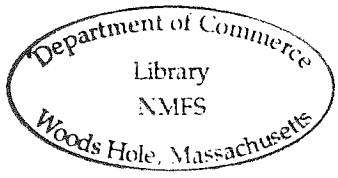
NOAA Technical Memorandum NMFS-F/NEC-54

Epizootic Ulcerative Syndromes in Coastal/Estuarine Fish



U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
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Woods Hole, Massachusetts

June 1988

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Epizootic Ulcerative Syndromes in Coastal/Estuarine Fish

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ABSTRACT

Epizootic ulcerative syndromes have been reported with increasing frequency in fish from many parts of the world, including the east coast of the United States. Etiology is uncertain for many outbreaks, although viruses, bacteria, fungi, and other pathogens have been proposed in specific geographic locations. Environmental stress, often as a consequence of pollution, has also been implicated in at least some of the reported epizootics.

The ulcerative lesions do not constitute a single disease entity, since their characteristics may be quite different in different host species and areas. Such lesions can be considered as generalized responses of fish to infection and/or abnormal environmental conditions. Types of ulcerations have been described, some with several developmental stages, and mortalities have been observed in some outbreaks.

CONTENTS

INTRODUCTION

Ulcerations are highly visible abnormalities seen, usually in very low prevalences, in coastal/estuarine fish of many species. Although precise etiologies have been determined only infrequently, mechanical injuries, bacterial infections, parasitic infestations, and lamprey wounds have been suggested as possible causes of some of the relatively rare occurrences noted.

Lately though--within the past two decades--there have been numerous reports from various parts of the world of what can be described as "epizootic ulcerative syndromes" in fish, in which the dominant gross pathology consists of superficial or penetrating ulcers, and in which significant segments of local populations may be affected. In a number of outbreaks, the ulcerative condition clearly leads to mortality, although evidence in the form of scarring also suggests some recoveries. Usually one species is most severely affected in any given locality, although other species may exhibit lesser intensities of ulceration.

Naturally, a search has been made for infectious agents--particularly viral, bacterial, or fungal--which may act as primary pathogens or secondary invaders. Such organisms have been found, and in some cases proposed as causative organisms responsible for the ulcerative condition. In many epizootics, though, the possible contributory or even dominant role of environmental stressors, such as sudden changes in temperature or salinity, extremes of these and other chemical and physical factors, increases in organic content of water, or industrial pollutants, has been pointed out (Fig. 1).

The development and histopathology of these "epizootic ulcerative syndromes" are different enough to suggest the existence of dissimilar entities, each with complex, probably multifactorial etiology, but in some cases with a probable primary pathogen (such as a rhabdovirus or Vibrio anguillarum) being responsible for initiating the condition.

This paper examines some of the more extensive outbreaks of "ulcerative syndromes," particularly from the viewpoints of causation and effects on populations. Table 1 summarizes information on each outbreak to be considered, and the following sections treat each one in more detail.

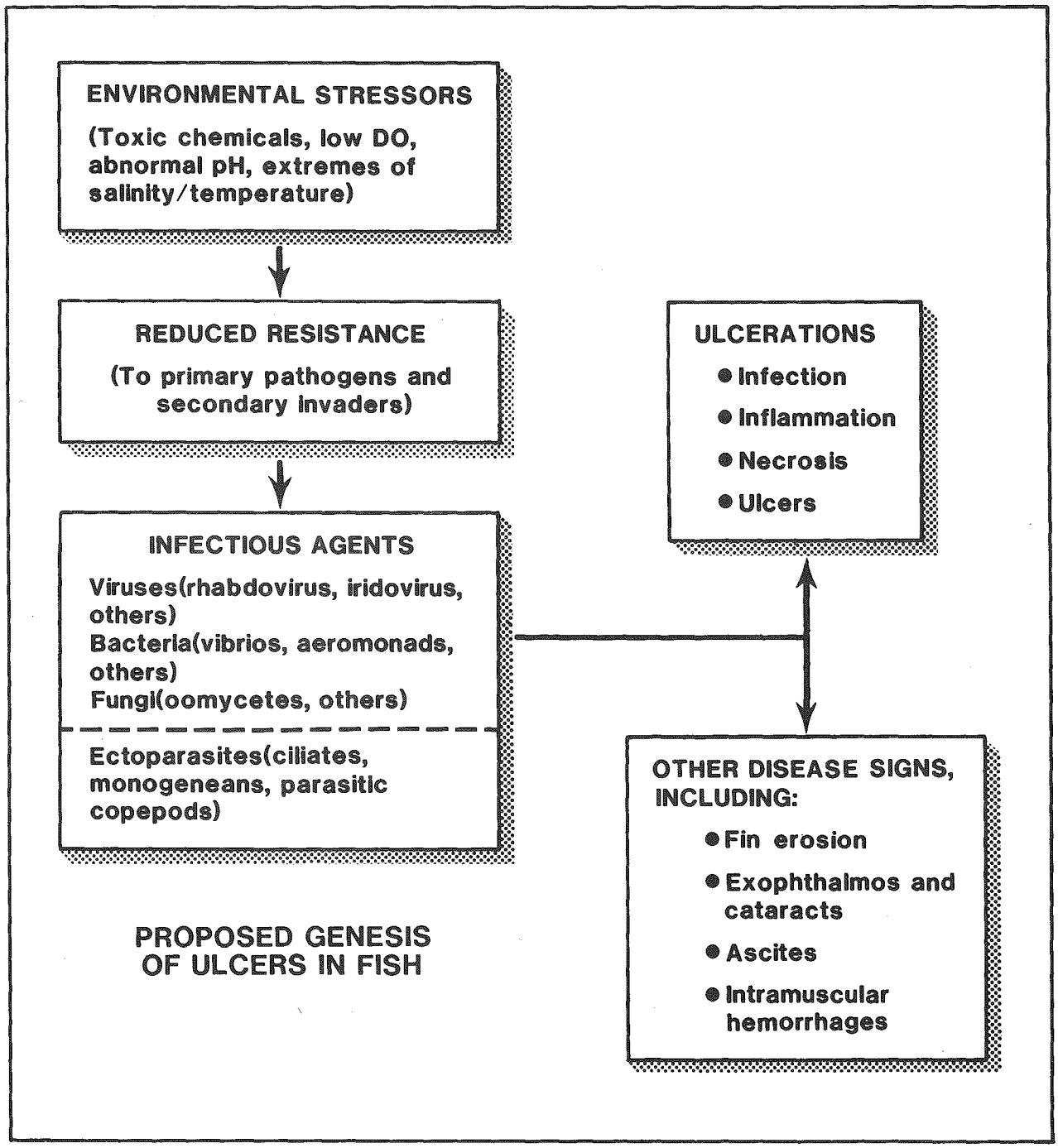

Figure 1. Proposed genesis of ulcers in fish.

Table 1. Ulcerative diseases in coastal/estuarine fish.

Disease	Geographic area	Outbreak period(s)	Cause
Ulcerative mycosis of Atlantic menhaden	Atlantic coast of U.S., from Chesapeake Bay to Florida	1978 to present	Oomycete fungi (presumptive)
Red spot disease of mullet and barramundi	Australia, New Guinea	1972-77; 1975; 1986 to present	Unknown (rhabdovirus suspected)
Ulcerative disease of mullet and other species	Southeast Asia	1980 to present	Unknown (rhabdovirus suspected)
Ulcus syndrome in cod	Denmark	1972 to present	Unknown (bacteria and viruses implicated)
Red disease of eels	Europe, Japan	1718 to present (Europe); 1975 to present (Japan)	Vibrio anguillarum
Spring ulcer disease of eels	Denmark (ulcers also seen in cod from Georges Bank and Gulf of Maine)	1973 to present (1978-80)	Unknown (several bacterial genera implicated)
Red sore disease of sheepshead and black drum	Gulf of Mexico	1965 to present	Ciliate Epistylis and Aeromonas hydrophila
Ulcer disease of red hake	Inner New York Bight (also reported from Mass. Bay by J. Ziskowski (pers. commun.[1]))	1978 to present	Unknown
Ulcers of mullet	France	1978	Associated with "Amoco Cadiz" oil spill

[1]National Marine Fisheries Service, Milford, Connecticut 06460

ULCERATIVE MYCOSIS OF ATLANTIC MENHADEN AND OTHER SPECIES

Beginning in 1978 (with the possibility of earlier occurrences), observations have been made of penetrating ulcers in Atlantic menhaden, mullet, and other species taken in U.S. coastal/estuarine waters from Chesapeake Bay southward to Florida. Menhaden have been most seriously affected, with reported prevalences in young-of-the-year fish of up to 90% (Ahrenholz et al., 1987), and with mortalities that have been considered disease-related. Ulcers were most common near the vent region of affected individuals (Fig. 2), although they were found elsewhere on the fish as well. The disease elicits an intense granulomatous inflammatory response, and oomycete fungi (particularly of the genera Aphanomyces and Saprolegnia) have been found (in some studies in North Carolina) in over 95% of all lesions, whether early or late.* Histologically, fungal hyphae are encapsulated by prominent epithelioid granulomas. Potentially pathogenic bacteria have also been isolated from ulcers, but a primary bacterial etiology for the condition has not been established and no attempts at viral isolation have been reported. The oomycete fungi are the only organisms consistently present in the earliest stages of the disease, but it is also possible, of course, that some environmental stressor may participate in initiating lesions by injuring epithelium or underlying tissues, by changing the surface bacterial flora, by modifying the mucous layer, or by reducing immunocompetence.

