NOAA Technical Memorandum NMFS F/NWR - 16

Symposium: The Role of Diseases in Marine Fisheries Management

Reprinted from the Transactions of the Fiftieth North American Wildlife and Natural Resources Conference, March 15-20, 1985. Wildlife Management Institute, Washington, D.C.

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U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
National Marine Fisheries Service

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Chairman:
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Introduction

Aaron Rosenfield
National Marine Fisheries Service
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Good morning, ladies and gentlemen. My name is Aaron Rosenfield. I am the director of the National Marine Fisheries Service, Northeast Fisheries Center, Oxford Laboratory, located in Oxford, Maryland, on the Eastern Shore of the Chesapeake Bay.

In convening this symposium with Mr. Benson Drucker of our Washington office and Dr. Carl J. Sindermann, director of the Sandy Hook Laboratory, I am happy to say that we received enormous support and encouragement from NOAA's assistant administrator for fisheries, Mr. William G. Gordon.

Mr. Gordon and I go back together a long way beginning as members of our predecessor agency, the Bureau of Commercial Fisheries, under the Department of the Interior. Mr. Gordon has long been recognized worldwide as a fisheries officer. His contributions and accomplishments as a manager and administrator in the field of fisheries are many. He has not only served importantly as a headquarters official in Washington, D.C., but he has also served as deputy director of the National Marine Fisheries Service, northeast region, then as director of the region, and today as our highest fisheries administrator. I know he has some important remarks to make and it is a pleasure at this time to present Mr. Gordon.

Role of Disease in Marine Fish and Shellfish Management

William G. Gordon
National Marine Fisheries Service, NOAA
Washington, D.C.

Thank you for your kind introduction, Dr. Rosenfield. It is indeed a pleasure to be here today and to welcome so many of my colleagues and friends, all of whom have a strong and abiding interest in the theme of this meeting—the "Role of Disease in Marine Fish and Shellfish Management."

The subject of this meeting is relatively new—that is, the linkage of disease studies to fisheries management. Most meetings, symposia, workshops, and other dialogues covering the subject of diseases of marine animals are on a scientist to scientist basis with coverage primarily on the characteristics of the disease organism, how it is spread, etc., with little concern to the fishery(ies) itself. When I was approached by members of my research staff requesting support from my office to hold this symposium, I agreed, but only if certain conditions were met, namely:

1. That the information be put together and presented in such a way that it has meaning and relevance to the managers, the decision makers, as well as to plans and policy makers;
2. That the scientists go back and reexamine their data and be able to demonstrate to their audience that disease really has an important influence on the population dynamics of marine fish and shellfish, *and most importantly*,
3. That the information generated from these data will demonstrate that we may be able to do something about the effects of disease; for example, ensure product quality and human safety, predict when epidemics occur, control the spread of disease or prevent infection, and mitigate the effects of disease through environmental conservation or, if need be, habitat modification.

I must mention that many factors other than the influence of disease must be taken into consideration when developing and implementing marine fishery programs and management strategies. However, disease as a factor is often ignored or at best only marginally included in the decision and policy making and budgeting processes. Although the reports to be given here will emphasize infectious diseases—that is, disease caused by living agents such as viruses, bacteria, fungi, protozoa, and other micro- and macroparasites—I trust the audience accepts the notion and will always keep in mind that pollution and natural environmental stresses can exacerbate the effects of infectious agents on the animal. Just as importantly, animals weakened by an infection or by parasitism can be rendered more susceptible to pollution and other stress conditions, thereby possibly affecting their health, their behavior, or their reproductive capability or potential.

Finally, some infectious agents and parasites and some pollutants or contaminants, in addition to directly affecting the well being of marine animals and populations, can enter the food web to affect other living forms, including domestic animals and man!

Today, we will hear from Dr. William J. Hargis who will discuss epidemics (epizootics) in populations of marine fish and shellfish and how the information

derived from these epidemiologic studies can be used in developing fishery management plans and strategies. Dr. Hargis is well known in the marine community for his contributions to fishery biology and management of wild stocks, and for his broad background in the field of parasitology.

Dr. Hargis will be followed by Dr. Carl J. Sindermann who will discuss the role of disease in the cultivation of marine fish and shellfish. Dr. Sindermann is recognized internationally in this field and he will describe the usefulness of his experiences and information derived from them in developing national and international plans for the control of marine diseases and propose systems and present guidelines that are designed to prevent the spread of diseases from one marine or estuarine ecosystem to another.

Dr. Spencer Garrett has spent most of his professional career working in the field of marine product quality and safety, as well as developing inspection guidelines and regulations for interstate shipment of all types of marine seafood. He is particularly interested in ensuring that harmful microbial agents, parasites, and chemical contaminants do not reach and affect the health of man.

Our last speaker will be Dr. Ivar E. Strand. Dr. Strand has taken on a most difficult but essential task. He is an *economist* and not an *expert* in the field of marine diseases, or public health. However, Dr. Strand brings to us information on the economic impact of the direct and indirect effects of diseases on marine populations. His perceptions are those of a resource economist. He recognizes that real dollar amounts represent the losses or gains associated with the effects of disease, contamination, or poor quality of marine animals and their products.

In summary then, I congratulate the convenors of this symposium. I believe and I think you will agree that they have brought together an eminently qualified group to address us on this important subject and that by the end of this symposium we will have gained enormously from the reports we will hear and the discussions in which we will participate.

With that, I now turn the program over to Dr. Rosenfield who will chair the first segment of this symposium.

Remarks of the Chairman

Aaron Rosenfield
Northeast Fisheries Center
National Marine Fisheries Service
Oxford, Maryland

If I may exercise my prerogative as chairman, I would like to highlight some perplexing problems that now confront marine fisheries managers, all of which require immediate study and resolution. Some of these problem items may only be touched upon by the speakers, and in some cases the speakers may take considerable time elaborating upon these examples.

Molluscan Diseases: At least four virus entities are now known to affect marine molluscs. In addition, a number of other microbial agents, such as bacteria, fungi, and protozoa are known to cause extensive marine shellfish mortalities. There is some speculation that one of these viruses (presumably an IPN virus) may also infect fishes. Another infectious agent, although not yet confirmed as being a virus, may yet be implicated in neoplasia of soft clams, causing depletion of populations throughout the range on the northeast coast of the United States. An as yet unidentified and unnamed organism has been reported to have caused devastating mortalities of razor clams on the U.S. West Coast, thereby destroying a large and important recreational fishery. There is considerable concern in this country and throughout the world regarding systems to prevent or control the spread of shellfish pathogens, how to diagnose their presence, what quarantine and embargo systems can be put in place, what helpful laws and regulations are there to protect ecosystems and their biota, and do these laws and regulations have meaningful enforcement capability. Federal and state governments are now working together to devise sanitary codes to ensure quality and safety standards for growing waters and for shellfish are fully met, thereby protecting the health of the public. Furthermore, government agencies, state fishery commissions, industry, and regional councils are attempting to implement a molluscan shellfish health and inspection system modeled after the Department of Agriculture's Animal and Plant Health Inspection Systems (APHIS) and Poultry Improvement Plan that should markedly enhance shellfish productivity and quality.

Crustaceans: At least nine virus entities have now been described from blue crabs. In addition, five new virus disease entities have been reported from commercially important shrimp species that affect various stages of their development. The fishery manager is now confronted with such questions as to what role do these viruses play in causing epizootics (epidemics) of crustaceans, what environmental conditions exacerbate viral infections, and what must we do to prevent or mitigate the effects of viral diseases in culture systems or in the marine environment. These are but a few questions that confront the fishery manager at this very moment on the Gulf Coast of the United States, Hawaii, and throughout the world, or wherever shrimp are being cultured and transported.

Fishes: A number of parasites, both micro and macro, are now known to cause disease and affect fish health. Some of these organisms may act in an adventitious way to infect or overcome the host when the host's immune or disease resistance mechanisms are reduced by environmental stress, be these stresses, natural toxins, man-introduced contaminants, or environmental extremes. New virus diseases of fish are continually being found. Papers are being prepared to describe viral agents and other microbial agents that apparently affect clupeids and flatfish along the Middle Atlantic Coast, dramatically reducing population abundance. I'm certain you will hear more about these and other epizootics as this symposium progresses.

Probably one of the largest issues facing fishery managers today relates to the tumors and neoplasms, or cancers, if you will, that are being found in surprisingly high numbers in some marine and freshwater fish populations. These conditions may be caused by man's effluvia into the oceans and estuaries. These are issues that the fishery managers cannot ignore. It indicates that not only do fishery managers have to manage fish stocks, but they also have to manage the habitats as well. It is necessary for both the scientist and the manager to work together and look into such questions as cause and effect relationships, risk assessments, and how to calculate and model the effects of disease, and to use this information to predict disease outbreaks, or control them.

It goes without saying that people management is important also. Socioeconomics always enter the picture, as well as political considerations. Those of us working in the field of marine diseases have a lot of work to do, and in most cases because there are so few among us in the field, we have to find our own answers, have to find our own support, and do our own educating. We are indeed fortunate to have the medical, biomedical, and veterinary communities to call upon when certain needs arise. However, these groups are not all-knowing. For, after all, our patients are the inhabitants of the seas, the estuaries, and other aquatic environments such as culture ponds and tidal tributaries, and it is only occasionally possible to make definitive comparisons and draw parallels with homeothermic (warm-blooded) forms in our marine disease studies. These inhabitants belong to a wide array of phyla with many families, and classes, and subordinate taxa. All are poikilotherms (cold blooded), almost totally at the mercy of their environment and/or man's actions. Furthermore, these creatures range in size from the miniscule to the relatively gigantic (some of the larger sharks and tunas). Their diversity of shape, external and internal structure and anatomy, physiology, and biochemistry are staggering, making the logistics of capture, dissection, examination, and analyses very difficult indeed.

I have just touched upon a few highly visible problems. There are many, many more that could have been mentioned and used as examples. However, I am sure you will hear about many of these from our speakers, whom I would like now to present.

Quantitative Effects of Marine Diseases on Fish and Shellfish Populations

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Introduction

Wildlife specialists probably do not need convincing of the importance of the several components of "natural" mortality in populations dynamics of their organisms. Predators, parasites, and pathogens are regular, potent, and familiar threats and their effects are observed and counted relatively easily. Those of fish or mammals living in large lakes, inland seas, estuaries, embayments, and oceans are not readily visible and marine ecologists, fishery scientists and fishery managers generally seem unconvinced of the significance of natural mortality, especially disease, in determining availability and survival of their subject resources. Developing or enhancing their appreciation of natural factors as determiners of individual survival and population strength, i.e., the elements of natural mortality, is one objective of this review.

Quantitative studies of morbidity and mortality and their most probable causes are easiest when dealing with humans in "civilized" countries or situations. Records of births, deaths, and ages are maintained. Efforts are made to establish the health of individuals seen by medical personnel or determine and record cause(s) of death by attending physicians. In such situations great social concern results in expenditures of energy and resources in determination and enumeration of illnesses and deaths and their causes.

The same, to a somewhat lesser degree, can be said for animals and plants of economic value, i.e., pets, livestock, ornamental plants, agricultural crops, species of research interest, and trees (forests) under cultivation and management. In such managed circumstances the numbers of individuals in populations of interest can be counted, and the condition, or status of health, of individuals can be observed and causes of morbidity and mortality determined, usually. In the wild, terrestrial animals and plants can often be directly observed and enumerations are often possible; live and dead animals and plants or their condition, traces, and remains can be examined and counted.

Generally the most difficult of the ecologically and economically important organisms to observe for condition and mortality are aquatic species (or groups of species) which live in large bodies of water. The larger rivers; great inland seas or lakes such as the Great Lakes of the United States and Lake Baikal, and the Aral, Black and Caspian Seas of the Soviet Union and its borders, and many others; large estuaries, coastal waters and shelf waters of the seas (where most commercially important marine species reside); and, the deep oceans, in that order, are especially refractory to observations and enumeration.

Thus, quantitative observations of economically or ecologically valuable marine populations, surrounded and obscured as they are by water or silt and sediments, are

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comparatively more difficult and usually much more expensive than those on land. Accurate and precise population enumerations and epidemiological studies are difficult. However, there are some epidemiological or quantitative bright spots, mostly among the invertebrates. The larger, sessile molluscs whose shells often protect moribund bodies and contain their remains for brief periods after death can be counted and even examined if samples are taken frequently and carefully. For example, one can sample coastal oyster beds and secure healthy oysters, obviously moribund "gapers" (alive but sick), and recent and old "boxes" (i.e. older or recently dead individuals) with relative ease. Population levels can be managed on oyster beds by careful replacement and more tightly-controlled caged populations can be maintained in nature for experiments. By determining condition index and judicious subsampling or biopsy the observer can obtain data on many elements of health (or illness), death, and the etiological agent or other possible causes pertaining. Thus, certain aspects of individual condition, prevalence of disease, incidence of infection and degree, level, or severity of infection may be determined quantitatively in accessible populations of large molluscs. (Some aspects of condition and possible cause of death can even be determined in the remains of large fossil bivalves.) Soft-bodied or otherwise fragile or cryptic invertebrates are less easily observed and counted.

Marine vertebrates (finfishes), being mobile and generally fragile, are more difficult of enumeration than molluscs (though advanced acoustic and other survey and quantification techniques enable improved *in situ* observations of finfish) and leave fewer traces. Weak ones are quickly culled, dead bodies rapidly consumed or rotted and their fragile remains quickly scattered. Hence estimates of the effects of an epidemic's existence and mortality often are difficult.

Marine fishery population dynamicists regularly develop and employ numerical and graphical models while attempting to estimate the size and future availability of the useful resource. They also use them to estimate the actual and potential effects or mortality caused by harvesting activities (total fishing-related mortality) on those stocks. Such determinations, or estimations, are extremely useful to marine fishery managers. Without them effective management is impossible!

More particularly, such models are used in attempts to determine, in advance, the various levels of harvesting activity which may be allowed. One goal, of course, is to allow commercial catches at levels which will provide product and income for harvesters, processors, retailers and other economic participants and quality food for consumers (at affordable prices). Supplies are needed for subsistence and recreational fishermen also. A second, and even more essential, goal is to retain sufficient numbers of sexually active, mature animals to allow their reproduction and continuation of the target stock at certain required levels.

A large number of mathematical and graphical methods and models is available for making estimates of present and future stock sizes and biomass (Beverton and Holt 1957, Gulland 1969, Lackey and Nielson 1980, Pitcher and Hart 1982, Ricker 1958, 1975, Royce 1972, and Sissenwine et al. 1978). Most are relatively simple (and/or straightforward) but even those simplified versions in general use are based upon certain basic assumptions which, though specific, may be partially unverified and of varying levels of uncertainty and significance. Futher, the better ones require considerable quantities of current and long-term data regarding estimated stock-sizes, fishing mortality (usually reported as catches), natural mortality, and other

important relevant factors or parameters. Were the models to be operated at peak efficiency, these estimates or actual numbers should also include actual fishing effort expended per-unit-of-catch and all other significant elements of fishing-related mortality (i.e., deaths caused by or during the harvesting process, such as handling and culling, and gear damage) as well. Ideally, natural mortality (deaths due to non-harvesting related causes—such as predation, environmental stress of all origins, and senescence and disease of all types) also would be measured and not merely inferred or deduced. In practice, natural mortality is usually assumed at some constant level or calculated or graphically derived by some indirect method.

