made detailed considerations of host-parasite relationships and on factors influencing levels of infection. Principles of epidemiology relating to fish parasites were established and it is clear that these principles can be applied directly to other disease conditions of fish.

Studies in the 1970s on the role of disease in natural mortality in wild fish populations (with particular reference to Ichthyophonus in haddock and plaice) gave an insight into the relationships between prevalence levels,incidence of infection and mortality rates. This is an essential basis for pollution related pathology studies and a similar sequence of background investigations (if only on a restricted time scale and scope) will be necessary to enable logical selection of the most suitable disease condition for study in a particular area.

If data collected by other workers(whether in-house or from other laboratories) is used, accurate standardisation of methods is essential before meaningful comparison of results from different sources is possible. Realisation of this problem led to the I.C.E.S. Workshop on board FRV Anton Dohrn in 1984, the recommendations of which will be published in the near future. Such discussions have significantly influenced aspects of disease monitoring in varous laboratories and continued integration and co operation at the planning, sampling, diagnostic and reporting stages is essential for a meaningful overall picture to be obtained in an area such as the North Sea. Similarly, although it is possible for detailed studies of small areas to be independently carried out, co operation is necessary for cross comparison with other areas to be made.

(b)Resources. The cost of mounting specialist disease cruises and the demands these make on manpower and facilities make it impossible in most laboratories to run such cruises at very regular intervals. Attempts to overcome this have included use of commercial catches because of ready access to landings. This has proved unsatisfactory in the past because of the time elapsed since capture (making material useless for histopathology, and causing the loss of some conditions eg hyperplasia, parasites or difficulty in their identification), often inexact information on the position caught, rejection of small fish at sea and the habitual rejection of diseased fish at sea by fishermen. Consequently, use of routine stock assessment research cruises has been attempted, as has been recommended by ICES, but it was found that attempts to use non-disease experts were inadequate because of commitment to other priorities, degree of training required and the inconsistency of

effort found between different individual staff. Data were only acceptable if a specialist disease worker was included on the research cruise, and this approach was followed during initial broadly based studies. However, when areas,diseases and fish species of particular interest were more clearly defined, use of such surveys was found to be wasteful in time and effort because much of the sampling which was being carried out was of a type and in areas of little interest. For these reasons the preferred option is to use specifically disease cruises staffed by disease specialists, even if this requires less frequent sampling. The need to have confidence in field data particularly when making cross comparisons seasonally, annually, geographically and internationally is stressed.A possible improvement on purely specialist disease cruises lies in the integration with pollution studies so long as the principal objective of the different study groups coincides.

(c)Natural disease variability. Several studies have shown that natural phenomena have major influences on natural levels of disease in a fish population, particularly season, geographical area, inshore/offshore distribution, fish age, maturation, and migration. In fact some of these variabilities are relatively well understood and form the basis for biological tag investigations such as are being carried out by K.MacKenzie in herring and mackerel. Variation in disease due to natural causes is so significant that for pollution monitoring studies either:
(i) a full investigation is made of the natural variability of the disease of interest before the possible contribution of pollution is assessed. This is an ideal situation which would greatly improve confidence in any subsequent statements being made on the role of pollution, but is clearly a major task for any one disease and is often beyond the capabilities of most research programmes.
(ii)the effect of natural variation on the data is reduced by taking as closely identical samples as possible, in terms of season, area, fish species, fish size, diseases etc, over a large number of years and attempting to distinguish abnormal trends in levels. This is the option that many labs will follow. However,such data does not allow close comparison with data from different seasons, areas etc unless the disease pattern is known and is comparable in the different areas.

(d) Relationship between prevalence levels and sample size. When studying fish disease levels two choices exist:
(i) intensive sampling for many disease conditions in a wide variety of organs and tissues. Because of the effort involved it is usually only possible to consider a small number of fish, and only in a limited area Surveys involving histopathological examination of every fish fall into this category.
(ii)extensive sampling for fewer conditions using minimal effort methods in a large number of fish. This approach is essential when conditions with significant pathogenicity are being included in the study, as these may be present with very low prevalence levels and missed in small samples of fish. As the pathogenicity status of many of the conditions at present being considered as potentially suitable for pollution monitoring studies is inadequately known, use of extensive samples has been preferred in most studies.

(e)Choice of disease/pathology. The selection of appropriate disease conditions for study was again extensively considered by the ICES Anton Dohrn Workshop where it was concluded that it was not necessarily the most obviously observed, easily sampled or economically the most important that would be most suitable. Experience has shown that practical considerations and available expertise strongly influence the choice of disease in a particular programme. Grossly observable conditions are preferable as these allow examination of larger numbers of fish, for the same reason dependance on histology and other time-consuming techniques is not

desirable in routine diagnosis (though they may be useful in determining primary aetiology or if increased sensitivity is desired). Conditions selected should be clearly identifiable and not easily confused with other conditions. Conditions of the skin, fins and gills have been identified as potentially the most useful as they require minimum dissection for observation and because of their direct contact with the environment. For intensive or experimental studies, liver (as a centre for detoxification) and haemopoetic tissue have been suggested as useful organs for study and there is evidence that pancreas may be an organ which is particularly sensitive to change, including pollution.