The progression of the disease in menhaden, as described by Noga and Dykstra (1986) can take two courses:

> Type I -- Lesions begin as small flat red areas up to 5 mm in diameter, which progress to scale and skin loss, exposing muscle. This leads to formation of ulcers of up to 30 mm diameter with a central zone of necrotic tissue containing fungal hyphae.
>
> Type II -- Lesions begin as raised masses (granulomas) up to 3 mm high on the flanks. The central necrotic core then sloughs, leaving a crater-shaped cavity up to 15 mm deep.

---

*It is relevant to note here that representatives of this group of fungi have been seen in ulcerated fish in Australasia, and "mycotic granulomas" caused by an Aphanomyces sp. have been reported from ayu (Plecoglossus altivelis) cultivated in fresh water in Japan (Hatai et al., 1977; Noga and Dykstra, 1986).

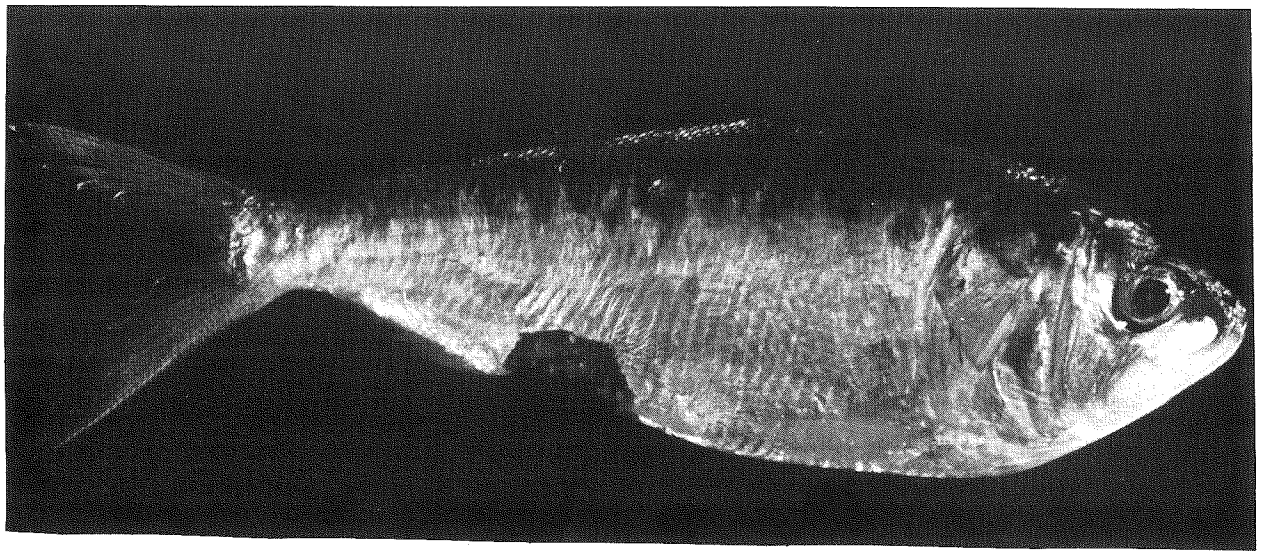

Figure 2. Penetrating ulcer in the vent region of juvenile menhaden. (Photograph provided by Dr. E. J. Noga.)

In either type, tissue reaction takes the form of intense chronic inflammation associated with formation of granulomas consisting of macrophages, epithelioid cells, and a thin rim of fibroblasts. The lesions are never well-encapsulated. An intense granulomatous response to the fungi indicates that deep tissue erosion is a consequence of fungal activity, whatever the primary pathogen (Dykstra et al., 1986).

The epizootiology of "ulcerative mycosis" has been and is being examined. Relevant information is that:

(1) The disease occurs in low salinity waters of bays and sounds. Some young-of-the-year menhaden with ulcers have been seen outside these areas, but prevalences there are low.

(2) Some ulcers have been seen in fish taken in salinities up to 25 ppt; in these the fungi (typically fresh or brackish water inhabitants) may be protected from the high salinities of the lower estuaries by close association with host tissues.

(3) Koch's postulates have not been satisfied for any of the fungi isolated.

(4) The disease, formerly known as "red sore" reached epizootic proportions in menhaden from Pamlico Sound in 1984, when up to 80% of individuals from pound net catches had characteristic deep ulcers (Dykstra et al., 1986). Prevalence dropped precipitously in 1985, averaging less than 1%.

(5) Although the oomycete fungi isolated from ulcerated menhaden are typically freshwater forms, at least one of the Aphanomyces isolates produced zoospores in cultures containing 2-20 ppt NaCl, and zoospore production was actually depressed in salt-free media (Dykstra et al., 1986). Optimum salinity was 2-6 ppt.

A recent summary of the ulcer disease as it occurs in Florida waters (Grier and Quintero, 1987) has added substantially to information from the southern extreme of its reported distribution. Significant findings include these:

(1) The disease is common in mullet (Mugil cephalus and M. curema) taken near drainage canals, but other species are affected, including yellowmouth trout (Cynoscion regalis), Atlantic menhaden (Brevoortia tyrannus) from the St. Johns River, gizzard shad (Dorosoma cepedianum) from Lake George on the St. Johns River, catfish (Ictalurus nebulosus) from Lake George, and jack crevalle (Caranx hippos) from Bayboro Harbor.

(2) Observations of "sick" mullet began in 1978, with reports from the St. Johns River, St. Lucie River, Punta Gorda, Manatee River, and elsewhere on the east and west coasts of Florida (fishermen claim to have seen "sick" mullet in the St. Johns River as early as 1972).

(3) The common appearance of the disease in mullet is in the form of large penetrating hemorrhagic ulcers on the flanks (Manatee River samples).

(4) Affected fish are usually seen in salinities less than 20 ppt, mostly in brackish water near canal locks.

(5) Prevalences in 1985 were up to 50% in mullet from the Manatee River, 5-8% in menhaden from the St. Johns River, and 6-8% in yellowmouth trout.

(6) Fungal hyphae have not been seen in some ulcerated mullet from the Manatee River or in ulcerated menhaden from the St. Johns River, and bacterial infections have been suspected. This points out the possibility that several diseases may be present in any given area, and may sometimes elicit similar gross signs.

A concomitant examination of the ulcer disease in menhaden from Pamlico Sound and other middle Atlantic sampling sites disclosed that some level of the disease was present in 1982 to 1986 year-classes, with the highest in the 1984 year-class (Ahrenholz et al., 1987). The study was based on young-of-the-year sampling and commercial catch sampling--part of a long-term program of the National Marine Fisheries Service. One conclusion reached, which has broader implications in fish population dynamics, was that mass mortalities in one year-class, whether the result of ulcers or other disease conditions, would have to be extremely large (> 70%) to have a noticeable impact on the population (Vaughan et al., 1986). The authors pointed out that mass mortalities of an abundant species like menhaden might be very noticeable in an environmental sense, but still involve only a small percentage of the total recruiting year-class, and hence have small impact on the population.

Present information about "ulcerative mycosis" on the east coast of United States is still inadequate to answer basic questions about a primary pathogen or the role of environmental degradation. Expanding interest in the disease by university, state, and federal research groups should result in accelerated acquisition of new information.

"RED SPOT" DISEASE OF MULLET, BARRAMUNDI, AND OTHER SPECIES IN AUSTRALASIA

Large external ulcers were observed in Australian estuarine fish, notably mullet (Mugil cephalus) and barramundi (Lates calcarifer), beginning in 1972. First reported from Queensland, the disease spread subsequently to New South Wales and New Guinea (McKenzie and Hall, 1976; Rodgers and Burke, 1981). Vibrio anguillarum was identified early in the Queensland outbreak as the causative organism, since it was the only potential pathogen associated with early lesions (Burke and Rodgers, 1981). Highest prevalences of ulcers were seen at times of rapidly decreasing salinities; one survey in 1976 (Lake Cootharaba, Queensland) disclosed peak prevalences in mullet of 25%.