Natural mortality varies with age, involving not only deaths due to senescence or old age, but also those occurring among younger cohorts since large numbers of deaths occur among zygotes, embryos, larvae, and juvenile stages (see May 1973). Environmental stress, natural and anthropogenic, may also be large contributors to deaths of estuarine and marine organisms directly and indirectly (Swanson and Sindermann 1977, Sindermann 1980). The significance of major alterations in climatic or long-term weather patterns which produce abnormally cold winters, dry springs and summers (and increased salinities in estuarine and coastal waters), prolonged unusual wind patterns, altered currents and abnormal downwelling or upwelling at sea (El-Nino for example), long suspected or inferred, is now being observed and measured (Austin and Ingham 1978, Joseph 1972, Knauss 1978, Norcross 1983, Simpson 1953). Actual numerical estimates based upon observations are rarely available for either of these components of *M,* or even for *M* itself.

Considering the frequency and efficiency of commercial fishing harvesting activities, one would infer or conclude, quite naturally, that fishing mortality should be easily quantifiable with accuracy as well as precision. Quite often, however, it is not because of inadequate reporting by harvesters and, at times, port samplers. The primary causes of continued inadequacy of fishing mortality data (which really should be fishing-related mortality) are the common lack of data on culls (deaths or survivals) and gear-caused mortality (i.e., net and dredge damage resulting in death). Further, fisheries whose target resources are of small body size, such as shrimp, frequently destroy large numbers of juvenile finfishes of commercial or recreatonal value. These young fishes are captured and killed where they are most vulnerable, on their nursery grounds. No records of these mortalities exist in most cases. In some instances effort data are not recorded in useful form. Often reliable estimates of or data on fishing effort are unavailable from harvesters even where records exist. Usually, long-term catch and effort data from recreational fisheries, which for some species in some places equal or exceed commercial catches, are unavailable or weak,

Thus, accurate and/or precise data often are unavailable for use in calculations or models. Also, the equations and graphical models themselves are frequently based upon weak numbers and often involve unverified and possibly specious assumptions. Consequently, estimates of the (presumably) observable, controllable, and quantifiable element in the total mortality equation *(Z)*, fishing mortality *(F)* are, themselves, often uncertain. Since estimates of the other, and equally important element in the total mortality equation, natural mortality *(M),* are usually derived from already inaccurate, and possibly imprecise, estimates deriving from fishery catch data, they too must be of questionable accuracy and precision.

In their study of the epidemiology (epizootiology) of infectious disease in commercially important feral marine fish populations, Munro et al. (1983) stressed these points, indicating especially that natural mortality *(M)* usually involves elements of predation, reproductive debilities, food-limitations, senility, and disease, certain of which are known to be or can be quite large. Making the picture even more distressing is the general lack of quantitative data on host-parasite, host-pathogen,and other biotic and abiotic disease interactions.

Undoubtedly parasites and pathogens have attacked marine animals for eons. The interactions, challenges, and accommodations apparent in present-day species generally testify to long relationships. Existence of parasites was first recorded by Aristotle in his De Historia Animalium in 330 B.C. (McGregor 1963). Numerous reports have followed, with most appearing since 1850. Most past studies of marine parasitism have focused primarily upon the parasites of the hosts, their identification and distribution on the host as well as geographically. Interactions between host and parasite and the qualitative and quantitative effects of parasitism and disease upon individual hosts and populations have been largely ignored. Fortunately this situation is changing and marine parasitologists and disease specialists devote increasing attention to the responses of the hosts to parasitic challenges and to pathogen-related disease (Kennedy 1976). Concern is for responses of the hosts as individuals and in groups (populations). As a result, qualitative knowledge of host/parasite and host-pathogen interactions has improved considerably.

More directly to the focus of this report, quantitative efforts to evaluate the biotic factors contributing to morbidity and mortality and their effects on host groups are improving also (Aho and Kennedy 1984, Anderson 1979, 1982, Anderson and May 1979, 1980, 1982, Andrews 1984a, b, Bradley 1982, Campana 1983, Crofton 1971a, b, Eisen 1983, Esch 1977, Ford and Haskin 1982, Grizel 1983, Haskin and Ford 1982, Hassell 1982, Holmes 1982, Jones 1973, Lester 1984, McVicar 1980, 1981, May 1973, 1982, 1983, May and Anderson 1979, Möller 1984, Munro 1983, Munro et al. 1983, Ossiander and Wedemeyer 1973, Worlund and Taylor 1983). Valuable and encouraging as such efforts are, they remain hampered by the weaknesses in the fishery information mentioned above, which make realistic quantification of total mortality and its components extremely dificult.

Management of fisheries and of the natural stocks upon which they depend is important and must continue despite softness in population estimates and in quantification of *Z, F,* and *M*. Available models and estimates involving both fishing and scientific survey data must be used in population dynamics calculations regardless of weaknesses. Survival of the species and of the stocks, continued availability of suitable supplies of food and recreational fishes and shellfishes, and the economic viability of commercial and recreational fishing industries are important to the country and world. Existing fishery populations and epidemiological equations and models, necessary bases for such management, are the only available foundations of stock estimates, and for allocations.

Continuing efforts at improving data, equations, models, and management decisions based thereupon must be pressed. The need is for improvements in understanding, estimation,and quantification of all causes of natural mortality and for data on fishing mortality, including damage and deaths caused incidentally as well. Fishing mortality equations, models, and estimations can be improved relatively easily if industry and managers wish to do so. Acquisition of direct natural mortality

information will be more difficult, requiring specially planned and directed, large-scale scientific surveys, field studies, and laboratory research.

For the efforts to be increased significantly (necessary for understanding to improve) marine fishery specialists and managers must be stongly convinced of the need for such information. Encouragement of their conviction is another objective of this review. To assist in its accomplishment, several significant examples of host-parasite-disease interactions are presented and discussed below. Examples, stressing well-documented or recently discovered disease situations, include three invertebrates, mollusc (the Atlantic oyster, *Crassostrea virginica)* and two crustaceans (the blue crab, *Callinectes sapidus,* and the Tanner crab, *Chionoecetes bairdi)* and four finfishes, the herring *(Clupea harengus),* the summer flounder *(Paralichthys dentatus),* the white perch *(Morone americana)* and the menhaden *(Brevoortia tyrannus).*

Invertebrates

Invertebrates are of especial interest to marine epidemiologists because of the apparent lack of ability in individuals to develop acquired immunity in response to challenge from pathogens. This affects their vulnerability as well as the course, severity, and outcome of any disease they may fall prey to. The extensive records of sickness and mortality of certain economically important ones are valuable epidemiological resources.

The Atlantic oyster, Crassostrea virginica, *versus MSX, or Delaware Bay Disease.*

Perhaps the most intensively studied of all marine invertebrates are oysters of the genera *Ostrea* and *Crassostrea.* Employed as food worldwide for centuries, their socioeconomic importance probably exceeds that of any other molluscan group. Problems associated with the oyster have occupied many practical culturists and scientists since recorded Roman times at least. In the United States, records of oyster production and problems extend well into the nineteeth century (Brooks 1905, Galtsoff 1964, Haven et al. 1978).

Tighly-closing calcarious valves prevent dessication and enable oysters to survive long periods of time out of water. Hence, they have been transferred from one body of water to another the world around. Movement of populations, species, and genera coastwide and across continents and oceans have resulted in introductons of alien hosts, competitors, predators, parasites, and pathogens into new areas (Hargis and DuPuy, in press, Sindermann 1970).

Oysters are hardy, extensively harvested and sampled, under culture, and frequently transplanted great distances, and they occur mostly in relatively accessible shallow estuaries and coastal waters of the world. Sessile and easily manipulated and sampled, they are readily observed in the wild, under culture, and in the laboratory. Further, even in the wild those of greatest economic importance occur in beds or shoals consisting of many relatively-long lived individuals. Such oyster communities may become fairly stable and attract large number of taxonomically-varied associates, including commensals, mutuals, competitors, predators, parasites, and facultative and obligate pathogens. As filter-feeders they take in large volumes of particle-laden water in the processes of feeding and respiration. As an example, a

mature (4-inch, or 10.16 cm, shell-length) *Crassostrea virginica,* the commercially valuable native oyster of the northwest Atlantic coast, can pump and process as much as 450 litres (or up to 119 gallons) of water in a 24-hour period. (Rates vary, of course, and are often much lower. Three hundred l/day, or 79.26 gallons, is considered a "safe" average, Morales, pers. comm.). Because they usually occur in large masses of individuals, they are capable of creating significant currents around their beds. Judging from the volume of water moved during laboratory observations and from measurements of water processed and the large amounts of feces (ingested and processed materials) and pseudofeces (consisting of agglomerated particles not taken into the gut) deposited by a single oyster, the opportunity for accumulation and ingestion of large numbers of cysts, eggs, and other potentially infective particles of varous parasites and pathogens is great.

Were one searching for a marine organism upon which mortality models (including fishing-related mortality and natural mortality of all types) could be based, the oyster should be hard to surpass. Records indicate epidemics of various sorts in wild and cultivated populations for decades. Extensive disease studies have been reported (i.e., Andrews 1982, 1984a, b, Ford and Haskin 1982, Galtsoff 1964, Hargis 1985a, Haskin and Ford 1982, Prytherch 1931, and many others).

Among the recorded occurrences of large-scale mortalities of oysters in the mid-Atlantic region, one, occurring during the winter of 1929-30 in the lower Chesapeake region (Virginia), is said to have caused the loss of over one-half million bushels of oysters (and one-half-million dollars), attributed to pathogen-related disease and environmental stress (Prytherch 1931). Mortalities of oysters in the Chesapeake region continued sporadically, prompting more intensive studies of the factors involved. Though interrupted by World War II, researches were resumed and intensified in the late 1940s at the Virginia Fisheries Laboratory in Yorktown (forerunner of present-day VIMS) by Drs. J. D. Andrews, Willis Hewitt, and others. Discovery of the fungus-disease (previously known from the Gulf of Mexico) called "Dermo" after *Dermocystidium,* the genus to which it was then ascribed, in oysters of the lower Bay was a milestone in Chesapeake Bay disease studies. The etiological agent, now called *Perkinsus marinus,* was found to be a cause of continuing chronic-level mortalities in oysters in the region.

Of greater significance recently has been the occurrence of the more virulent MSX disease in 1959 in these same areas and in Delaware Bay, where it had apparently started two years earlier in 1957 (Andrews 1982, Andrews 1984a, b, Hargis 1985b). According to Ford and Haskin (1982) 90-95 percent of oysters in lower Delaware Bay died shortly after the mortality began. Forewarned, Chesapeake Bay scientists anticipated mortalities in local populations and developed special disease survey programs to record their onset and progress. They were not disappointed, and by 1960 mortalities in the lower Chesapeake reached as high as 80 percent. Subsequent research by scientists from all Delaware and Chesapeake Bay states and the National Marine Fisheries Service established the most-probable etiological agent as the protozoan *Haplosporidium nelsoni* (Phylum Ascetospora: Class Stellatosporea), which has been the subject of intensive study since. Though Koch's postulates have not yet been satisfied for MSX (or Delaware Bay), disease, epidemiological, and correlated histopathological evidence is so strong as to be almost undeniable that *H. nelsoni* is the cause. This situation is not unusual among marine diseases, the effectors of many of which have not yet been rigorously proven. Efforts continue to

isolate the most-probable etiologic agent, use the isolate to produce the disease in disease-free susceptibles, and then reisolate it to remove all possible doubt.

More to our immediate purposes however, annual harvesting data obtained before, during, and after the onset of the disease in Delaware and Chesapeake Bays show severe reductions in yields due to resulting mortalities. Independent scientific sampling programs, always useful in augmenting and verifying commercial catch records, confirms the decimation of populations on cultured and "feral" beds in the higher salinity portions (i.e., >15-18 ‰) (Haskin and Ford 1982). As Sindermann (1968) explained, oyster production in Delaware Bay, where most oysters were cultured on high-salinity beds, plummeted in 1957 and has remained low since (Figure 1). Virginia production also declined during 1959-60 and has remained depressed (Figure 2). Private (planted or cultured) beds in Virginia subsided after 1959 and, though compensating plantings made on private beds in lower-salinity regions prevented total yields from dropping even further, the high-salinity beds most heavily infested were abandoned and have been essentially out of production since (Figure 3) (Andrews 1984a, Hargis 1985b, Haven et al. 1978, Sindermann 1968). These disease-related declines in oyster production in Delaware and Chesapeake Bays added greatly to the recent abrupt downward perturbations in the long-declining curve of east-coast U.S. oyster production (Figure 4).

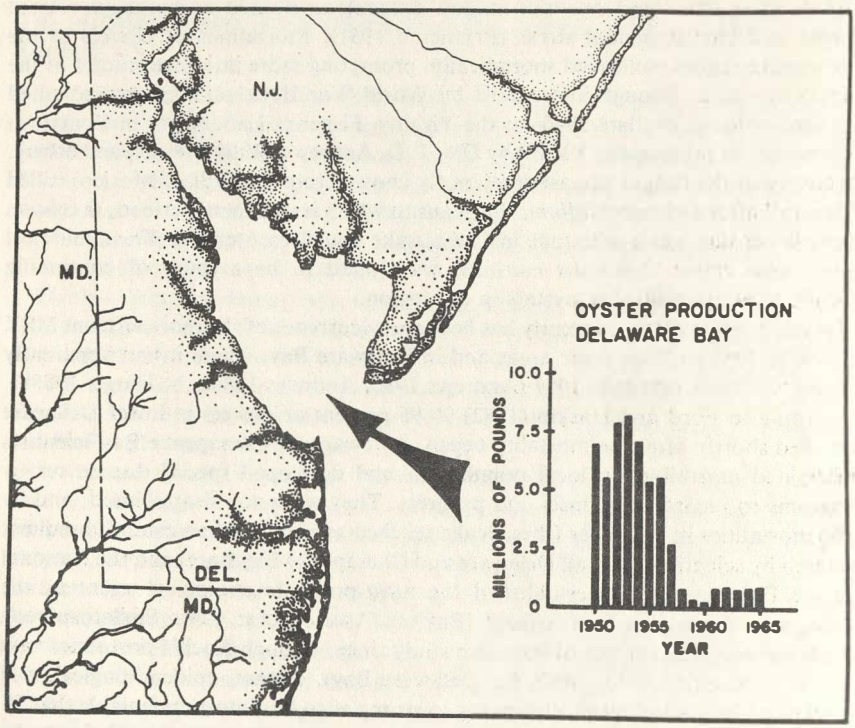


Figure 1. Oyster production in New Jersey waters of Delaware Bay, 1950-65 (from Sindermann 1968).