(f) Choice of fish species. It was recognised by the ICES Anton Dohrn Workshop that no one species of fish would probably be appropriate for study in the whole ICES sea area. Certainly it can be seen from existing programmes that local abundance of a fish species, facilities for catching, existing knowledge of diseases, international interest and even national economic importance have influenced the choice of the fish being studied. However, these considerations should not be overriding. Particular attention should be paid to obtaining sample homogeneity by avoiding areas of overlapping populations of fish and to achieve this, good knowledge of the boundaries of different stocks of the fish species is required. If the fish species chosen has a migratory behaviour then clearly information is required on this topic also, and ideally, completely or relatively static organisms would make the best subjects for pollution monitoring/pathology studies.

(g) Age of fish. The survival curve for a fish species normally shows that the proportion of survivors from egg to recruit is considerably less than 1% and that most of the mortality occurs in the early stages of life. The evidence shows that existing fish populations levels are reached as a result of mortality principally due to natural causes such as starvation and predation. Consequently most fish in this age group are destined to die whether or not natural disease or possible pollution induced disease plays a part. If the study objective is to examine the effects of pollution on fish populations these early stages of development are not suitable groups to study. However, it is well established that developing stages of fish are particularly susceptible to environmental change and chemical insult in particular (as is witnessed by their use in chemical toxicity trials and as is seen in fish cultivation), and consequently they should make good material for monitoring of pollution.

Rapid depletion of fish numbers continues in pre-recruit stages of development (10 to 15% per day is common) and clearly the size of the surviving part of the population entering the adult population will be significantly influenced by the rate of loss at this stage. Theoretically any factor which changes mortality rate in this part of the population development could therefore have a major influence on recruited fish stock sizes. If pollution is suspected to contribute to the appearance of debilitating fish diseases, then pre-recruit age groups of fish populations would seem to be the most appropriate to study effects of pollution on a large scale. A significant problem frequently encountered in studying disease in juvenile fish is the low tolerance levels of individuals to damage of any form, resulting in rapid mortality and removal from the population. Consequently, prevalence levels of disease in such groups will inevitably be very low, requiring examination of large numbers of fish in order to establish disease levels. Studies attempting to establish relationships between pollution and disease in wild fish populations have therefore concentrated on older age groups of fish in which disease tolerance levels are higher.

6. Summary.

Fish pathology is potentially useful in monitoring pollution as it measures relatively subtle and often non lethal changes in the structure of organisms and should be more sensitive than direct mortality effects. It is a biologically based index, with evidence of responsiveness to environmental change, it can be measured in a natural population often without sophisticated methods and its biological significance is possibly more easily assessed than direct chemical measurements. However, pathological studies must be closely integrated with other pollution index studies, biotic and abiotic. A pathological condition is the end result of a long sequence of events within tissues and cells and hence lacks some sensitivity, it lacks specificity because of the limited ways in which tissue can respond, often lacks durability in the absence of causitive agents because of the powers of regeneration of fish tissue and by often showing pathogenicity can be difficult to measure because of differential mortality in the fish population. No specifically pathognomic lesions to pollutants have been identified and consequently pathology will probably be most useful as an indirect index. It has been shown that lowered environmental quality can result in increased disease, but because of the strong influence of natural factors on disease levels and lack of knowledge of fish disease, it has not yet been possible to clearly establish links between pollution and disease in wild populations. Absolute levels of fish disease may be of less significance than trends over a period of time. It is most logical to look for correlations between pollution and disease in areas of changing pollution status.

A firm data base is required for pathology/pollution studies with particular emphasis on principles of epidemiology, mortality effects and the relationship between prevalence and incidence of infection. Accurate standardisation of data from different sources is essential for comparison of different areas. Resources, particularly cost and manpower availability, impose severe constraints on a study programme, and although methods of employing commercial catches and joint cruises have been attempted, the preferred approach is to use specifically disease cruises staffed by specialists. Because of the extent of influence of natural phenomena on fish disease levels and the impracticality of performing frequent and extensive disease cruises, an alternative is suggested in which the effect of natural variation is reduced by standardising sampling as far as possible in terms of season, fish species, area and disease studied. Similarly, because disease often occurs at low prevalence levels, extensive sampling of few conditions in large numbers of fish is preferred to intensive studies of a few fish. Appropriate diseases for study are those easily and accurately diagnosed, preferably with a known aetiology and which have information available on their natural variation. Knowledge of fish stocks and their distribution is essential to allow sampling homogeneity, and use of relatively static groups or species of fish preferable. Early stages of fish would seem most appropriate for pollution effects studies, pre-recruit and older fish for pollution monitoring studies.