Secondary fungal infections were seen in advanced lesions of mullet and other species, so very low salinities may influence the degree of secondary invasion (Hine, 1975). This conclusion was supported by the observation that ulcerated fish were not found in offshore samples.

The course of the disease, known as "red spot" or "Bundaberg disease," has been described in three phases (Rodgers and Burke, 1981):

(1) Early lesions, characterized by inflammation of scale pockets and localized hemorrhages;

(2) Later lesions, characterized by spread of the inflammation, scale erosion, and epidermal degeneration; and

(3) Advanced lesions, characterized by absence of scales, sloughing of the epidermis and dermis, and ulcers with hemorrhagic margins. Fungal hyphae are apparent in dermal and muscle tissues, accompanied by necrosis and fibroblastic proliferation around hyphae, leading to formation of mycotic granulomas.

A reappearance of red spot disease, this time in waters of the Northern Territory, occurred in 1986 (Grey, 1987). Mullet, barramundi, and other estuarine and catadromous species were affected, and a rhabdovirus was isolated, but not confirmed as an etiological agent.

These and other ulcerative conditions in Australasia and Southeast Asia were examined by an FAO expert consultation on fish diseases (FAO, 1986) which concluded that the lesions seen in fish from Australia/Papua New Guinea were "fungal granulomata with occasional foci of bacterial necrosis." The fungus was "branching, nonseptate, nonspore forming, somewhat similar to Ichthyophonus." Primary etiology was not determined, but the disease, on clinical and histological grounds, seemed to be a

different disease from that in Southeast Asia (to be considered next). The group of experts also concluded that although vibrios and aeromonads may be involved in the pathogenesis of ulcer disease, it is as secondary invaders and not primary agents of infection.

It seems unlikely, in Australasian fish, that fungi or bacteria are the primary pathogens, but a viral agent (or agents) may be involved. The progression of the disease is similar to spring viremia of carp, so a rhabdovirus might be responsible for initiating the lesions.

ULCERATIVE DISEASES IN SOUTHEAST ASIAN FISH

Ulcerations of fish have become a problem in Southeast Asia, where they occur in fish farms as well as in natural waters. The ulcer condition is particularly acute in fresh water--rivers, ponds, rice paddies, farm ponds--and especially among air-breathing fish, but it occurs in brackish water species too. A recent country-by-country summary of the disease (Boonyaratapalin, 1983; Tonguthai, 1985), and an examination of samples, disclosed the following:

(1) Fish from Indonesia (principally catfish and carp) had ulcerations and spinal deformities but the disease was distinct from that found in the Burma/Lao/Thailand/Malaysia area, and also distinct from the red spot disease found in Australia/Papua New Guinea.

(2) The Burma/Lao/Thailand/Malaysia samples were characterized by large eroding ulcers, emaciation, and inflammation of the viscera (a condition not seen in Australian samples).

(3) In samples from Thailand, large cells with disintegrating nuclei were seen in kidney and spleen adjacent to areas of renal tubular or hematopoietic degeneration. Such cells were types which appeared to specifically demonstrate the presence of rhabdovirus-like particles. This cell type was seen in snakeheads and swamp eels from five locations in Thailand.

(4) Later stages of ulcerations were invaded by slender branching fungi, which penetrated internally from the dermis.

(5) The pathology seen did not suggest an acute primary toxic disease.

(6) Bacteria, principally Aeromonas hydrophila, probably play a terminal role in ulcerated Southeast Asian fish.

(7) Ulcerative disease has been reported in estuarine coastal fish in Southeast Asia--particularly in mullet from Singapore and Thailand. In some cases phytoplankton blooms preceded the appearance of ulcers.

The Food and Agriculture Organization of the United Nations (FAO), responding to concerns about epizootics of ulcerative disease conditions among wild and cultured fish in Southeast Asia and Australasia which have occurred since 1972, established a Technical Cooperation Programme to investigate causes. Considering the possibility that the outbreaks represented a major pandemic or ecological disaster, a study team of fish disease specialists conducted in 1985 extensive field and laboratory investigations in areas where epizootics existed or had occurred. Their report, and the results of an FAO "Expert Consultation on Ulcerative Fish Diseases in the Asia-Pacific Region" held in 1986 were both published (Roberts et al., 1986; FAO, 1986). The following general conclusions were reached:

(1) In Southeast Asia there is a serious specific epizootic ulcerative disease which must be differentiated from the normal range of ulcerative conditions occurring at low levels in most fish populations.

(2) The specific Southeast Asian condition shows a number of similarities with the outbreaks in Australia and Papua New Guinea, but it is considered on balance that these two conditions are likely to be separate syndromes.

(3) Pesticides are not directly responsible for the specific epizootic ulcerative syndromes in Australasia or in the Southeast Asian disease; there is no correlation between pesticide use and the distribution of the disease and some highly affected areas have very low application levels of pesticide.

(4) The disease has shown no demonstrable relationship to other environmental parameters, but several outbreaks have coincided with rainfall and lowered temperatures.

(5) Epizootic levels of the ulcerative disease have been reported in Thailand, Burma, and Lao PDR in 1985 and 1986, with recent indication of its spread to the Philippines as well. (This conclusion was confirmed by a recent report (Llobrera and Gacutan, 1987) of an ulcerative disease outbreak in mudfish/snakehead, Ophiocephalus striatus, catfish, Clarias batrachus, crucian carp, Carassius carassius, goby, Glossogobius giurus, and other species in Laguna de Bay, Philippines, with consistent isolation of Aeromonas hydrophila from lesions.)

(6) A rhabdovirus is considered the primary pathogen in at least some outbreaks in Southeast Asia. Fungi are found consistently in later stages of typical Australasian and Southeast Asian ulcerations, but pathological effects are different. In Australasian fish the fungus is contained and fish recover; in Southeast Asian fish the fungus is invasive, often penetrating to the internal organs.

In addition to the suspected rhabdovirus infections reported by Tonguthai (1985), a birnavirus (called SGV) was isolated from sand gobies (Oxyeleotris marmoratus) with a lethal ulcer disease, sampled from freshwater culture cages in Thailand (Hedrick et al., 1986). The virus differed from birnavirus isolates from salmonids by being cytopathic at 30°C, and replicating in cell lines from warm-water fish. The authors did not, however, claim a definitive role for SGV in the etiology of ulcer disease in Thailand.

In another recent study, a rhabdovirus was isolated from ulcerated wild- and pond-cultured fish in Thailand (Frerichs et al., 1986). Snakeheads, an economically important species, have suffered severe losses from the disease beginning in 1980, and the authors considered the rhabdovirus as the "likely primary causal agent of the disease."

"ULCUS SYNDROME" OF COD IN DENMARK

A specific ulcerative condition of Atlantic cod, Gadus morhua, from Danish waters was first described by Jensen and Larsen (1976). Initially, bacteria (Vibrio anguillarum and Aeromonas sp.) were thought to be important pathogens (Larsen and Jensen, 1977, 1979), but viruses were also isolated from diseased fish and one, an iridovirus, may be the primary pathogen (Jensen, 1983; Jensen et al., 1979; Jensen and Larsen, 1982). Since the syndrome is more prevalent in polluted waters (Jensen et al., 1980; Larsen and Jensen, 1982), it may well be that the cause of the disease is multifactorial, involving a virus, bacteria, and environmental stressors (Larsen, 1983).

But whatever its genesis, the ulcer condition passes through a series of well-defined stages (Jensen and Larsen, 1979; Jensen, 1983):

(I) Papulovesicular stage, in which multiple small elevations of the skin, 2-8 mm in diameter, develop and may hemorrhage. Histologic sections disclose extensive edema and intense granulocytic invasion (Fig. 3-1).

(II) Erosive stage, characterized by crater-shaped holes in the skin, of a size similar to the original lesion, and with edema and cell infiltration.

(III) Early ulcerative stage, characterized by small ulcers with central necrotic areas. In a few cases the musculature may be exposed (Fig. 3-2).

(IV) Late ulcerative stage is characterized by one or a few large ulcers, 2-8 cm in diameter. These ulcers develop from a single papule or from confluence of several ulcers. Both superficial and penetrating ulcers occur. Histologically, extensive granulation tissue is present, while underlying musculature is necrotic with inflammatory cell infiltration (Fig. 3-3).

(V) Healing stage is characterized by small white patches in the skin, usually occurring in late autumn, and usually following Stages I and II (although large ulcers may also be healed, with formation of large scars). Histologically, the inflammatory cell infiltration is replaced by fibroblasts, and scales and pigment cells are absent (Fig. 3-4).

Some aspects of the epizootiology of ulcus syndrome of cod are known, and others are assumed:

- Ulcerations appear to have an infectious etiology, but more than one microorganism may be involved in pathogenesis (Jensen, 1983).
- High water temperatures seem to result in increased prevalences, reaching a peak in early autumn, while the healing stage is seen when water temperatures decrease below 7°C in early winter.
- Prevalence of ulcus syndrome was found to be directly correlated with intensity of pollution from carbohydrate processing plants (Jensen and Larsen, 1976, 1978), although other environmental variables may be important.
- Pronounced annual variations in prevalences occur.