Infected areas in Delaware Bay show signs of recovery, probably due to development of resistance in progeny of survivors (Andrews 1984a, Ford and Haskin 1982). Recent data (Andrews, pers. comm.) indicates that MSX disease may be showing signs of remission in certain previously heavily infested areas in the lower Chesapeake also. Whether or not these signs of surcease precede a longer period of remission or even disappearance, a quarter-century of disease-caused scarcities and difficulties and continued overfishing have caused resource reductions and economic losses of great magnitude (see Strand and Lipton 1985).

As shown, considerable data have been gathered by scientists and fishery managers demonstrating the effects of disease-related epidemics on wild and cultured

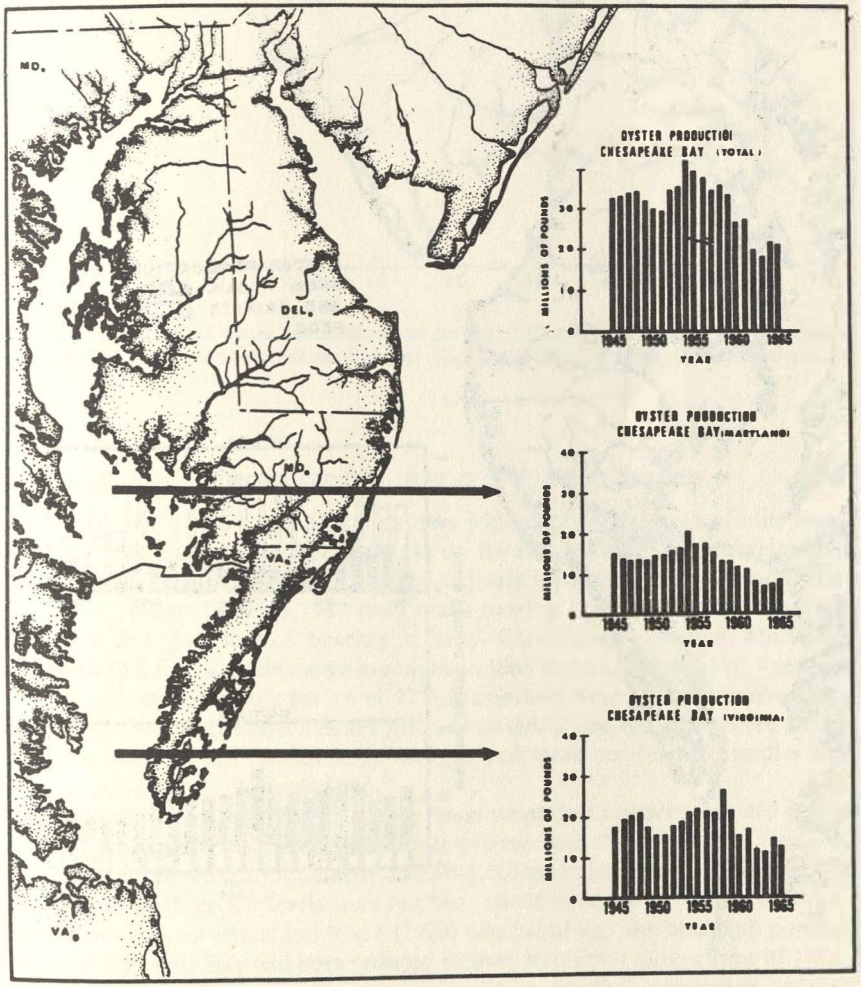


Figure 2. Oyster production in Maryland and Virginia waters of Chesapeake Bay 1945-65 (from Sindermann 1968).

Crassostrea virginica populations and on the dependent commercial fisheries. Corollary information from field and laboratory observations on reproduction, predation, survival, disease-levels, and catches provide perhaps the best intensive long-term data base on the quantitative effects of disease on estuarine/marine population ever accumulated. It has been widely publicized in public and scientific media and the results employed by public and private managers alike. Undoubtedly, much remains to be learned regarding the effects of known disease incidents upon these mollusc populations. However, the fishery and disease data available should enable population dynamicists and epidemioloigists alike to test and refine concepts of F and M.

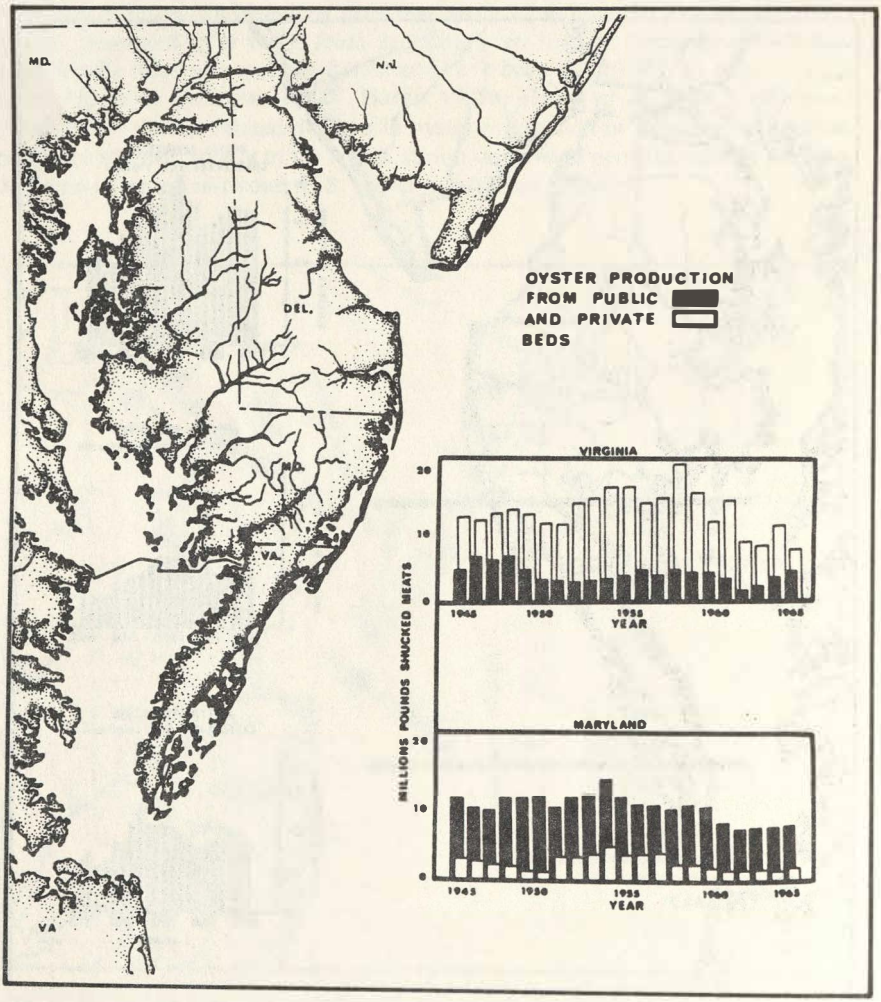


Figure 3. Oyster production from public and private beds, Maryland and Virginia, 1945-65 (from Sindermann 1968).

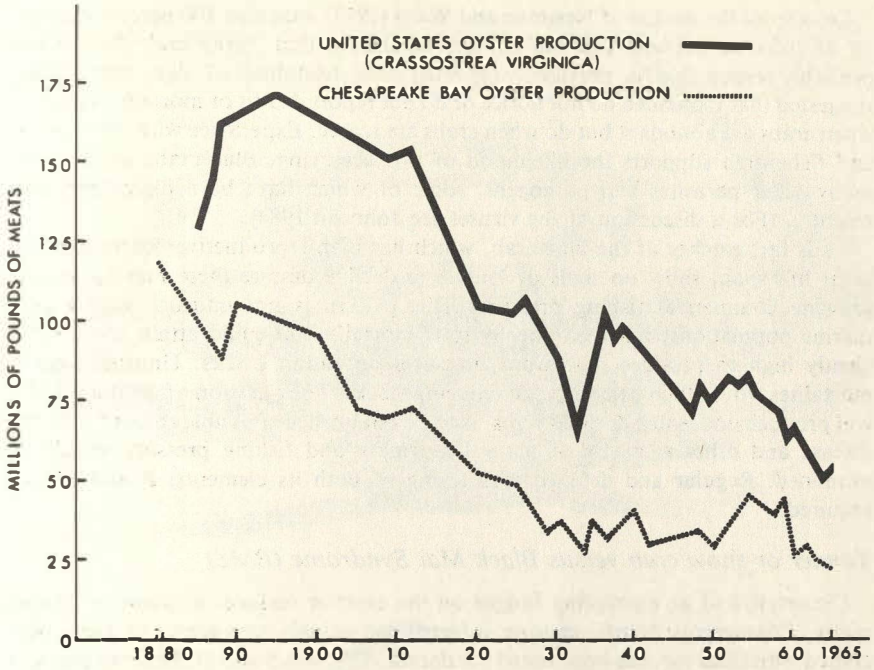


Figure 4. Production of oyster *(Crassostrea virginica)* in Chesapeake Bay and in the United States, 1880 to 1965. Modified from Galtsoff (1956) and Engle (1966). (From Sindermann 1968.)

The blue crab, Callinectes sapidus, *versus Gray Crab Disease*

This condition of gray exoskeletal patches with associated deaths of blue crabs was first noted in soft-crab shedding tanks on the Eastern Shore of Maryland and Virginia in 1965. Up to 30 percent of the animals involved in these commercial-shedding operations died. In 1967 dead crabs bearing similar gray discolorations appeared in crab pots and on beaches in South Carolina and Georgia. Moribund crabs found in South Carolina were examined in 1968 and a small number, 3 percent in one sample and 5 percent (or 14 of 277) in another, were found infected by an ameboid organism in the host's hemolymph or blood. These epizootics were attributed to the protozoan, *Paramoeba perniciosa,* and more careful field studies and laboratory experiments were planned.

In the laboratory it was found that the hemolymph from heavily infested (gray-appearing) crabs would not clot and that all animals with "very heavy" infections (i.e., a ratio of amoebae to total cells of 0.96-1.00) of *P. perniciosa* died within 24 hours after those infection levels were reached. Based upon survivorship in laboratory-held animals, Newman and Ward (1973) concluded that the blue crab population of Chincoteague Bay had been reduced by over 30 percent during June of 1971. A relationship between death and rising water temperatures is suspected because deaths increased as the water warmed.

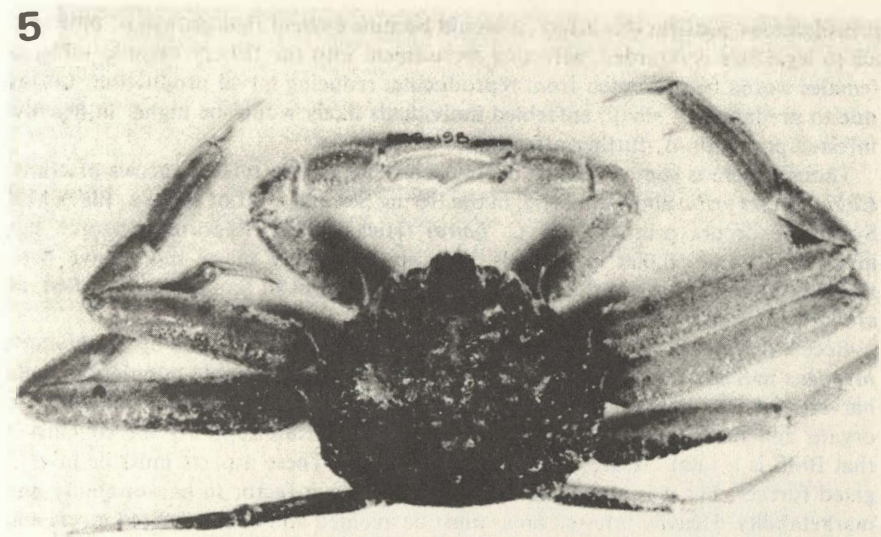
Data from the studies of Newman and Ward (1973) indicated 100 percent morbidity of infected animals and the authors concluded that "gray crab disease was probably responsible for previously reported mass mortalities of blue crabs." They suggested that fishermen do not notice or do not report deaths or mortalities in years when crabs are abundant but do when crabs are scarce. Experience with the fisheries and fisherman supports the likelihood of this occurring. Blue crabs are hosts to many other parasites and pathogens, some of which have been discovered only recently. (For a discussion of the viruses see Johnson 1984).

Thus far, catches of the blue crab, which has high reproductive potential and a short life span, show no signs of long-term decline despite these mortalities and growing commercial fishing pressure. This pattern is not unusual among such marine populations, but theoretically total mortality *(Z)* could attain levels sufficiently high to produce downward pressures on future stocks. Unusual natural mortalites would then be decisive in reducing stocks. The question of what level of Z will produce noticeable declines remains to be established. Possible roles of both the disease and other elements of natural mortality and fishing pressure should be examined. Regular and detailed monitoring of both its elements, F and M, are required.

Tanner or snow crab versus Black Mat Syndrome (BMS)

Occurrence of an encrusting fungus on the exterior surfaces of snow or Tanner crabs, *Chionecetes bairdi*, causing infected individuals to appear to have been coated with thick tar, has been noted for decades (Figures 5 and 6). Because particles tended to flake off during processing and contaminate the meats, the fungus affected marketability of the product. Processors avoided heavily-encrusted crabs and harvesters heavily-infested areas, and the condition caused significant reduction of useful resources and losses due to rejection of product. Black Mat Syndrome was regarded for a time as being primarily an economic nuisance (Sparks 1982, 1984).

During early studies the causative organism was misidentified as an ubiquitous soil fungus (*Phoma fimenti*). Later it was found to be a new species and named *Trichomaris invadens* (Hibbits et al. 1981). It infected mostly *C. bairdi* and occurred mainly in certain geographical areas and was believed not to invade internal organs of the hosts. Through histological studies the fungus parasite was found to be a more serious pathogen, penetrating the exoskeleton, proliferating in subepidermal tissues, and invading other internal organs (Sparks and Hibbits 1979). It also caused a statistically significant shift in the blood picture of infected crabs. Individuals with greater numbers of internal organs showed more pronounced shifts of differential hemocyte counts, featuring marked increases in percentages of eosinophilic granulocytes in those infected (Mix and Sparks 1980). Gonads in heavily-infected females contain necrotic and disintegrating ova. That reproductive potential is reduced is demonstrated by reduction of egg-clutch sizes in infected females and the fact that >90% of barren females had BMS (Hicks 1982). Gill lamellae are destroyed and replaced by proliferating hyphae. Presence of large numbers of "skip molt" crabs in infected populations indicates that molting is probably hindered. Should later



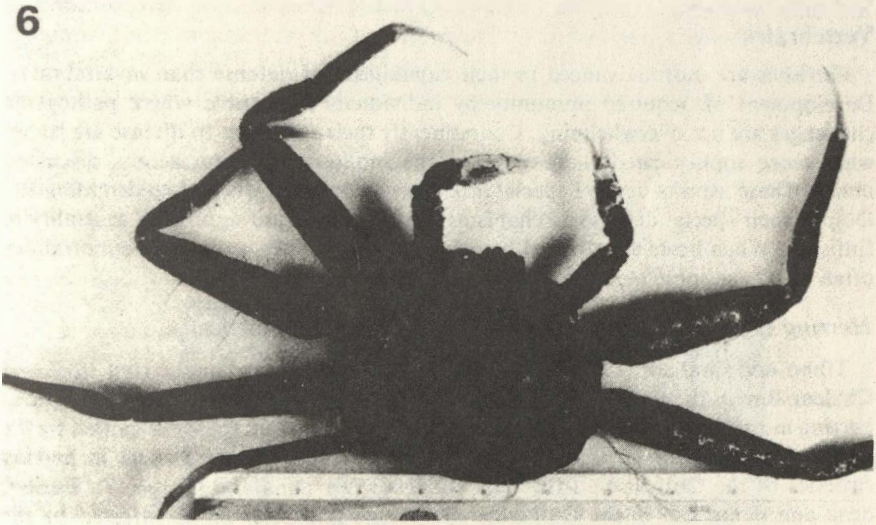


Figure 5. A Tanner crab with black mat syndrome (BMS). Most of the dorsal cephalothorax is covered and the encrustation is spreading to the legs.Most of the round structures on the 3rd right walking leg are epibionts, not fruiting bodies (after Sparks 1984).