The extensive literature on ulcus syndrome of cod and its well-defined developmental stages place this ulcerative condition among the better understood of its genre, despite continuing uncertainty about precise etiology.

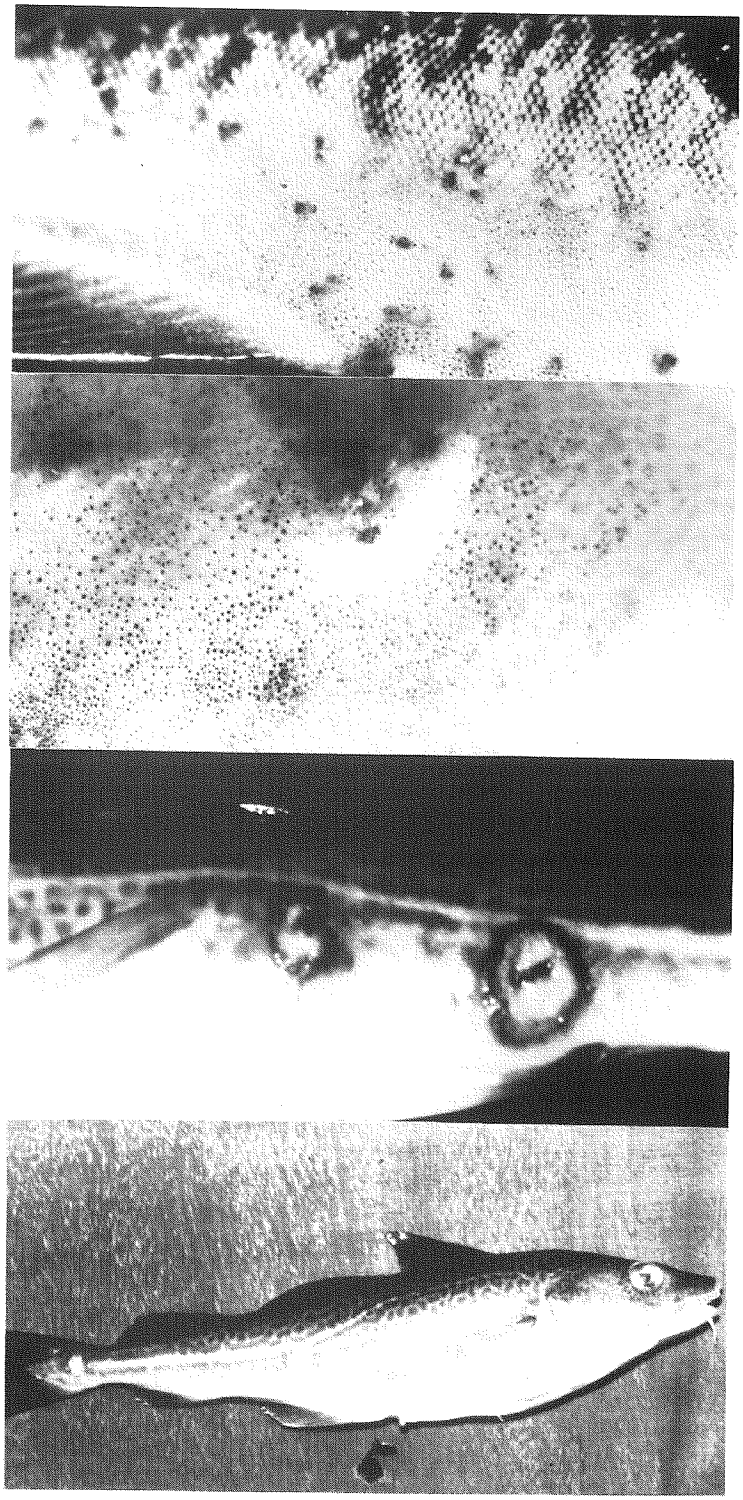

Figure 3. Stages in the ulcus syndrome of cod -- (1) papulo-vesicular stage, (2) early ulcerative stage, (3) late ulcerative stage, (4) healing stage. (Photographs provided by Dr. I. Dalsgaard.)

ULCERATIONS RELATED TO VIBRIOSIS IN MARINE FISH

Vibrio and similar infections have been implicated in a number of reports of ulcerations in fish--in fact, next to fin erosion, ulcerations with bacterial etiology are probably the most common abnormality in fish from polluted waters. Ulcers may be integumentary or penetrating; where bacterial isolations have been made from ulcerated tissue, Vibrio anguillarum has been by far the most predominant organism, with pseudomonads and aeromonads in lesser abundance.

Ulcerations in winter flounder, Pseudopleuronectes americanus, of Narragansett Bay were reported by Levin et al. (1972). The acute ulcerative lesions were thought to be caused by V. anguillarum infections, and the ulcerative phase was reproduced in fish exposed experimentally to cultured V. anguillarum isolates. A report by Robohm and Brown (1978) described systemic bacterial infections and ulcerative lesions of the tail and dorsal muscles in summer flounder from Connecticut waters (Fig. 4). A highly pathogenic Vibrio sp. was isolated, and experimental infections were produced by subcutaneous inoculation and by seeding holding tanks with bacteria at levels of 360/ml. Ulcers at the inoculation site and subcutaneous hemorrhages along the bases of fins characterized experimental infections. These observations resemble those of Levin et al. (1972) in winter flounder.

Ulcerations, probably of bacterial etiology, have been reported in fish of several species from the Irish Sea. Perkins et al. (1972) and Shelton and Wilson (1973) reported ulcers from European flounders (Platichthys flesus), dab, and plaice. Prevalences were low (1-4%) in most instances.

Skin lesions are common in saithe (Pollachius virens) during sporadic epizootics which have occurred on the coast of Norway since 1962 (Egidius et al., 1983). Mortalities in wild and captive stocks, sometimes reaching 100% in local areas, characterized the outbreaks. Vibrio anguillarum was isolated consistently from dead and dying fish.

Ulcerations or external lesions on fish may, of course, have a number of causes other than bacterial infections. They may be due to net damage or other surface abrasions, or to predator attacks. Some protozoa (Myxozoa and Microspora) can infect muscle or skin tissue and multiply to produce gross cysts. These infections mature to produce many characteristic spores, and in the process the overlying epidermis may be sloughed, resulting in ulcers with usually smooth borders. However, it seems to be a reasonable generalization that many of the infections that produce grossly visible ulcerations in fish are bacterial, and are often due to pathogens of the genera Vibrio, Pseudomonas, or Aeromonas (Lamolet et al., 1976). Ulceration often begins with scale loss or formation of small papules, followed by sloughing

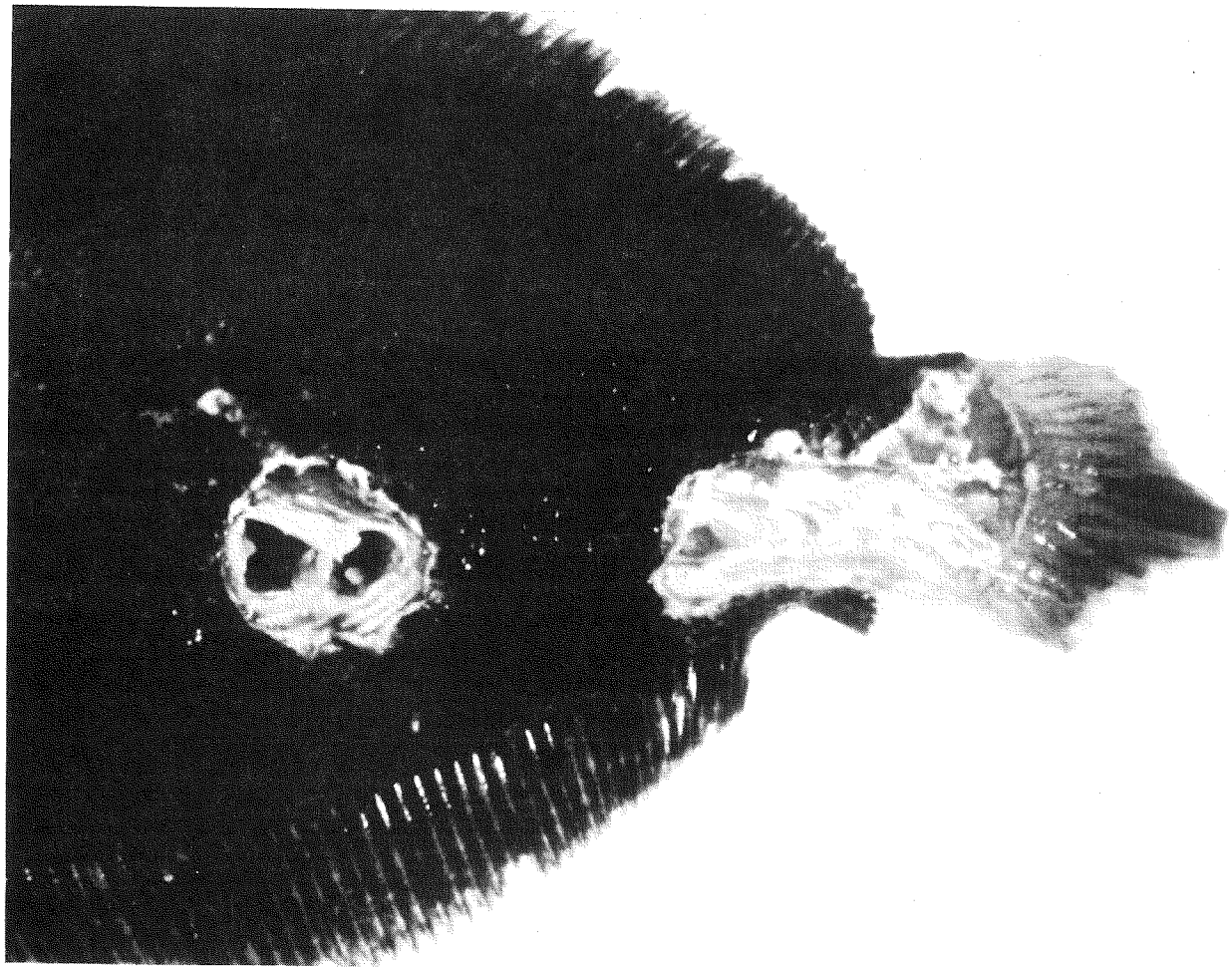

Figure 4. Ulceration in summer flounder, Paralichthys dentatus, caused by Vibrio sp. (Photograph provided by Dr. R. A. Robohm.)

of the skin, exposing the underlying muscles, which may also be destroyed. Bacterial ulcers may have rough or raised irregular margins, and will often be hemorrhagic. Ulcers may or may not be associated with fin erosion.

ULCERS OF MULLET AND OTHER SPECIES OF FISH ON THE COAST OF FRANCE

A major survey of the occurrence of ulcers in fish was undertaken by France--CNEXO (now part of IFREMER)--during the period from 1978 to 1981. This was a response to attention being paid to ulcerations of fish by several other European countries, with the underlying concern that of possible pollution effects on fisheries and ecosystems (see, for example, McIntyre and Pearce, 1980). The program was multidisciplinary "to determine the conditions and specific factors provoking ulceration in fish" (CNEXO, 1981).

Since this study represents one of the largest national attempts to document the occurrence of ulcerations in marine fish, a detailed review of principal findings would seem important. These are:

(1) "A large proportion of lesioned fish are flat fish or sendentary species although some migratory species such as shad, mullet, flounder and cod are also affected. Prolonged contact with certain sites contaminated by wastes could encourage outbreaks of lesions but the exact process has still to be determined."

(2) "The majority of the species observed seem to suffer a similar number of lesions except for the dab. The population of this species taken in the Seine Bay for example, represented 5% of the species caught. The proportion of dab suffering from lesions was 1% of the dab taken, which is comparable to the other proportions, but 65% of the total lesioned fish which is abnormal but less than the 95% of 1977. It would seem therefore that the dab is particularly vulnerable and this confirms results obtained in the Irish Sea and the German Bight of the North Sea where they are affected by epidermal papillomas."

(3) "Pathological examinations yield a description and classification of external lesions which come under the general term of 'necrosis.' Apart from 'lymphocystis' and the fin rot disease, which have been dealt with extensively in the literature (as well as some traumatic or parasitical ulcerations), external lesions consist for the most part of 'necrosis' or cutaneous ulceration developing pathologically (congestive, haemorrhagic or granular), in the process of scarring

or even scarred. The histological characteristics of these lesions underline their homogeneity; they can in fact be seen as successive stages of the same pathological process."

(4) "Although public opinion is most impressed by external lesions, these are not the only kind found in this study. Very much more important from the pathological point of view are the internal lesions of the viscera, mainly in the liver and spleen which have been identified in ulcerated and apparently ulcer-free species. They may even prove to be a significant link in the outbreak of external lesions once the correlation between 'cutaneous necrosis' and visceral lesions already noted has been more widely confirmed."

(5) "The responsibility of hydrocarbons in the appearance of certain types of ulceration such as fin rot is already widely documented in the literature. The responsibility of organic pollutants has also been adequately confirmed by examination of fish caught in areas of chronic pollution and zones falling within the effluents of oil refineries and petro-chemical plants in the Mediterranean; this is also the case of incidents of massive pollution such as followed the wreck of the Amoco Cadiz off the Breton coast."

(6) "Bacteriological investigations also confirm the conclusions of the literature concerning the role of bacteria and viruses in the development of necrotic ulceration. Despite a general increase in their activity, the strains which colonize the ulcerations do not seem to be the primary cause of ulceration. However bacterial activity can combine with other forms of aggression such as wounds, the detergent action of surface active chemical compounds or that of physico-chemical factors such as the pH. This synergy can then provoke a secondary infection which will in turn worsen the primary lesion."

In the general coastal survey, ulcerated mullet were taken in the Mediterranean Sea at the mouth of Toulon's major sewer outfall. White skin lesions were seen in mullet from the Imperia region of the Italian coast, the Bay of Naples, Sicily, and the Bizerta region of Tunisia.

In an independent study, superficial and penetrating ulcers were reported in samples of mullet obtained on the north coast of Brittany six months after the "Amoco Cadiz" oil spill in March 1978 (Balouet and Baudin-Laurencin, 1983). Affected fish were emaciated, and 50 to 80% of the catch was reported to show lesions (unspecified).

ULCERS OF RED HAKE IN THE NEW YORK BIGHT

Fish of the New York Bight have won notoriety principally because of the widespread occurrence of fin erosion (Mahoney et al., 1973; Murchelano and Ziskowski, 1976). Recently, though, at least one fish species (the red hake, Urophycis chuss) has been reported with prominent hemorrhagic ulcerations. Samples from a sewage sludge dumpsite in 1978 and 1979 disclosed a prevalence of 12.5%; no ulcerations were seen in red hake from reference sites outside the Bight (Murchelano and Ziskowski, 1979). Ulcers were distributed randomly on the trunk of the fish (Fig. 5), with multiple lesions commonly seen. Anecdotal information from fishermen and from earlier trawl surveys disclosed no previous observations of the conspicuous (5-10 mm) ulcers. Subsequent trawling surveys, however, disclosed low prevalences of ulcers (maximum 0.35%) in red hake from Georges Bank and the Gulf of Maine sampled from 1979 to 1983 (Ziskowski et al., 1987).

Other than its association with a highly polluted coastal area, the ulcer disease of red hake has not been linked to any etiologic agent. Microbial infections were not reported, nor was systemic invasion seen in any samples tested. Obviously, further studies of this ulcerative syndrome are needed, especially since it occurs with greatest frequency in an area, the New York Bight apex, where fin erosion--which has been related statistically to environmental degradation--has been reported, sometimes at high prevalences, in 22 species of fish.

"RED SORE" DISEASE OF FISH IN THE SOUTHEASTERN UNITED STATES

An ulcerative condition common in freshwater and euryhaline fish of the Gulf of Mexico has occurred at epizootic levels (over 50% prevalences) in bluegills, bass, channel catfish, sheepshead, and black drum taken in fresh and brackish water. The disease is thought to result from synergistic action of a stalked ciliate, Epistylis (Heteropolaria) sp. and the bacterium Aeromonas hydrophila (Rogers, 1972; Overstreet and Howse, 1977). An association of "red sore" disease with pollution has been postulated (Overstreet, 1978) based on observations of dramatic increases in prevalence in low salinity bays of Mississippi where water flow and flushing rates have decreased and pollution (especially organic loading) has increased (Rogers, 1970). The disease is usually found in fish from waters with salinities of less than 5 ppt, but the most severe hemorrhagic form has been seen in marine invaders of brackish water habitats--species such as the sheepshead and black drum.

Lesions surrounding the ciliates may be deep and hemorrhagic; Aeromas hydrophila is consistently present, and infections are often systemic. Mortalities may occur as a consequence of infections, under some conditions (Rogers,

Figure 5. Ulcers of red hake from the New York Bight. (Photograph provided by J. Ziskowski.)

1972). Thus, a tripartite system seems to be at work in "red sore" disease, involving initial invasion of the integument by the ciliate Epistylis sp.; secondary invasion by bacteria, especially A. hydrophila, resulting in systemic infections; and the possible influence of environmental stressors, such as high organic content of brackish bays, which enhance ciliate growth. Low salinities of estuaries themselves can impose osmotic stress on marine invaders--which often have the most severe form of ulcerations.