Figure 6. A heavily encrusted crab, almost completely covered by fungal hyphae and fruiting bodies (BMS) (after Sparks 1984).

investigations confirm this effect, it would become evident that growth of individuals to legal size is retarded, affecting recruitment into the fishery because infected females would be prevented from reproducing, reducing larval production. Losses due to predation of small, enfeebled individuals likely would be higher in heavily-infested populations, further affecting total numbers.

Though there is some evidence of its occurrence on the related species of crabs, *Chionoecetes grilio* and *C. tanneri*, in the Bering Sea and Gulf of Alaska, Black Mat Syndrome occurs primarily on *C. bairdi* (Hicks 1982). Reported research has involved around 50,000 individuals. (By now probably many more have been studied). In some areas of the Gulf of Alaska 65 percent of females are infected, as are large numbers of males.

Recent declines in fishery catches from areas known to be infected by *Trichomaris invadens* indicate that it may be a factor in reducing harvestable populations (and harvests) of Tanner crabs. Widespread invasiveness of important internal tissue and organs and the weakening of host defensive mechanisms supports the conclusion that BMS is a fatal disease and is causing declines. These aspects must be investigated further, but it is already obvious that BMS is a factor in harvestability and marketability. Heavily infested areas must be avoided and contaminated meats and legs cannot be marketed.

Vertebrates

Finfishes are more advanced in their capabilities of defense than invertebrates. Development of acquired immunity by individuals is possible where pathogenic challenges are not overwhelming. Consequently their responses to disease are somewhat more sophisticated than those of the molluscs and crustaceans discussed above. These aspects deserve special attention by pathologists and epidemiologists. Despite their special defense mechanisms, disease can cause significant mortality in finfishes. When hosts are stressed by other factors, sudden and massive mortalities often result, as some of the following case histories will show.

Herring (Clupea harengus) *versus* Ichthyophonus *Disease*

Tibbo and Graham (1963) reviewed the effects of disease on herring stocks of Chaleur Bay in the Gulf of St. Lawrence. This study of the heavy and widespread herring mortalities which occurred in 1954 concluded that an epidemic caused by the fungus, *Ichthyophonus hoferi*, was the primary cause of the decline in herring fisheries of the Gulf and on the west coast of Newfoundland (Figure 7). Earlier, dead and dying fish in the Gulf of St. Lawrence had been found infected by the organism. Also, Sindermann (1958) had demonstrated that the pathogen caused systemic infections which, in the acute phase, resulted in the deaths of the fishes involved.

During those herring kills, moribund individuals were seen in the waters and dead fish littered the beaches and were caught in the trawls of ground-fish draggers. The number and rate of deaths must have been immense since predators and scavengers could not remove them in timely fashion. The magnitude of the mortality and its effects on the abundance of herring could only be estimated roughly. Sindermann (1958) conservatively estimated that one-half of the mature *C. harengus* in the Gulf of St. Lawrence were destroyed during the period 1954-1956.

From careful examinations of available data, Tibbo and Graham (1963) attributed an observed decrease in mean age and reduction in number of year-classes represented in commercial catches following the epidemic to the disease. They also concluded that the faster-than-normal growth rate noted in surviving herring stocks resulted from the decrease in competition among survivors for food and other density-dependent factors due to removals by death from those stocks. A decrease in the abundance of herring larvae following the epidemic (epizootic) was also detected and these authors hypothesized a connection. Interestingly, they observed that Kohler (1961) has associated a rapid increase in mean-lengths of 6-10 year old Atlantic cod (*Gadus morhua*) in the Gulf of St. Lawrence with the abundance of moribund [and recently dead?] herring, normally less readily available to them, upon which they had fed. Thus, though numbers and probably total biomass of the herring were reduced, faster and greater growth of surviving individuals of host populations and of their predators and scavengers likely compensated somewhat. These compensatory ecological effects did little to reduce the economic losses of local herring fishermen.

In reaching the conclusion that this pathogen had, indeed, caused decimation of herring stocks, a number of environmental and biological factors, which could have theoretically produced the decline, were carefully considered and ruled out by Tibbo and Graham (1963). These authors concluded by saying that "the effect of disease on a marine fish population is usually included in estimating mortality rates but seldom is it accompanied by factual information." They also indicated that their "study provides some information of the ecological consequences of an epizootic in herring *and urged* [emphasis mine]" continuing efforts to describe the sequence of

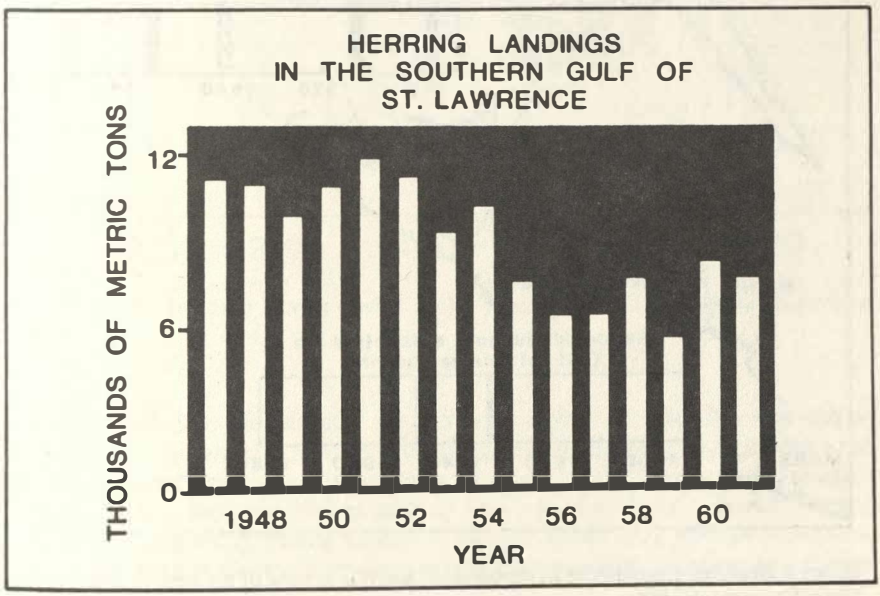


Figure 7. Herring landings in the southern Gulf of St. Lawrence (from Sindermann 1985).

events in similar disease situations, a recommendation that must be echoed. Their competent study, one of the most thorough reviews of events before, during, and after a major epidemic in an important finfish population, can be regarded as a classic amongst such works. If future efforts are as thorough, knowledge of the quantitative role of disease in wild marine fishes will be greatly improved.

Ichthyophonus disease also probably caused recorded declines of populations and fishery catches in the Gulf of Maine in the 1930s and 1940s and in both Gulfs earlier (Figure 8). The mortalities of the mid-1950s are believed responsible for the marked reductions of herring catches in the North Atlantic depicted in Figure 9 (Sindermann 1979, 1985a).

This fungus disease has killed alewives (*Alosa pseudoharengus*) and mackerel (*Scomber scombrus*), but not cod (Sindermann 1966); therefore it is not specific to the herring. Nor is it confined to the Gulf of Maine and St. Lawrence or even the western North Atlantic. It is an old and persistent disease, having been reported as early as 1893. McVicar (1982) conducted a review of this genus of pathogenic fungus and reported that other species, such as the ocean pout (*Zoarces anguillaris*) had been found infected in deep cooler waters off of Massachusetts. It has been detected in plaice (*Pleuronectes platessa*), haddock (*Melanogrammus aeglefinus*) and mackerel on the west and east coasts of Scotland, around the Isle of Man, and in the

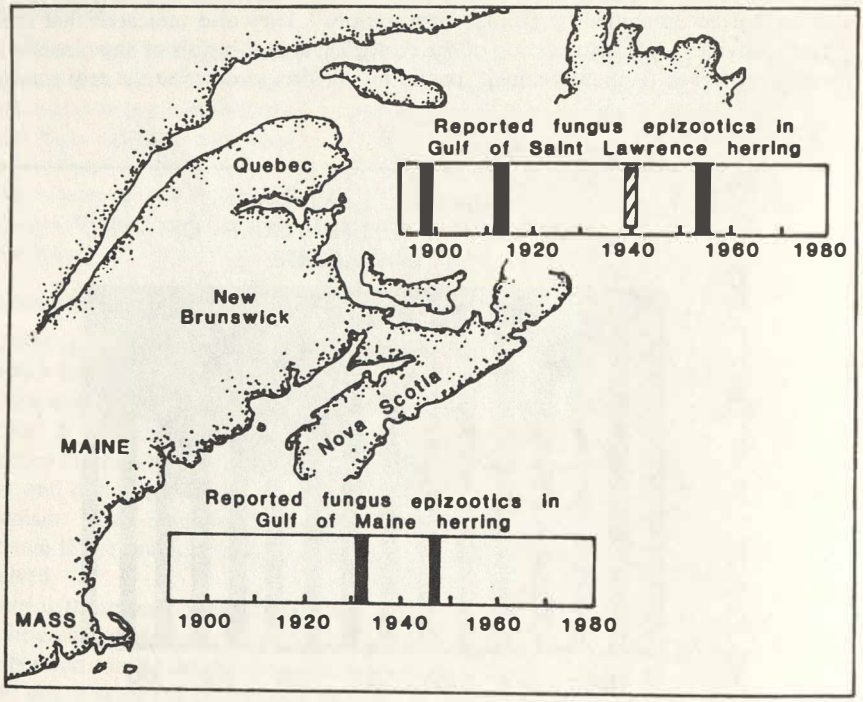


Figure 8. Reported epizootics of *Ichthyophonus hoferi* in herring of the western North Atlantic (from Sindermann 1985).

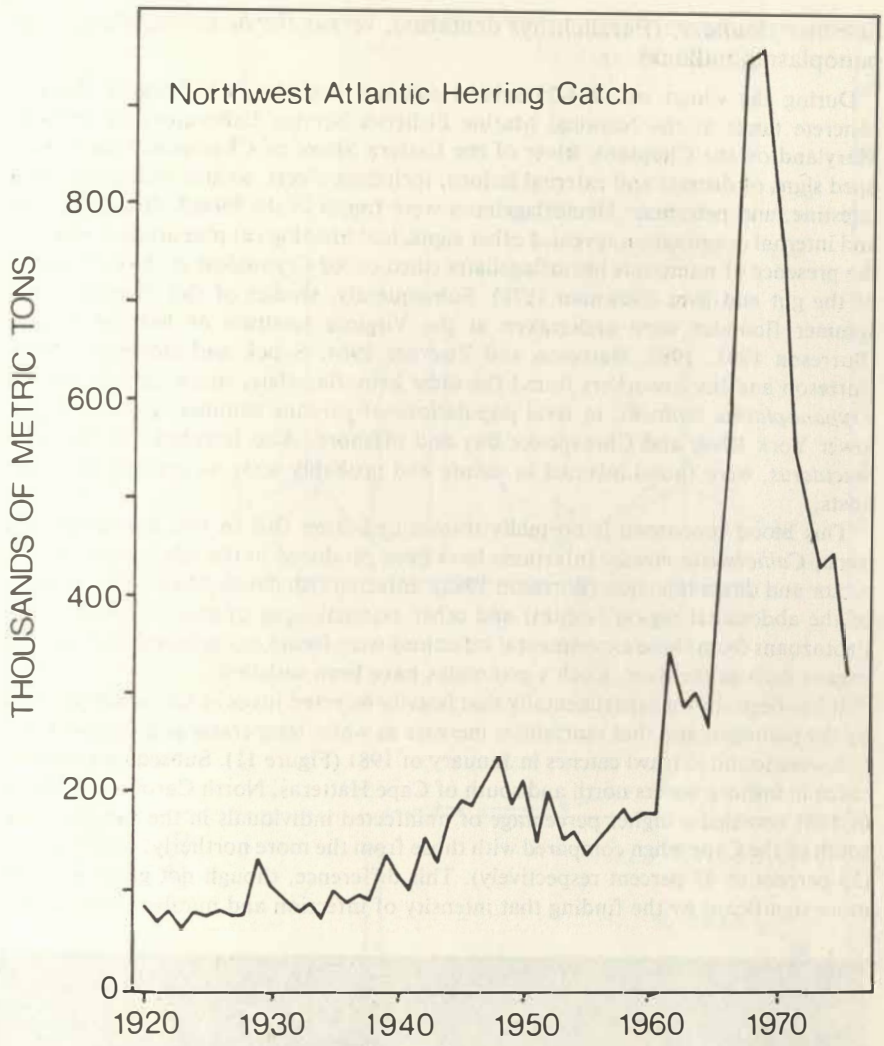


Figure 9. Recent trends in herring catches in the western North Atlantic (from Sindermann 1985).

northern North Sea. He indicated that no other records of mass fish kills due to *Ichthyophonus hoferi* have been reported in natural populations since the 1950s but has found significant annual mortalities of plaice (>55 percent) off northern Scotland. It has been reported as causing heavy mortalities of cultured rainbow trout (*Salmo gairdneri*), killing almost 50 percent of the total stock of one farm. Clearly this fungus disease requires continued attention of scientists and fishery managers alike.

Summer flounder, (Paralichthys dentatus), *versus the hemoflagellate,* Trypanoplasma bullocki

During the winter of 1974-75 several summer flounder, *P. dentatus*, held in concrete tanks at the National Marine Fisheries Service Laboratory in Oxford, Maryland on the Choptank River of the Eastern Shore of Chesapeake Bay developed signs of distress and external lesions, including ulcers, ascites with protruding intestine, and petechiae. Hemoflagellates were found in the blood. Gross external and internal examination revealed other signs, and histological preparations showed the presence of numerous hemoflagellates (then called *Cryptobia*) in the submucosa of the gut and liver (Newman 1978). Subsequently, studies of this disease of the summer flounder were undertaken at the Virginia Institute of Marine Science (Burreson 1981, 1982, Burreson and Zwerner 1984, Sypek and Burreson 1983). Burreson and his coworkers found the same hemoflagellate, more recently named *Trypanoplasma bullocki*, in feral populations of juvenile summer flounder in the lower York River and Chesapeake Bay and offshore. Also hogchokers, *Trinectes maculatus*, were found infected in nature and probably serve as resident reservoir hosts.

This blood protozoan is normally transmitted from fish to fish by the aquatic leech, *Calliobdella vivida*. Infections have been produced in the laboratory via this vector and direct injection (Burreson 1982). Infected fish developed severe-swellings of the abdominal region (ascites) and other external signs of distress (Figure 10). Protozoans from those experimental infections were found in the blood and internal organs such as the liver. Koch's postulates have been satisfied.

It has been shown experimentally that heavily-infected juvenile hosts can be killed by the pathogen and that mortalities increase as water temperatures decrease. Dead fish were found in trawl catches in January of 1981 (Figure 11). Subsequent samples taken in inshore waters north and south of Cape Hatteras, North Carolina in March of 1981 revealed a higher percentage of uninfected individuals in the sample taken south of the Cape when compared with those from the more northerly, colder waters (53 percent to 47 percent respectively). This difference, though not great, is made more significant by the finding that intensity of infection and numbers with ascites

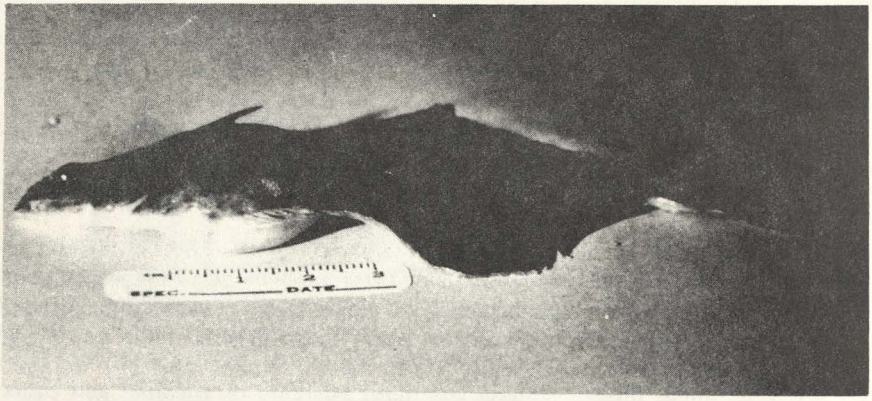


Figure 10. Juvenile Summer Flounder *(Paralichthys dentatus)* with ascites due to *T. bullocki* (Burreson photo).

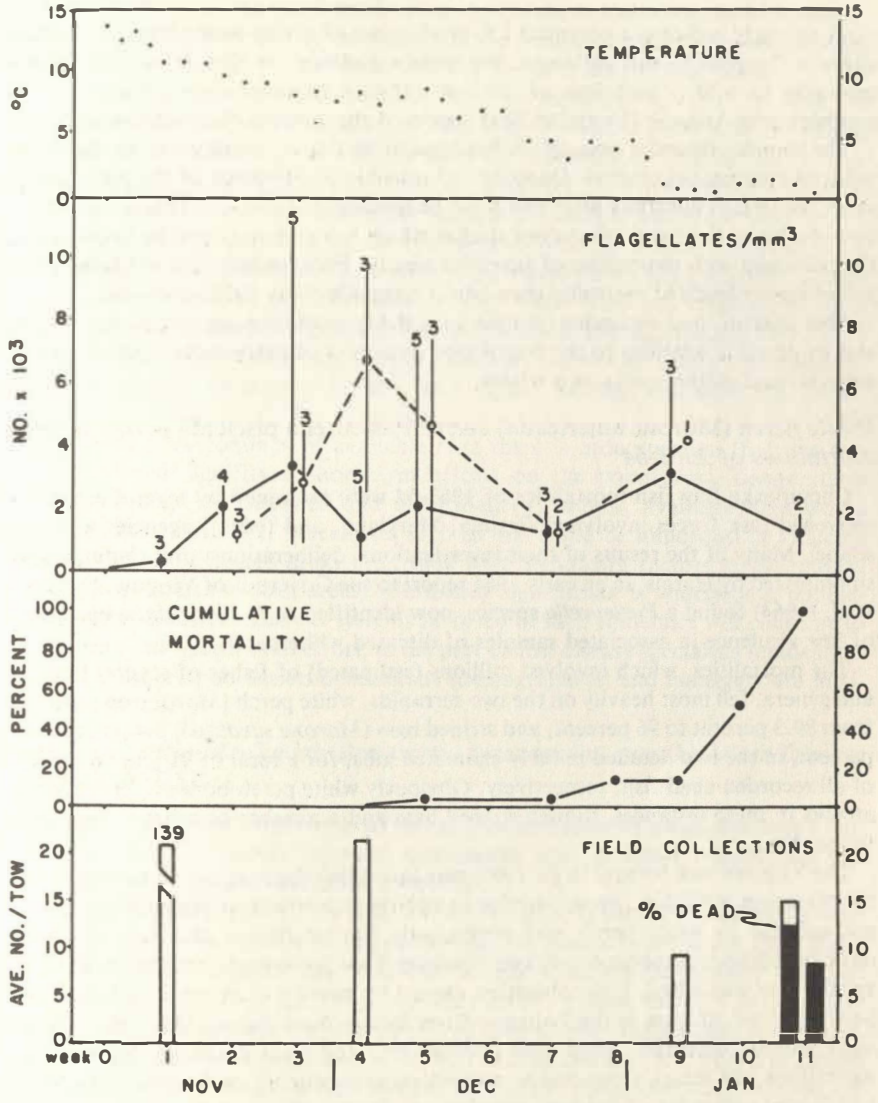


Figure 11. Results of experimental studies and field collections. Solid line in Flagellates/mm^3 connects means of syringe inoculated fish, dashed line connects means of leech inoculated fish. Vertical lines are ranges. Numbers above ranges are number of fish examined (from Burreson 1981).

were higher in the sample from the cooler waters north of Cape Hatteras. These findings confirms a relationship between pathogenicity of the parasite, or increased susceptibility of the host, and low water temperatures.

The higher incidence of parasites and increased death rates of infected hosts in laboratory populations and the evidence of morbidity and mortality in feral popula-

tions strongly indicate a potential for production of severe mortalities in juvenile summer flounder by this pathogen. Burreson's studies show that *T. bullocki* causes mortality in wild populations of juvenile summer flounder during winter in the southern mid-Atlantic (Virginian Sea) region of the western North Atlantic.

The summer flounder population has been in decline in recent years, as shown by reduced commercial catches. Questions of possible involvement of the pathogen in these reductions naturally arise and must be investigated further. This is one of the few combined field and laboratory studies which has demonstrated involvement of the pathogen with mortalities of juveniles clearly. Because juveniles are believed to suffer higher levels of mortality than adults normally (May 1973), this case deserves further careful, and expanded, studies into the quantitative aspects of the disease and its possible relations to the population dynamics of prerecruits, year classes or cohorts, and of the stocks as a whole.

White perch (Morone americana) *versus* Pasteurella piscicida *in the summer mortalities of 1963-64*

Chesapeake Bay fish mortalities of 1963-64 were examined by several especially convened task forces involving Virginia, Maryland, and federal agencies and personnel. Many of the results of their investigations, deliberations, and findings were summarized by Hargis, in an early 1965 report to the Governor of Virginia. Snieszko et al. (1964) found a *Pasteurella* species, now identified as *P. piscicida*, a bacterium of low virulence in associated samples of diseased white perch.

The mortalities, which involved millions (estimated) of fishes of several families and genera, fell most heavily on the two serranids, white perch (*Morone americana*), from 89.3 percent to 96 percent, and striped bass (*Morone saxatilis*), 2.4 percent to 0 percent, in the two detailed reliably-estimated kills, for a total of 91 and 96 percent of all recorded dead fish, respectively. Obviously white perch bore the brunt of the attacks in these instances, though striped bass and a number of other species were found dead.

The Virginia task force (Hargis 1965) concluded that the mortality was brought on by "compression" (i.e., crowding due to upriver movement of high salinity waters not suitable to white perch and ecologically similar fishes) and possibly other unfavorable hydrographic conditions resulting from prolonged drought in the Chesapeake Bay watershed. Eutrophication caused by over-fertilization could also have been involved, at least in the Potomac River below Washington, D.C. where dense algal blooms occurred. They also inferred that the final cause of many of the mortalities was attack from one or more disease-producing pathogens exacerbated by lowered resistance of the host populations. *Pasteurella piscicida* recovered from white perch and striped bass (and other fishes) in 1964, but not from normal white perch in 1965, probably was the etiological agent. Evidently it produced extensive bacteremia in infected individuals. The normally low virulence of the bacterium coupled with observations of stressful environmental conditions in the areas showing greatest kills in 1963 tend to confirm the conclusion that both were active in producing this massive mortality episode.

An estimated several million fish were involved in the 1963 mortalities involving pre-adult and adult fish. Sindermann (1970), observing that landings in 1964 (the year immediately following the 1963 epidemic) were as little as 622,000 lbs. (282,139 kg) compared with a 3-year average of 1,500,000 lbs. (680,400 kg) for the preceding

years, concluded that they had produced a significant reduction in fishable stocks. However, Hargis (1965) had noted that catches in pound nets and by the recreational fishermen operating in the kill areas did not seem to be reduced because of the mortalities. This apparent inconsistency may be explained by the fact that the fishermen in question were operating in an area into which the white perch, striped bass, and other fishes had been compressed by upstream moving higher (and to white perch—intolerable) salinities. The "compression" or crowding of large numbers of individual fishes into a smaller volume of suitable water not only set up conditions of predisposition to the development of bacterial disease but made more fish available to the fishermen in the area than would have been normally. Consequently, the mortalities, extensive though they were, were not reflected in local catches. This finding would indicate that to determine the significance of such an epidemic to the fishery one would have to examine system-wide catches (as related to effort) rather than local ones for the years before, during, and after the disease incident.

Despite the shortcomings of available data there is little question that this epidemic produced significant short-term effects on the population. Longer term effects remain to be examined and quantified (if, indeed, available data would support additional study). Mortalities such as this could be important in reducing fishable stocks in affected areas (i.e., they were significant factors in the natural mortality (M) of fish in such areas). Until their significance is understood, population estimates and management systems involved in such epidemics will continue to be compromised. Additional effort on the part of fish disease specialists (epidemiologists) and fishery population dynamicists (demographers) and managers are justified, even required.

Juvenile Atlantic Menhaden (Brevoortia tyrannus) *versus the Ulcer Disease Syndrome (UDS)*

The active case which follows is so recent (continuing in all areas even now) that this report is based upon internal memoranda and personal communications between the agencies and specialists involved.

In October of 1984, D. E. Zwerner of the Virginia Institute of Marine Science received five lots of preserved young-of-the-year menhaden taken in the York River Virginia (lower Chesapeake Bay) and the Chester River on the Eastern Shore of Maryland (upper Chesapeake Bay). All individuals in those samples bore integumental lesions. Those from Virginia displayed ulcers, some shallow and some deep, on the ventral surfaces of their bodies, mostly around the anal vent (Figures 12 and 13). Fewer were higher up on the flanks. The Chester River samples exhibited eye lesions such as cataracts and exopthalmia (Zwerner, pers. comm.). These samples had been taken by castnet from the centers of swimming schools to reduce chances of capturing diseased and damaged stragglers confined by infirmities to the peripheries of those schools (Guthrie and Kroger 1974) during the regular prerecruit sampling program of the National Marine Fisheries Service menhaden group at Beaufort, North Carolina. The field sampler, Mr. Guthrie, requested assistance with identification of the causes of the ulcers.

Histologically-processed materials from the integumental lesions were examined and found to contain extensive necrosis of epidermal and dermal elements as well as of underlying muscles, peritoneum, and even internal organs, where the ulcers were

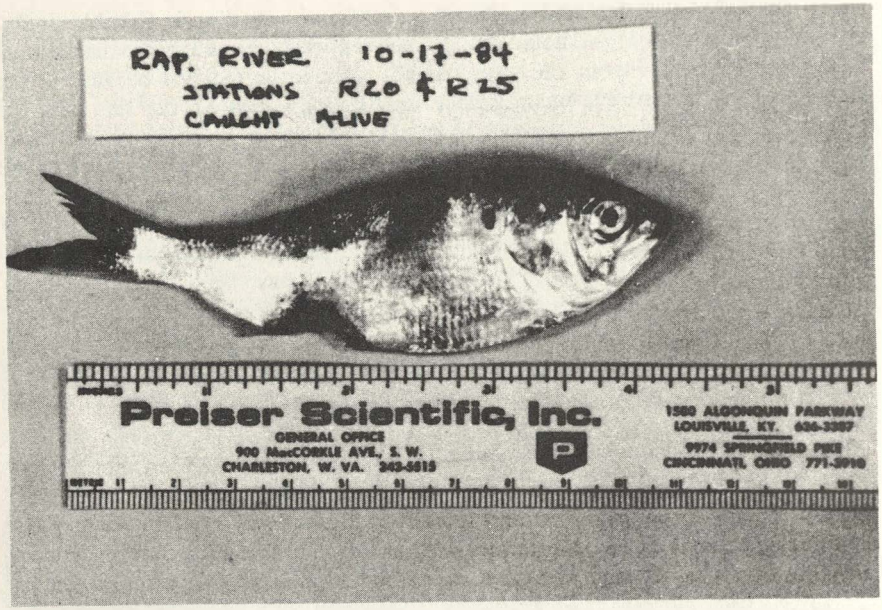


Figure 12. Juvenile menhaden *(B. tyrannus)* with U.D.S. from Rappahannock river, Va. Alive.

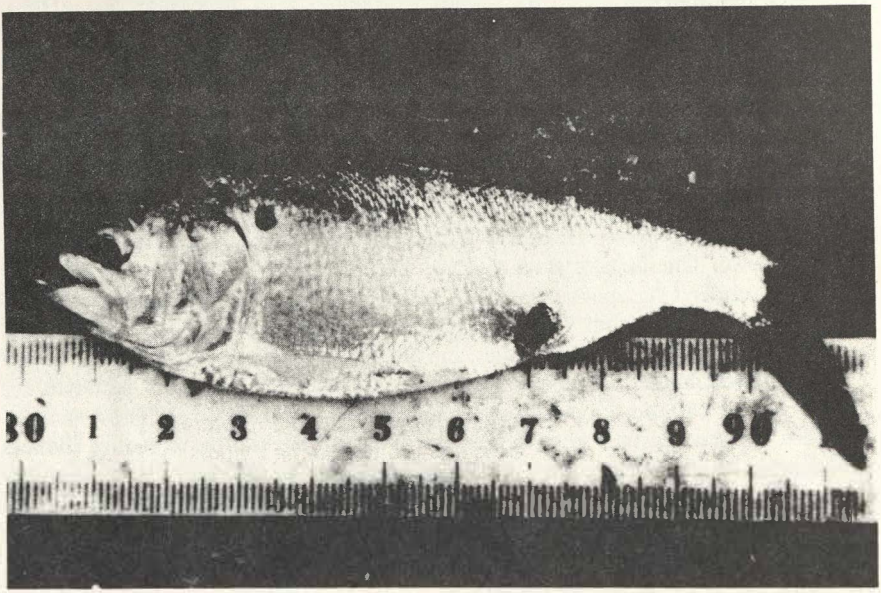


Figure 13. Juvenile menhaden *(B. tyrannus)* with U.D.S. from Rappahannock River, Va. Alive.

deep. Aseptate hyphae of fungus were found in the prepared specimens, along with bacteria. The granulomatous tissues and the hyphal structures involved resembled *Ichthyophonus* infections (Zwerner, pers. comm.).