Red sore is also a common disease in North Carolina rivers and reservoirs, where it was reported to have caused extensive mortalities of largemouth bass (Micropterus salmoides), striped bass (Morone saxatilis), white perch (Morone americanus), and other species during the 1970's (Miller and Chapman, 1976). Recent work with a form of the disease in largemouth bass has indicated that the etiology is complex. Ulcerations were thought, in some instances, to result from primary Aeromonas infections, with Epistylis as a secondary invader (Hazen et al., 1978; Huizinga et al., 1979). Elevated water temperatures and poor body condition of the fish were correlated with increased susceptibility to infection. In a recent study, over 55% of all largemouth bass examined from one North Carolina river in 1984 and 1985 had skin lesions (Noga, 1986). Many of the lesions were not, however, true "red sore" disease as defined by Huizinga et al. (1979). The most common pathogen seen in early lesions in Noga's study was Lernaea cruciata, which he considered a primary disease agent, rather than bacteria.

SPRING ULCER DISEASE OF EELS IN DENMARK

Ulcerations have been observed and reported in European eels for more than half a century (Bruun and Heiberg, 1932), but until recently they have been included with signs of "red disease" of eels, caused by Vibrio anguillarum. That the ulcerations were part of a different disease syndrome, called "spring ulcer disease," was pointed out by Hansen and Bonde (1973) and reaffirmed by Dalsgaard (1981) and by Jensen et al. (1983). The ulcer disease is most abundant at colder temperatures in spring, whereas "red disease" is most common in late summer, and is characterized by progressive reddening of the skin and fins, but not typically by ulceration.

Prevalence of spring ulcer disease varies geographically; 86% of eels in one Danish fjord were diseased in a study in 1979 (Jensen et al., 1983). Laboratory holding experiments indicated high mortality rates among affected fish, and three bacterial species--Aeromonas hydrophila, Vibrio sp., and Alcaligenes sp.--were considered to be involved in producing infections.

The course of the disease, as described by Jensen et al. (1983), is as follows:

(1) Primary lesions appear as light pinpoint-sized spots along the lateral line

(2) Hyperemia occurs; then a hemorrhagic ulcer forms, and expands in size

(3) Advanced ulcers have a grayish center surrounded by a hemorrhagic zone (Fig. 6) which gradually narrows

(4) Perforations appear in the grayish portions of the lesions, exposing underlying tissues

(5) Hemorrhagic lesions may also appear simultaneously in the caudal region of the eel

(6) Diseased eels disappeared from samples by July; some anecdotal information indicates that healing may occur.

A possible role for environmental factors--especially high populations of Aeromonas hydrophila--was indicated by the fact that when healthy eels were transferred to a fjord in which the disease was abundant and A. hydrophila counts were high, the introduced stock developed ulcers.

For comparative purposes, it should be pointed out that "red disease" of eels is a much more serious problem than "spring ulcer disease." "Red disease" has been known in European eels for almost a century (Canestrini, 1893), and has been responsible for extensive mortalities in several countries (Fedderson, 1896a, b, 1897a, b; Bergman, 1909; Bruun and Heiberg, 1932, 1935; Nybelin, 1935; Bagge and Bagge, 1956). Signs of vibriosis of eels or "red disease" were summarized recently by Jensen et al. (1983):

> "The first external symptoms are usually a red coloration at the end of the tail or on the body along the dorsal and anal fins; along with this the eels become less lively and swim, if stimulated, with stiff wriggling movements--not so powerful, however, that the fish cannot easily be taken in the hands again. If undisturbed it rests quietly on the bottom for the most part. The red coloration gradually increases along the fins until it appears as dark red streaks over longer or shorter distances, and a diffuse reddening spreads over most of the body. The lymph heart in the tail works more and more slowly until at last, it ceases to beat. The eels often die before the red coloration is well developed."

"Red disease" caused by Vibrio anguillarum has also become a problem in Japanese eel culture (Muroga et al., 1976a, b; Nishibuchi and Muroga, 1977; Miyazaki et al., 1977) along with "red spot disease"--characterized by minute skin hemorrhages and caused by Pseudomonas anguilliseptica (Muroga et al., 1973, 1975,

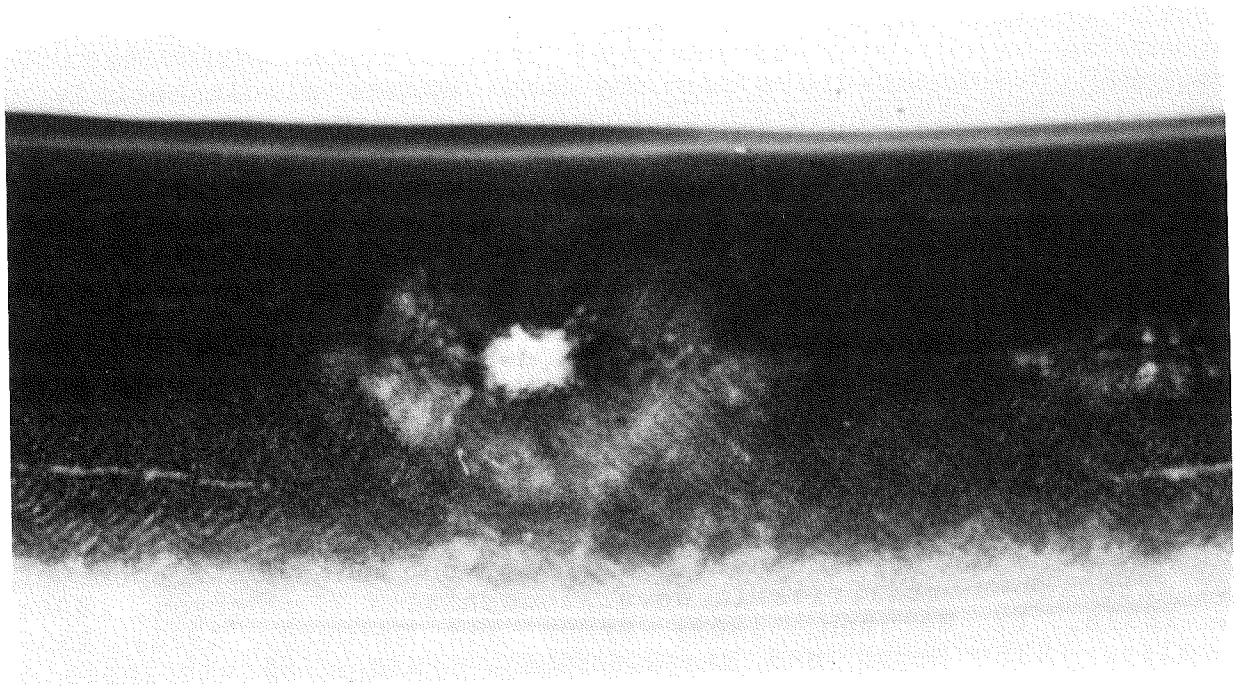

Figure 6. Spring ulcer disease of eels -- large necrotic lesion surrounded by a broad hemorrhagic zone. (Photograph provided by Dr. I. Dalsgaard.)

1977). Vibriosis ("red disease") in Japanese eels, Anguilla japonica, has external manifestations which are different from "red disease" of European eels--small hemorrhagic areas on the body surface develop into extensive hemorrhagic, then necrotic, lesions (ulcers) in advanced infections.

OTHER ULCERATIVE CONDITIONS IN FISH

Since this paper is concerned with one of the most common gross pathological conditions in fish, and one that is characteristic of many kinds of infections, it may seem a little unfair to isolate and describe only a sample of the most dramatic of them. To provide a proper perspective, it should be pointed out that many other types of ulcerative lesions have been reported, and that some of the well-known diseases of fish (such as furunculosis) may sometimes include ulcers as a manifestation of advanced infections.

Among the ulcerative conditions not emphasized in this document but worthy of some attention are "ulcerative dermal necrosis" of salmonids in Europe, ulcers of sea herring with advanced fungal (Ichthyophonus) and myxosporean (Kudoa) infections, "lateral line necrosis" in cod from northern Europe (E. Egidius cited in Möller and Anders, 1983), and even "fin erosion" in many species of fish--a condition which fits the broadest definition of ulcers as "open lesions with necrotic tissues."

But the examples considered in detail in this paper should provide adequate support for the observation that ulcerative syndromes can occur in outbreak proportions, as well as at low enzootic levels in fish populations, and that during the outbreaks extensive mortalities may occur.