Similar appearing skin leasions were seen by VIMS survey crews around the same time, and live samples of ulcerated individuals were taken during a specially-arranged sampling effort in November. Examinations of freshly prepared materials revealed the same hyphal-organism as seen in the earlier samples. Cultures contained both bacteria and the fungus. A few spot (*Leiostomus xanthurus*), butterfish (*Peprilus triacanthus*), mummichog (*Fundulus heteroclitus*), and hogchokers (*Trinectes maculatus*) taken during the trawl sampling efforts had similar lesions (Figure 14), but most affected were menhaden. For convenience the disease is being called Ulcer Disease Syndrome (UDS) temporarily. As in the earlier samples, the hyphae of the fungus were widespread in the ulcerous tissue and extended into underlying dermal, muscular, and peritoneal elements and into nearby organs.

(Incidentally, no eye lesions were seen in these Virginia-caught specimens. The ocular conditions found in the menhaden from Chester River, Maryland have not been investigated further as yet. Eyes from hastily prepared specimens are usually unsuitable for detailed studies and no additional fresh samples have been made available. Follow-up studies of this condition using espcially-preserved specimens are planned since cataracts are frequently seen in estuarine animals from heavily contaminated waters in the Chesapeake, such as the Elizabeth River, and we are studying them, especially for possible usage in bioassay work.)

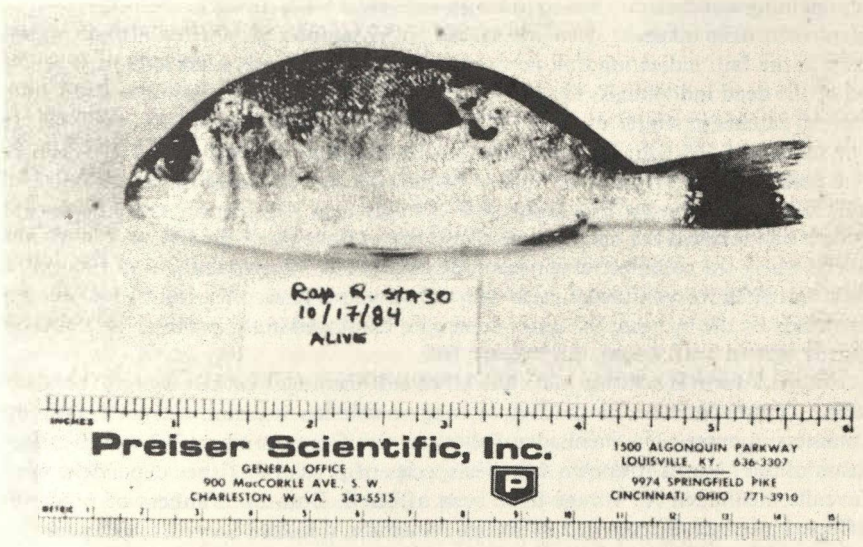


Figure 14. Spot *(Leiostomus xanthurus)* with U.D.S. taken from Rappahannock River, Va. Alive.

Contact with scientists in North Carolina revealed that ulcer-bearing menhaden had been captured two years before in 1982 in Pamlico and Albermarle Sounds and some of their tributaries. Other species such as southern flounder (*Paralichthys lethostigma*), weakfish (*Cynoscion regalis*), spot (*Leiostomus xanthurus*), silver perch (*Bairdiella chrysura*), striped bass (*Morone saxatilis*), white perch (*M. americana*), and pinfish (*Lagodon rhomboides*) were found with integumental lesions. As in Virginia waters, however, 0-class menhaden were the most commonly affected species. The same condition was seen by a North Carolina scientist in menhaden from the St. Johns River in north Florida. Recent newspaper accounts from Florida indicate that the condition is severe in some reaches of that river. Florida officials are known to be concerned (Joyce, pers. comm.). Dr. Burrell, director of the state marine laboratory at Fort Johnson near Charleston, confirms (pers. comm.) that diseased menhaden have been seen in South Carolina. Ulcer-bearing menhaden are reported in Georgia waters also (Musick, pers. comm.). Evidently, this Ulcer Disease Syndrome epidemic is widespread throughout the lower mid-Atlantic and South Atlantic.

Scientists at North Carolina State University also found fungi with broad, branching aseptate hyphae in the ulcers of diseased local fishes and isolated them in culture. Efforts continue to characterize the organisms, believed by most to be the primary pathogen at this time, and to satisfy Koch's postulates.

In some samples, UDS disease was found to affect over 80 percent of the juvenile menhaden captured. Diseased portions of southern flounder (*P. lethostigma*) catches, an important commercial and recreational fish, have had to be discarded at sea, dockside, or in the marketing and distribution systems in North Carolina, representing an economic loss to industry caused by UDS. Dead juvenile (prerecruit) menhaden were taken in scientific survey trawl samples in North Carolina waters later in the fall, indicating that predators and scavengers were not able to consume all of the dead individuals. Deaths must have been significant. Samples from commercial catches in winter of 1984-1985 were taken by NMFS Beaufort personnel. In one sample of 567 fish, 7 individuals (1.2 percent) and in another of 3,395 fish, 62 (1.6 percent) bore "crater-like" ulcers of the type seen in the estuaries earlier. This indicated that diseased fish from N.C. sounds and rivers from the Chesapeake system had reached the open waters of the ocean (outside of the barrier islands and inlets) where the commercial catches were taken. The low percentages of fish found infected offshore contrasted with the very much higher ones found inshore are explained by the fact that the purse-seine gear employed in the commercial fishery is biased against such young (and small) fish.

Not yet known is whether UDS has killed sufficient numbers of 0+individuals to affect future commercial catches. Young menhaden are not recruited into the commercial purse-seine menhaden fishery in significant numbers before reaching 2 years of age. Nor is it known whether species of predatory fishes dependent upon juvenile menhaden for forage have been affected. Data on numbers of predators affected by possible reduction of forage reduction, if indeed it occurs, will probably never be of sufficiently high resolution to show such changes. These two questions are the most significant practical ones involved in evaluating the significance of this disease. An assessment of these effects should be attempted. The possible long-term effects of the disease on the other involved species should also be considered in future research and surveys.

The disease evidently will continue into 1985 since field survey crews of North Carolina have reported the appearance of several diseased menhaden (and the gizzard shad, *Dorosoma cepedianum*) in samples in March of this year (Hawkins, pers. comm.)

Normally, fungi of the type these appear to resemble most are not primary but secondary invaders. Consequently many feel that the fishes become infected and eventually die because they have been stressed, possibly due in part to unfavorable environmental conditions. This is an interesting surmise since many of the estuarine waters in which it is found, for example, the Rappahannock and York Rivers of Virginia and the Sounds of North Carolina, are not ordinarily thought of as being contaminated. Large population centers and heavy industries are not located upon them and none are known to be experiencing significant environmental problems. Water quality problems may be involved, however, and the U.S. Fish and Wildlife Service has taken water samples in North Carolina for analysis to help investigate the possibility that environmental contamination might be involved. The results are not available to us as yet. The possibility also exists that bacteria are the primary invaders and the fungus secondary. There are several marine fungi, including *Ichthyophonus hoferi*, discussed above, which have been known to be primary pathogens of certain estuarine and marine species for decades.

The state and federal agencies involved, concerned over the welfare of the species affected, the upper level predators dependent upon them, and the commercial fisheries which are based upon them, plan continuing studies. Their purposes are: (1) to identify the organism(s) involved, (2) determine whether it (they) causes the ulcer disease, (3) to establish the mortality level resulting, and (4) to identify the possible effects upon the numbers and biomass of pre-recruits and recruits. Perhaps this work will be of value in determining the possible significance of this currently active disease in natural mortality.

Discussion

The cases described above provide support for concluding that disease is a significant factor in causing death, and determining survival, in several different marine populations. Though the invertebrate cases cited are few, involving only one mollusc and two crustaceans, many more examples are available.

Cases of diseases in molluscs are especially numerous. Viruses, chlamydians, bacteria, protozoans, and metazoans are found in a wide variety of molluscs (and crustaceans) of economic importance. Hargis (1985b) presented a partial list of identified diseases of bivalves in North American waters alone. Twenty-one different diseases, syndromes, or conditions involving *Crassostrea virginica, C. gigas,* several *Ostrea* spp., *Mercenaria mercenaria, Mya arenari, Argopecten irradians,* and other hosts based upon various reports (especially Sindermann 1977 and Farley 1981) were noted. The list undoubtedly can be increased.

For example, in one currently active case, that of the razor clam (*Siliqua patula*) on the coast of Washington State, the estimated population fell from 20 million clams in June of 1983 to 1.5 million in the winter of 1984-85. Public harvesting of this popular clam was stopped in 1983 and the ban has continued since. The closure, supposed to remain through the 1984-85 season, at least until July of 1985, may end sooner since the clam populations seem to have made a rapid recovery (Sparks, pers.

comm.). A likely etiological agent, found associated with lesions of the gills, has been termed NIX (Nucleus Inclusion X) since it occurs in the nuclei of cells in infected hosts.

Molluscs in other parts of the world are affected by pathogens also. Diseases with significant effects on host populations have been found in many coastal waters of France in *Ostrea edulis* (the European flat oyster) and *Crassostrea gigas*, (the Japanese oyster, imported ultimately from the far East) as well as in *Crassostrea angulata* (the Portugese oyster). A translation of the report by Henri Grizel (1983) by Sally V. Otto, pathologist of Maryland, discusses oyster diseases in France in some detail. Pathogens have also been found and, in some cases, implicated in deaths of oysters and other molluscs in Japan, Australia, New Zealand, Holland, and in the Mediterranean among other likely places.

Crustaceans of socioeconomic importance also are affected by virus, bacterial, protozoan, and metazoan diseases both in culture situations (Overstreet 1983, 1985) and in wild populations (Overstreet 1978, 1983). As with the molluscs, examples other than the case histories mentioned above are available. That the list of hosts includes mostly animals of economic importance such as the blue crab (*Callinectes sapidus*), the several commercial shrimps of the Gulfs of Mexico and Maine, and those of the northeast Pacific among others is no surprise. Research has been concentrated on them.

Lobsters, like blue crabs and other valuable decapods, including *Carcinus* and *Cancer*, have a number of diseases. Of the many known symbiote[1]/host relationships, that caused by the bacterium *Aerococcus viridans* (var.) *homari* is the most noteworthy. The disease, termed gaffkemia, occurring on both sides of the Atlantic is the most serious lobster malady known. This pathogen does its damage mostly in confined host populations, but the causative organism (Koch's postulate have been satisfied) occurs free-living in sea water. Not only do protozoan micro-organisms cause problems in lobsters but metazoans do also. For example, a nemertean worm of the Order Hoplonemertea has been implicated in predation or motality of the egg-masses of ovigerous females in confinement. It has also been found in wild lobsters. "The historical and potential impact of this nemertean on the reproductive potential of wild lobsters is not known" (Aiken et al. 1981). These and other diseases of *Homarus* species should be further followed to determine their role in feral populations as well as those in captivity.

Though many other examples of parasitism and disease in mollusc and crustacean invertebrates of socioeconomic importance could be cited (for examples, see Overstreet 1978, 1983, 1985, Sindermann 1977), considerable ignorance exists regarding their significance in mortality. Stewart (1984) has remarked that "Most parasitologists associated with lobsters believe strongly that this situation should be rectified." The same could be said about most important invertebrate diseases. Expressed more generally, there is a need to understand the role of disease in feral and cultured populations of all important marine organisms and its significance in the natural mortality factor (*M*) of fishery models.

As with the invertebrates, many other published examples of parasitism and disease among finfishes (teleosts and cartilaginous species alike) could be cited.

[1]The word symbiote, used here in the broad sense, includes and subsumes commensalism, mutualism, phoresis, and parasitism.

Bauer et al. (1981) discuss many of them, citing a number of examples and including an extensive list of references. Several of the papers in Hargis (1985a) also describe many cases of parasitism and pathogenicity in finfishes and include many citations. (See also Colwell and Grimes 1984, Dethlefsen 1984, Hetrick 1984).

Some recent work has dealt with metabolic responses of hosts to their parasitic invaders. For example, see van den Broek, (1978) who examined the effects of the copepod *Lernaeocera branchialis* upon the lipid biochemistry of the host whiting, *Merlangus merlangus*, (Linnaeus described both, early on). He reported that an average of 40.4 percent of the whitings were infected by copepod between 1973 and 1975 after the 0-class fish had moved inshore where they are "particularly vulnerable to parasitic infection." His experiments indicated that mature adults of *L. branchialis* cause significant reduction in weight of infected individuals. He found that the lipid content of their livers was more than 50 percent lower than those of uninfected fish. Phospholipids were lower also. These studies showed that whitings are adversely affected by the parasites, which cause physical damage to the gills as well as changing the host's metabolism. Other scientists, who reported that extent of injury to the host can vary from insignificant to complete emaciation, have studied the deleterious effects of parasites and pathogens upon their hosts more broadly. Kazachenko and Tatar (1985) concluded that parasitic copepods actually reduce the biomass and fishery yields of heavily infected stocks significantly. Kuperman (1973) discussed the importance of tapeworms in the ecology of their hosts. Collard (1970) considered parasites of mesopelagic fishes and their interactions and importance as has Campbell (1983) for deep-sea species.

Most parasitologists and other marine disease specialists are convinced that parasites and pathogens play a significant role in the ecology of fishery populations they affect. The case histories presented above are but a few which undergird their convictions. Though results of these and other studies of the effects of parasites and pathogens on their hosts often have not unequivocally established, the magnitude of possible reductions in biomass or numbers of individuals resulting, evidence that they are significant is sufficiently strong to warrant continuing and more definitive field and laboratory studies.

Summary

The extensive literatures of human medicine and epidemiology, agronomy, forestry, animal husbandry, and wildlife biology contain numerous historical and recent examples of the importance of disease in affecting health and survival of individuals and controlling population levels. Qualitative and quantitative evidence supports this contention. Numerous specific references establishing these statements are available, but citations should not be necessary. Disease and death are deemed important factors affecting health, welfare, and productivity of human populations. Whole systems of preventative and remedial human and veterinary medicine have been developed in most stable societies. Tremendous human and financial resources are involved.