CONCLUSIONS

Ulcerations, of complex and varied etiologies, occur at very low levels in most fish populations, and are among the most obvious gross pathological signs recognized in fish. Superimposed on these background levels of ulcerations are sporadic but often widespread epizootic occurrences of diseases or syndromes whose dominant external signs are superficial or penetrating ulcers. Species susceptibility is highly variable.

There is some information which suggests that many of the ulcerative conditions seen may be associated with environmental stressors such as low salinity, high temperature, and pollution (particularly organic loading of inshore waters). Gross signs of disease--such as ulcers or fin erosion--are often good indicators of stress; such generalized signs in fish are universal, but

highly variable by species and age. One of the clearest indications of an environmental influence on ulcerative diseases was seen in sea perch, Lates calcifer, and grouper, Epinephelus tauvina, cultured in Malaysia (Nash et al., 1987). Severe hemorrhagic ulcerative dermatitis followed by mortalities of 20-60% occurred annually in sea cages during the monsoon season, when nutrition and water quality were poor. Improvement in environmental conditions was considered by the authors to have led to termination of the disease problem--in which facultative bacteria (Pseudomonas sp.) were implicated.

There is also information linking some ulcerative conditions with microbial pathogens such as Vibrio anguillarum, viruses, and oomycetous fungi, although even in these instances a role for environmental stressors has not been excluded. The possible roles of fungi in ulcerative syndromes are especially problematic. In some (cod ulcus syndrome, spring ulcer disease of eels), fungi have not been reported; in others (red spot disease in Australasia, ulcerative diseases in Southeast Asia), fungi occur in advanced lesions and are considered secondary invaders; in still others (ulcerative mycosis of menhaden), fungi are considered primary pathogens. It seems reasonable that the usual sequence of events may be environmental stress, followed by primary microbial infection, followed in some cases by deep mycotic invasion, causing penetrating ulcers.

Ulcer diseases are of distinct types (and presumably distinct etiologies). For example, studies in Southeast Asia and Australasia have suggested three different conditions: (1) the Australasian type, (2) the Indonesian type, and (3) the Southeast Asian type of ulcerative diseases--all existing at the same time, and each with distinct developmental stages and histopathology.

Use of the terms "ulcer disease" or "ulcerative disease" conveys the connotation of specific disease entities--which is incorrect. It seems clear that ulcerations of fish may have complex multifactorial etiologies, including infectious and environmental components. External lesions that we call "ulcers" are only one of a number of generalized disease signs in fish. Others include fin erosion, exophthalmos and cataracts, ascites, intramuscular hemorrhages, and various granulomas (mycotic, helminthic, mycobacterial, etc.). These signs of disease are variable among different fish species, and may be exacerbated by environmental stressors. Ulcerations and/or granulomas are also common gross pathological signs in a number of fish diseases with specific bacterial etiologies (for example, furunculosis, vibriosis, pasteurellosis). Ulcers in fish, as disease signs but not as specific disease entities, have their counterparts in invertebrates too; they would be analogous to "yellow pustules" in molluscs, or black gills in crustaceans--as indicators of an infectious process. Although this paper is concerned solely with global distribution of epizootic ulcerations, it would be equally possible to isolate and discuss the distribution and abundance of

other gross pathologies, such as "fin erosion syndrome" or "exophthalmos" or "idiopathic granulomas"--and this probably should be done (but not here).

Recent increases in the frequency of reported occurrences of epizootic ulcerative syndromes in widely separated geographic areas invite the speculation that fish may be reacting to subtle environmental changes, effective over broad areas, resulting in increases in stress-related responses--one of which is the appearance of ulcerations. The nature of such environmental changes is not clear, but it is tempting to suspect a relationship to human activities, when we consider long-term modifications like the widespread dissemination of chlorinated hydrocarbons, or gradual increases in organic loading of estuarine/coastal waters, with concomitant algal blooms and anoxic episodes. It is unlikely that any single stressor would have universal effects, but the totality of effects, in different areas and in different species, could raise the level of visibility of such an obvious indicator as fish ulcers.

The epizootic ulcerative conditions described here suggest the existence of major ecological disturbances--and not merely an artificial assemblage of disparate localized phenomena. If we exclude those ulcerations clearly associated with specific pathogens--such as red disease of Japanese eels caused by Vibrio anguillarum--we are still left with an array of epizootic ulcerative syndromes, worldwide, whose etiology is still uncertain, and for which environmental stressors seem to lurk in the background.

It may be instructive, if only indirectly relevant, to dwell briefly on the global nature of biological phenomena such as the occurrence of ulcer diseases in fish. Other analogous examples exist; one would be the widespread bleaching and death of corals, as summarized recently by Brown (1987). Proposed causes include epizootic disease (Gladfelter, 1982; Antonius, 1985; Glynn et al., 1985) as well as a number of natural and man-induced environmental changes (effects of El Nino, hurricanes, herbicide pollution, increased sedimentation, temperature increases, changes in nutrient concentrations). Because of the worldwide distribution of reported coral reef deterioration, a multifactorial etiology must be postulated, as has been done with epizootic ulcerative syndromes of fish--but just as with fish ulcers, various forms of man-made pollution have been suspected to be partly responsible for the observed phenomena. Another (and final) example would be mass mortalities of several species of sea urchins, which have been reported within the past two decades from widely separated locations in the western hemisphere--Canada, California, and the Caribbean (Pearse et al., 1977; Scheibling and Stephenson, 1984; Lessios et al., 1984). As is the case with ulcer diseases of fish and bleaching of corals, an infectious agent has been suspected in at least some of the outbreaks, but an environmental component may also be involved.

RECOMMENDATIONS

Fish diseases are receiving greater attention as it becomes obvious that they may be significant factors in determining abundance of coastal/estuarine fish. Research on causes and effects of such diseases must be carefully planned and logically explained. While research plans vary with the nature of the disease, some common principles exist:

(1) To assure continued funding support for research and long-term disease monitoring, an effect of the disease on survival or fecundity must be demonstrated. This is difficult to accomplish, but must be an early and persistent activity of every investigation, otherwise interest in and funding of research dwindle rapidly. Too often, effects on populations are assessed only in terms of observed mass mortalities, when in reality mortality should be examined in three categories:

   (a) mass mortalities associated with acute disease outbreaks;

   (b) continuing mortalities resulting from chronic but progressive disease; and

   (c) indirect mortalities resulting from debilitation and consequent reduced survival potential--increasing the likelihood of deaths from predation or some other environmental stressor (Fig. 7).

   Additionally, population effects may result from reduced fecundity--by mechanical or physiological interference with maturation and spawning. It is this totality of disease effects on survival that must be conveyed to administrators and politicians--and not just the "oh my" or "media hype" aspects of mass mortalities.

(2) To achieve a quantitative assessment of disease effects, information must be available from field studies of total host population size. Additionally, information on incidence and mortality rates must be acquired if we are to determine disease impacts. ("Incidences" are important; "prevalences" may give a false concept of disease effects, since virulent diseases may kill quickly.)

(3) Long-term studies of fish diseases and the environment should be planned and carried out (short-term studies at the time of outbreaks have limited utility). Concurrent water quality measurements are of particular significance in any long-term program, since environmental stress is clearly a factor in many disease outbreaks.