Undoubtedly disease exerts significant pressures in fishery populations also. Fishery science, too, should attend disease closely. Scientists involved with scientific and economic aquaculture of shellfish and finfish are often made acutely aware that parasites and diseases can reduce the condition of their stocks and cause extensive mortalities if not controlled (Overstreet 1985, Sindermann 1977, 1985a,b). Despite

these findings, scientists and managers dealing with wild populations of marine shellfish and fish often ignore disease as an element in population estimation and in management procedures. But disease is not the only cause of natural mortality which is assummed or inferred in management of feral fishery resources; the factors of deaths or removals due to predation, environmental stress, and senescence are also.

Most population equations use sequential age-catch statistics to establish total mortality (Z). These same fishery statistics (preferably with effort data) are used to establish fishing mortality (F). Subtraction of F from Z ($Z-F=M$) provides natural mortality M. Two problems persist: (1) natural mortality (consisting of the natural extractive factors of obvious importance—predation, environmental stress, and death due to old age as well as disease) are assumed or inferred but not known, and (2) equally or more damaging, fishing mortality estimates are usually weak, either not including or assuming too many factors. Inaccuracy of catch data, lack of or weakness in effort statistics, and ignorance of fishing-related mortality, (i.e., gear damage, culling deaths and by catch deaths) reduce the accuracy and precision of fishery-related data available. In many instances long-term data on sportfishing catches are not available or incomplete. Therefore fishing mortality (F) estimates are somewhat weak. As a result, total mortality (Z) often is not accurately known. Obviously, natural mortality estimates, derived as they are by subtracting F from Z are often "soft" also.

The necessity of management has driven the development and application of the several equations and models employed in making population estimates, predictions, and allotments. The statistics generated must be used despite their limitations since they are the only "game in town" and the urgency of wise allocation, use, and distribution of marine fishery resources for human purposes is obvious. Marine fishery populations, long pressured in heavily-fished areas like the North Sea, the several fishing banks of the North Atlantic and the mid-Atlantic Bight as well as those of the northeast Pacific and elsewhere, have come under stress around the globe since high seas fishing was increased after World War II. Further, increasing fishing pressure on available stocks has been augmented by the deleterious effects of environmental degradation as shown above.

Though we must use the data bases and models (numerical and graphical) now available in making management decisions since they are the only scientific bases or estimates available, efforts should be vigorously pursued to improve them. There is no clear evidence that present management practices are succeeding in reversing population declines in most fishery resources now in demand. Whatever the cause or causes involved, most of the pressured stocks and fisheries operating upon them continue troubled. (Excellent examples supporting these statements are the sea scallop [*Placopecten magellanicus*] and the multispecies demersal finfisheries of New England and the upper Virginian Sea [mid-Atlantic Bight], both of which show signs of continuing decline.)

Several elements appear to be involved in the persistence of overharvesting in many fisheries. One is insufficient determination on the part of most governments or the societies they represent to take the steps necessary to acquire needed data and effectively regulate withdrawals. Other contributing factors identified above are the lack of detailed knowledge of the resources and the forces acting upon them and the persistent softness of stock estimates. Ignorance of the dynamics and amounts of

pressure caused by the several elements of natural mortality (M) plays a role. Overfishing is relative, depending not only upon the activities of the fishing industry but the "instantaneous" and long-term ability of the stocks being exploited to withstand those fishing withdrawals or pressures applied. All of the dynamic elements of natural mortality (M) are involved. Axiomatically, we would be better able to manage both stocks and fisheries (the socioeconomic activities based upon them) if all elements acting upon those stocks (and fisheries) were understood and incorporated in the equations and models upon which biological and economic decisions are based. Until we understand those factors in detail, management perforce must be conservative and buffers or reserves must be maintained, leaving untouched and unharvested stock that might have been otherwise used with no danger to the natural resource base. To manage with close tolerances requires accurate and precise information!

Science should never be satisfied with incomplete understanding or partial answers but should strive toward complete comprehension and for accuracy and precision in its information and predictions.

Aside from the continuing need for development of improved scientific and managerial understanding, are there other reasons for pursuing additional information on the quantitative significance of disease? After all, in-depth research on disease and essential monitoring of wild populations are costly of time, energy, and money and we cannot afford to waste effort and finances upon unimportant or unnecessary research.

Without the ability to assess the significance of parasites and pathogens (and other elements of disease) in the population cycles of important fishery resources, we cannot determine the risks and possible deleterious results, or the benefits in possible savings of energy, time, and money, of ignoring them. What we don't know could hurt us! If disease (by itself or interacting with metabolic deficiencies, natural catastrophes, or man-caused environmental stress) acts to severely and quickly reduce one or more cohorts of a fishery stock and fishing effort is not curtailed in time to relieve punishing pressure, long-term resource damage and severe economic dislocations could result.

Fishery populations are among the best known of Earth's living estuarine and marine systems. Much effort has been devoted to matching water quality to needs of those plants and animals. (Evidently not enough, however, since signs of increasing contamination of many—even most, estuarine and marine waters continue to mount!). As the ultimate bioassay systems, the living organisms against which we can measure adequacy of the environment must be fully and accurately understood and must be regularly measured (monitored) with accuracy and precision. More complete understanding of the factors involved in causing mortalities in marine populations will allow development of more adequate and effective environmental criteria and standards against which water quality management and enforcement activities can be planned, pursued, and evaluated. Fishery populations which are assured of future survival and abundance and are safe for use as human foods are guarantees of adequate water quality.

Finally, some aspects of disease-related mortality can almost certainly be managed (Sindermann 1977). Disease resistance can be produced by immunization, by deliberate encouragement of the development of resistant stocks in nature, or by genetic manipulation in the laboratory. Purposeful "cropping" and control of

planned or accidental transferrals of susceptible hosts or alien predators, parasites, and pathogens into an unprepared population are other options open to managers. To understand the possibilities of undertaking such management efforts and the necessity and justification for doing so we must know which controls are applicable and needed and where and when they could be applied, as well as the possible benefits and costs resulting.

Clearly, significant scientific, sociological, and economic reasons suggest continuation of present efforts to establish and understand the role of disease (and other elements of natural mutility) in wild, and captive, estuarine and marine populations. Equally clearly, evidence of its importance in the survival of fishery populations justifies expansion of basic and applied research and of scientifically- and managerially-oriented survey and monitoring efforts.

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The Role of Disease in the Management of Cultivated Marine Fish and Shellfish Populations

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Introduction

Wherever animals are crowded together under abnormal conditions, opportunity exists for the spread of infectious disease and for mortality of a significant part of the population. This principle, so dramatically demonstrated in Europe during the great plagues of the Dark Ages, forms a part of the management information base in marine aquaculture, and the reality of the principle has been affirmed repeatedly. In the past three decades, disease has emerged as one of the most significant technological problems in marine culture—to the point where, in some instances such as pen culture of Pacific salmon, the continued existence of the industry depended on solution of an existing disease problem (in this instance vibriosis).

Disease control is complicated by the continuing interplay of host susceptibility, pathogen virulence, and environmental influences. Frank pathogens and their effects can be dealt with reasonably well, but much of the damage to cultured populations is caused by facultative pathogens (bacteria and viruses especially) which exert effects when water quality is not maintained or when other stressors (abnormal temperature, oxygen deficiency, inadequate diet, overcrowding) exist in culture facilities. Disease is often the overt symptom of marginal culture conditions; control frequently consists of improving those conditions.

This brief review of highly selected aspects of the role of disease in management of cultivated marine fish and shellfish populations emphasizes only the following components of a very complex interaction of host, pathogen, and environment:

- disease control in extensive and intensive culture;
- variability in disease effects;
- disease control at different life history stages;
- long-term requirements for disease control;
- disease control by restricting transfers and introductions; and
- assessment of the significance of disease in marine aquaculture.

A principal message is that in the developing technology of marine aquaculture, the assurance of reasonable health of cultivated populations must be a major management objective.

Some Commonalities and Differences in Disease Control in Extensive Versus Intensive Culture

Marine aquaculture as it is practiced today can be artificially divided into *extensive aquaculture,* utilizing natural bodies of water, with only modest technological sophistication, with little environmental control, and with low growout densities,

and *intensive aquaculture,* in artificial bodies of water (raceways, tanks, constructed ponds), with higher levels of environmental control, higher technology applications, and high growout densities. Extensive culture would include oyster culture, as conducted in many countries, and ocean ranching of shrimp and salmon. Intensive culture would include shrimp culture in large circular tanks (as used in Japan) and cage culture of salmon or yellowtail.

Considering only the disease control component in managing such systems, some factors are common to both types, and some are unique to one or the other (Table 1). A healthy seed stock, adequate nutrition, prophylactic immunization of fish (particularly against vibriosis), good water quality, and early recognition and diagnosis of disease problems are control ingredients of universal application in any kind of system. Extensive culture methodology also includes environmental or stock manipulation (moving oysters to low salinities to reduce effects of particular pathogens, for example), and the necessity for quarantine and disease inspection of nonendemic animals being considered for introduction into natural waters. Intensive culture, on the other hand, can take advantage of chemoprophylaxis and chemotheraphy not feasible in extensive systems, and must be concerned continuously with stress reduction (in the form of maintenance of adequate water quality, nutrition, and population density in particular).

Figures 1 and 2 visualize the principal components of disease control programs in both types of culture systems.

Variability in Disease Effects on Cultivated Marine Populations

The phenomenon that we call "disease" in a cultivated fish or shellfish population represents a dynamic interactive complex of processes. These include, but are not limited to, whether the pathogen is primary or facultative; whether its infectivity and virulence are high or low; what the level of resistance of the host population is; whether recovered individuals act as carriers of infection; what reservoir hosts exist; and what environmental barriers to the spread of infection exist. Translated into effects on cultured populations, a particular disease may produce (1) a low (and usually tolerable) continuous background mortality; or (2) a moderate peak of mortality, after which the population is resistant; or (3) a sharp increase in mortality

Table1. Principal elements of a disease control program in marine aquaculture

Extensive culture	Intensive culture
• Healthy seed stock	• Healthy seed stock
• Adequate nutrition	• Adequate nutrition
• Prophylactic immunization (fish)	• Prophylactic immunization (fish)
• Good water quality	• good water quality
• Early recognition of a problem and diagnosis of its cause	• Early recognition of a problem and diagnosis of its cause
• Environmental/stock manipulation	
• Control of transfers and introductions	
	• Chemoprophylaxis and chemotherapy
	• Stress reduction

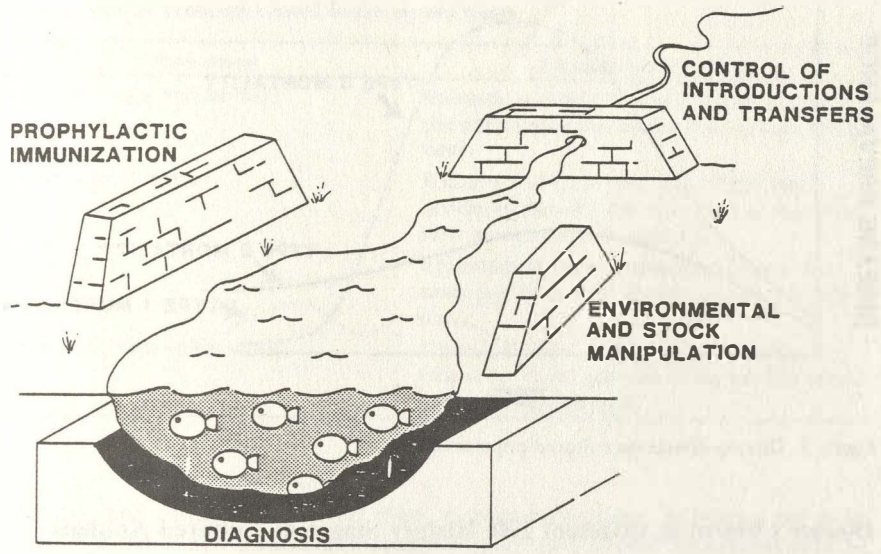

Figure 1. Disease control in extensive marine aquaculture.

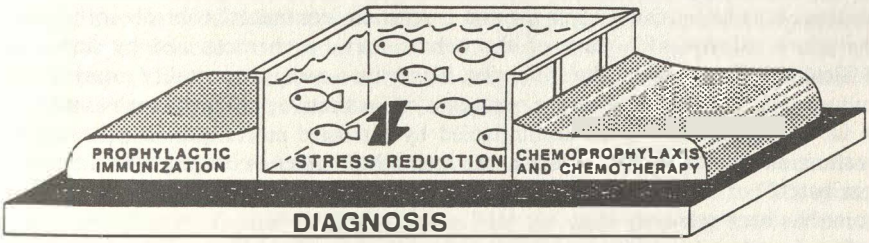

Figure 2. Disease control in intensive marine aquaculture (modified from Sindermann 1984)

(usually the loss of a significant part of the population) (Figure 3). Often, too, several concomitant diseases may produce low background mortalities; these may be masked by an epizootic caused by one of the pathogens, or by a different pathogen.

Faced with this array of possible effects, management strategies will vary with the extent of impact on the cultured population—usually assessed as the percentage of individuals that can be extracted by disease from the population daily and still make a profit. If the effect is small, the producer will usually decide to live with the disease; if the effect is moderate, the producer will invest modestly in disease control; if the effect is catastrophic, caused by an epizootic, panic results, but the damage has usually been done.

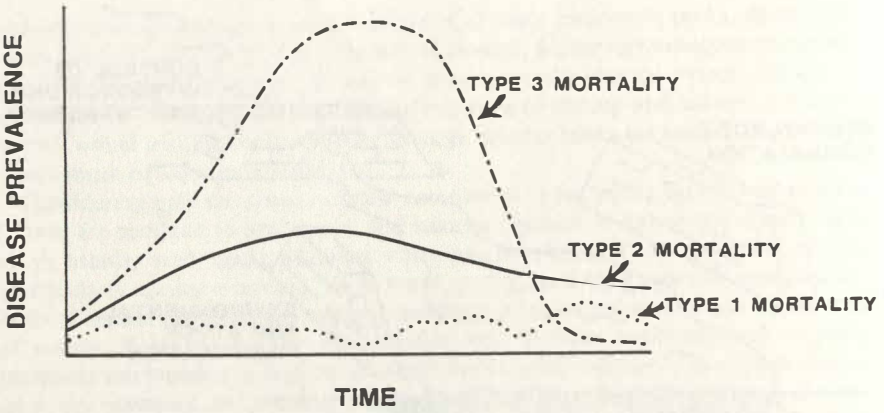

Figure 3. Disease effects on cultured populations

Disease Control at Different Life History Stages of Cultured Animals

An important but often overlooked truism in disease mangement of cultured animals is that the problems and the control strategies may be drastically different at each life history stage, from egg to adult (Table 2). This is in part because different pathogens may be involved, and in part because the extent of investment in and value of the animal increases with increasing age. Eggs and larvae of many marine species are usually available in quantity, but their *quality* must be maintained by investment in expensive equipment and careful environmental control—otherwise the entire subsequent history of the cohort may be characterized by lingering difficulties (slow growth, abnormalities, higher than average mortality rates). Poor culture conditions may encourage outbreaks of facultative pathogens such as vibrios in larval populations, often accompanied by extensive mortalities. Frequently, if production schedules can tolerate it, the method of choice in dealing with a "problem batch" of larvae is to discard it and start fresh, since the investment to that point has been relatively low.