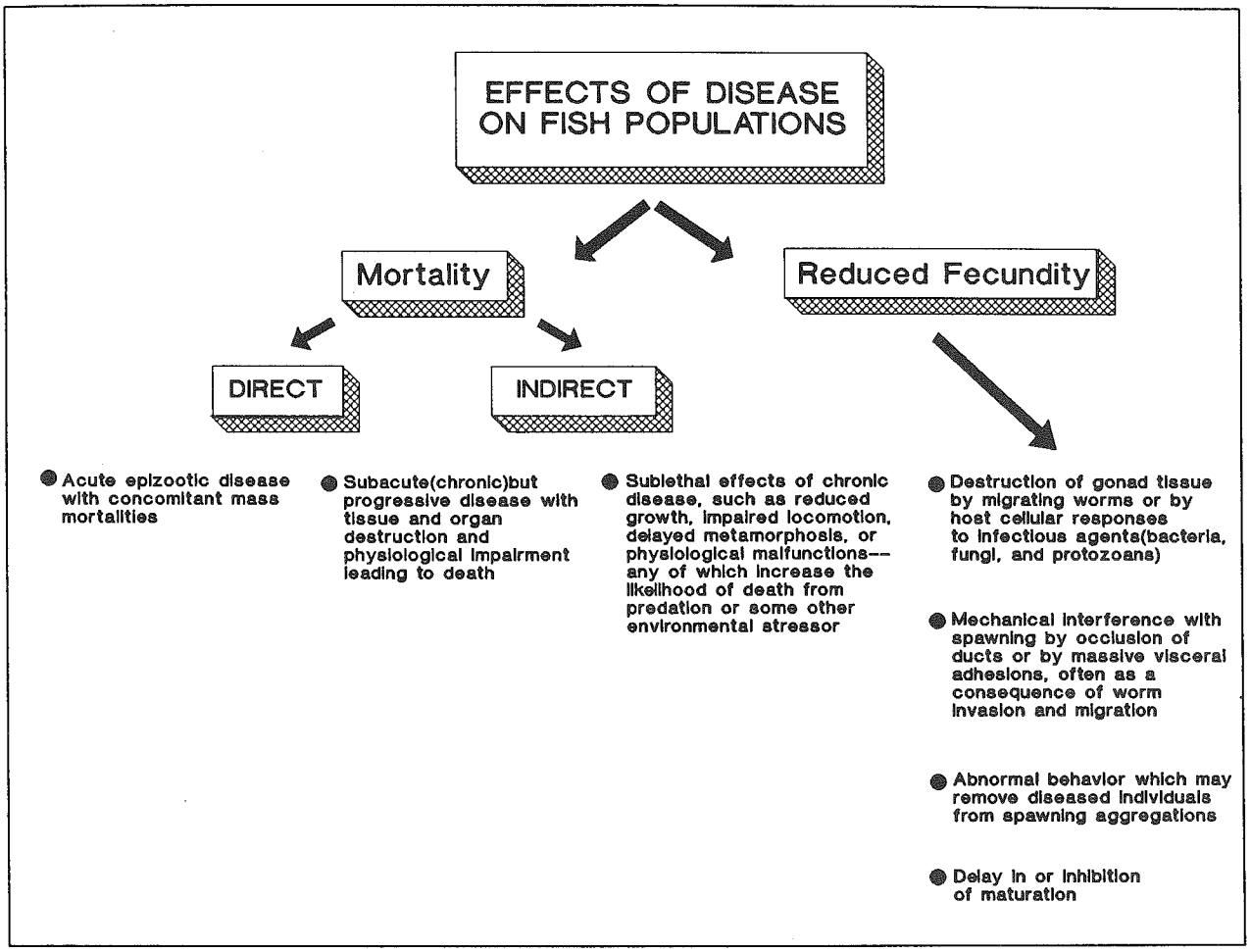

Figure 7. Effects of disease on fish populations.

(4) Field observations should be augmented by experimental work, especially oriented toward lethal and sublethal effects of disease.

(5) Disease investigations cannot be narrowly conceived--as, for example by the hiring of a competent virologist, or bacteriologist, or parasitologist (or even a mycologist). If understanding of disease effects is to be achieved, a team approach, drawing on many competencies (population dynamics, environmental assessments, statistics, immunology, and others) must be used, and adequate interaction assured (Fig. 8). Absence of such a mix of specialists will lead, as it has in the past, to all the frustrations of incomplete information--important scientifically but inconclusive for management purposes.

(6) Viral examinations should be part of every disease study, and a regional fish virological laboratory should exist or be created. Virological studies should emphasize transmission, especially possible vectors, and environmental factors which may be associated with outbreaks. Companion studies of bacterial and fungal secondary invaders should emphasize pathogenesis and mortality. Examining the literature on diseases of coastal/estuarine fish, it is apparent that viruses have often been overlooked (until recently), and secondary invaders have sometimes been proposed as primary pathogens. Decisions about precise etiology should never be hasty affairs, regardless of external pressures.

Looking more specifically at the current problem of epizootic ulcerative syndrome in Atlantic menhaden and other species on the U.S. east coast, a number of general comments could be made:

(1) Experimental studies of infectivity and pathogenicity of oomycete fungi from diseased fish should be emphasized, together with more routine studies of their taxonomy and salinity tolerances.

(2) Because of the probable multifactorial origins of ulcerative conditions in fish, a more general term, such as "epizootic ulcerative syndrome" proposed by a FAO consultation, should be used.

(3) Judging from findings in some other examples of epizootic ulcerative syndromes (ulcus syndrome in Denmark, ulcerative disease in Southeast Asia), an intensive search for possible viral infections should be conducted.

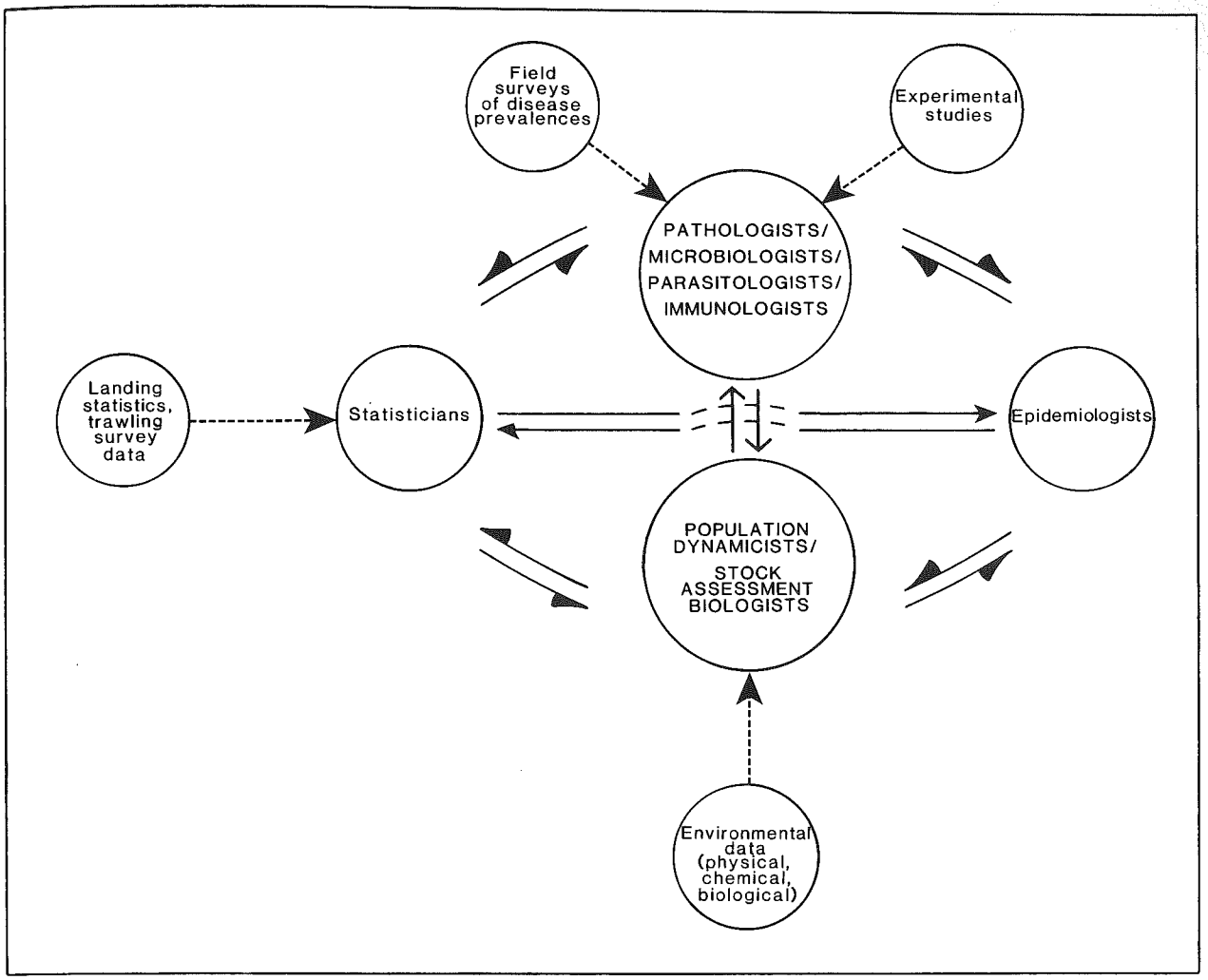

Figure 8. Components of a multidisciplinary approach to understanding effects of disease on fish populations.

(4) A "Regional Fish Disease Management Program" should develop as an outgrowth of the existing ulcer disease workshop and task force--with plans for continuity of studies and for broader considerations of other diseases after the ulcer crisis has passed. Present emphasis should remain with the ulcer condition, since this is an obvious crisis situation on the Atlantic coast.

Finally, it should be pointed out that in this or any disease investigation, it is often important to reserve judgment about etiology and effects. Are we on the Atlantic coast witnesses to a local manifestation of a worldwide panzootic of ulcerative diseases of fish, or is it just that the numbers of observers and observations of fish abnormalities are increasing, or are we simply assembling disparate events to reach conclusions that are unwarranted? Probably a soupçon of all three of these elements is involved, but these "epizootic ulcerative syndromes" are not insignificant events.

All of the foregoing suggests multiple causes of epizootic ulcerative syndromes, local and worldwide, and complex multifactorial elements in any single outbreak. We may in some instances be searching for a specific cause for a nonspecific disease sign--ulceration--which is only part of a complex disease process.

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