Post-larval and juvenile populations may also be subject to epizootics and mass mortalities, but nutritional deficiency diseases and chronic diseases producing low but continuous mortalities emerge as additional problems. This combination of acute and chronic diseases persists right up to market size.

A new disease factor enters with management of brood stocks—the presence, often in latent form, of infectious diseases which may be transmitted vertically through eggs. This can be a particular problem with some viral diseases (such as that caused by Infectious Hematopoietic Necrosis Virus—IHNV—in salmon) (Pilcher and Fryer 1980), and also some of the bacterial diseases (such as bacterial kidney disease of salmon) (Fryer and Sanders 1981).

Long-Term Requirements for Disease Control in Marine Aquaculture

Principal technological requirements for marine aquaculture are adequate nutrition, maintenance of good water quality, efficient system engineering, genetic

Table 2. Disease problems related to life history stages.

Culture phases	Principal disease problems
Seed stock (eggs and larvae)	Epizootic microbial diseases of larvae. Facultative pathogens enhanced by poor culture conditions.
Growout (post-larvae)	Epizootics of frank pathogens. Nutritional deficiency diseases. Chronic diseases producing slow growth and low mortalities.
Market sizes	Epizootics of frank pathogens. Chronic diseases producing slow growth and low mortalities.
Brood stock (spawning adults)	Chronic diseases leading to low continuous mortality. Pathogens which may be transmitted vertically through eggs.

selection, and disease control. Of these, disease control can be among the most demanding, since new disease entities appear, and those already recognized must be controlled. Where culture has persisted for long enough—as, for example, in Japanese yellowtail culture—the pattern that has emerged is one of sequential appearance and spread of new diseases, often accompanied by significant mortalities until control methods are developed (Table 3). The new diseases may persist at epizootic levels for several years, and then subside to form part of background mortalities, especially if effective control methods are developed. Almost invariably, though, there are additional disease entities, some already enzootic in the population and some introduced from other areas, which may emerge as problems in marine aquaculture. The process appears to be unending, and calls for availability of unique expertise in pathology at times of crisis, to insure early diagnosis and development of control measures.

Sometimes,though, even when expertise is available, years of study may be needed to develop understanding of the pathogen, its method of transmission, its life cycle, and its environmental requirements. This has been the case with recent mortalities of oysters in France and United States, in which obscure but virulent protistan pathogens have been recognized as etiological agents (Grizel et al. 1974, Ford and Haskin 1981) Fortunately, even in the absence of adequate scientific information, empirical methods of stock and environmental manipulation can be and have been developed, which allow the industry to survive, even in the presence of epizootic disease. These methods—in the case of the oyster diseases mentioned—include delayed planting of seed until after the infectious period is past (France) or planting seed in low salinity areas where the pathogen will not multiply (United States).

Virus diseases of fish and shellfish present a particularly difficult problem. Since aquaculture provides opportunity for close observation of all life stages of selected aquatic animals, information about their virus diseases has increased enormously in the past decade. This is especially true of shrimp, crabs, oysters, salmon, and eels. It is likely that the virus diseases already recognized, and many others not yet recognized, exist in latent form in populations, and may be provoked into potency by stresses of the culture environment (abnormal temperature or oxygen levels, high

Table 3. Sequential appearance of microbial diseases of cultured yellowtail (after Egusa 1980).

Disease	1960	1965	1970	1975	1980	% of total losses (1978)
Vibriosis		1963 × × × × × × × × × × × × × × ×				5
Nocardiosis			1967 × × × × × × × × × × × × ×			<1
Ichthyophoniasis			1967 × × × × × × × × × × × × ×			<1
Pseudotuberculosis				1969 × × × × × × × × ×		28
Streptococcosis					1974 × × × × ×	63
Lymphocystis					1975 × × ×	<1

population densities). Control of virus diseases is difficult, particularly in marine populations. Immunization and chemotherapy are not effective, so it is particularly important that the etiological agents not be introduced into cultured populations or culture facilities from infected sources (for an example of the potential consequences of such actions, see Lightner et al. 1983). Control measures at present consist principally of viral eradication by destruction of infected stocks and sterilization of facilities.

Understanding diseases and limiting their effects are clearly long-term needs in marine aquaculture. The required technology does not appear quickly or inexpensively, but satisfying progress has been made in recent decades.

Disease Control by Restricting Transfers and Introductions

Aquaculture exploitation has been a principal motivation for the movement of fish and shellfish from one geographic area to another—even from one continent to another. One possible consequence of such transfers and introductions is the insertion of pathogens into new host populations in recipient areas.

Within the past decade there has been growing national and international concern about possible consequences of such movements. State natural resource managers, faced with the necessity to make decisions about shipments of fish and shellfish destined for waters under their control, have been sensitized to the possibility of spreading diseases, and have been forced into risk assessments. International organizations, such as the International Council for the Exploration of the Sea (ICES), have developed codes of standard practices, by which national authorities can make decisions about international shipments on some rational scientific basis, rather than on a purely economic one.

At present, treatments for many marine fish and shellfish diseases are unavailable. This is particularly the case for viral and protozoan diseases. Thus limiting the spread of pathogens through aquaculture practices is therefore of particular importance. Quarantine and inspection procedures, and politically insensitive policies and regulations about imports of live animals, can be major deterrents to dissemination of pathogens.

The history and consequences of movements of salmonids worldwide provide an excellent case history of disease dissemination with infected exports. The history includes negative examples of failure of barriers or absence of barriers, as well as positive examples of exclusion of pathogens by vigilance and enforcement of reasonable regulations. Failures include the spread of whirling disease of salmonids

(caused by the protozoan *Myxosoma cerebralis)* from Europe to United States, and the introduction of Infectious Hematopoietic Necrosis Virus (IHNV) into Japan with salmon eggs from United States. Successes include the prevention of introduction of chum salmon virus to United States from Japan, and the exclusion (to the present time at least) of IHN virus from United States east coast salmon stocks.

Exclusionary practices should not, however, be too inflexible. Transfers and introductions for aquaculture purposes should be feasible, once adequate study has been made and risks assessed. Use of standard inspection protocols, possible quarantine provisions, and limited initial quantities of imports all provide a measure of assurance of reduced danger from the spread of disease. Figure 4 illustrates proposed steps in introducing a new species, following the ICES Code of Practice.

Assessment of the Significance of Disease in Marine Aquaculture

Accumulated evidence from oyster, shrimp, and salmon aquaculture demonstrates that disease-caused mortalities, and the necessity for disease control measures, are significant factors in evaluating profitability of any venture (Sindermann 1977). Some data exist, particularly for fresh-water species, about the costs of disease control. In United States federal salmonid fish hatcheries, for example, disease control costs have been estimated at 10-15 percent of total production costs, while several state hatcheries have estimated such costs at 20-30 percent. Mortalities due to disease, and the necessity for disease control measures, have been estimated at 25 percent of commercial production costs for rainbow trout, 10-25 percent for channel catfish, and 20-30 percent for shrimp.

Losses due to disease have been estimated in Japan (Kawatsu et al. 1976) for both freshwater and marine fish culture. Losses due to disease in 1973 in freshwater culture were estimated at about 5,800 tons (total harvest was 64,000 tons) and in marine culture at about 3,500 tons (total harvest in that year was 84,000 tons). These estimates are probably very conservative.

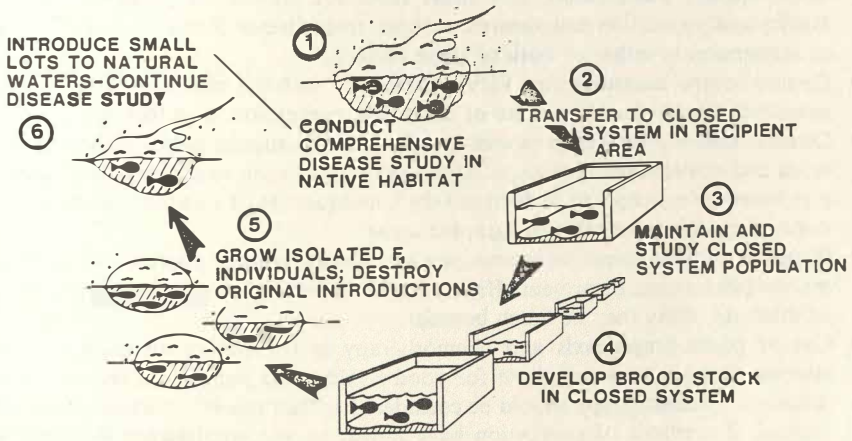

Figure 4. Proposed steps to reduce dangers of disease spread through introduction of non-indigenous species (from Sindermann 1984).

In addition to economic losses due to mortality, there are many control techniques which increase production costs:

- Disinfection of water by ultraviolet radiation, ozonization, or chlorination;
- Disinfection of holding facilities and equipment;
- Chemotherapeutic agents—used successively or alternatively to reduce likelihood of drug resistance—
- Vaccines; and
- Inspection and certification of eggs transferred from one geographic area to another.

A remarkably complete documentation of the effects of a single disease on oyster production was published recently by Haskin and Ford (1983). Their long-term study demonstrated that a disease of American oysters caused by the protozoan *Haplosporidium nelsoni* reduced production in one major area (Delaware Bay) by two-thirds, beginning in 1958, and has been responsible for suppression of the industry there since that time. High infection rates persist although recent mortalities have been only about half those recorded during the earlier epizootic peak. Stock manipulation—a shorter planting cycle in high salinity areas—and the development of disease resistance in surviving populations have allowed the industry to continue.

Conclusions: Some Implications of Disease in Management of Aquaculture Populations

Although it may seem a little unfair to other disciplines to isolate and emphasize disease control in aquaculture as a major technological objective, the reality is that the health of cultured animals must be a primary management concern. Once this mental hurdle is cleared and accepted as an operating principle, it becomes easy to construct a list of guidelines for disease management:

- Maintenance of animal health is a continuing struggle to control known agents and to diagnose and control new pathogens as they appear.
- Water quality maintenance and stress reduction should be principal foci of facility and population management actions, since disease is often a consequence of inattention to either or both of these factors.
- Disease control measures may vary significantly with life history stages, but are usually based on the triumvirate of diagnosis, prevention, and treatment.
- Disease control measures in extensive culture can be augmented by techniques of stock and environmental manipulation, and by attention to exclusionary principles based on attempts to understand the consequences of transfers or introductions of species from other geographic areas.
- Prophylactic immunization is emerging as a disease control method of choice in marine fish culture. Its present efficacy is with bacterial diseases such as vibriosis; its eventual utility may be much broader.
- Use of chemoprophylaxis and chemotherapy is feasible in intensive culture systems, but not as a substitute for good facility and population management practices. Chemotherapy should be considered a "last resort" method in disease control, if methods of prevention have failed, as was emphasized by Herman (1970) and Snieszko (1974).

This listing of management principles or operational guidelines could get extensive. Disease problems are important in aquaculture production, and effective control is a requirement for economic viability.

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Role of Diseases in Marine Fisheries Managment

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It has been estimated that living marine resources contribute approximately 15 billion dollars annually to the economy through commercial and recreational fishing activities, imports, exports, and joint ventures. It serves as an international bargaining tool, employs over 300,000 people, provides recreational benefits for over 35 million people, supplies 16 pounds (7.2kg) of nutritious food annually per capita, and provides a sole way of life for many individuals and small coastal communities (National Marine Fisheries Service 1985).

The extractive uses of the living marine resources involve a broad range of activities and interests that generally fall under two needs—supplying food and/or providing recreation, including aesthetic benefits. Those who use the living marine resource normally have "expectations" that fall into two seemingly different categories: (1) the continued use of the living marine resource within the constraints of ecological balance, and (2) the actual realization of benefits, such as profits and nutrition, from resource use. An overall desire of individuals and groups within the private sector is to use living marine resources for a profit, while the public expects that the resource is renewable and should be wisely used for the common good.

We in the National Marine Fisheries Service understand these needs, and, in fulfilling our agency mission of conservation and wise use of living marine resources, often find ourselves balancing the expectations of public trust and private interests in those resources. These public and private interests may appear antagonistic, but in fact they are complimentary. For example, public sector expectations are that the marine environment and living marine resources should not be subjected to excessive exploitation or other action that may jeopardize its continued use and that appropriate conservation be conducted to assure the resource's viability while allowing optimal extractive use for the common good. These public concerns are really no different from the private interest expectation of a constant fishery supply and a stable economic climate for continuing raw material acquisition. Obviously, both the public and private interests want access to a resource that is safe for consumption. While motivations may differ, the public/private expectations from living marine resources are decidedly similar and, as such, provide the basis for management of these resources, as has been demonstrated recently through the creation and efforts of the regional Fishery Management Councils by passage of the Fishery Conservation Zone Management Act. Though these regional councils have developed fishery management plans which focus more toward identifying biological considerations for optimum sustainable yield, they do have the legal authority to address product safety issues.

By way of definition, public health protection deals with only one aspect of consumer safety and, for purposes of this discussion, will be limited to protection provided for animal proteins including fishery products intended for human consumption. Both federal and state organizations provide consumer protection in the consumption of animal and marine proteins, although the philosophical approach

to each may differ. For example, the public health organizational and programmatic aspects of land-based animal food products are extensive with well-defined reporting systems and are fairly well coordinated at the national level by the U.S. Department of Agriculture. This program starts at the farm level with veterinarians who are involved with state livestock sanitary boards and animal health agencies located within each state. As the animals move from production centers to slaughterhouses and food plants, the animal health surveillance system is integrated into a formalized State/Federal Mandatory Inspection Program to ensure reasonable consumer protection by prohibiting suspect or violative products to be introduced into commerce. Complimenting this consumer protection activity are numerous technical and scientific support facilities with well-equipped and staffed diagnostic laboratories capable of examining animal disease in depth, as well as public health laboratories at the state level which process samples and reports from sanitarians at the county and municipal level. In effect then, land-based animal resources are managed for animal health and human health purposes through a formal broad scale program which integrates animal and public health disciplines (Fields 1977). This type of program evolved by a national policy recognition that (1) land-based animal production is food oriented, and (2) in the case of warm blooded animals, there are certain etiological disease agents that are directly transmitted to humans, necessitating elaborate animal health and subsequent product inspection systems. These logical combinations of animal and human health integrations have been well recognized by congress and have received modern legislative support, most recently through the passage of the Wholesome Meat and Poultry Acts of 1967 and 1968.

Such is not the case with fish and fishery products. In 1938, congress passed the Food and Drug Act, the original and sole legislative act for mandatory surveillance of fishery products. Prior to 1940, both meat inspection activities and food and drug activities were under USDA jurisdiction. During that year all foods with less than 2 percent meat or poultry flesh were transferred to what is now known as the Food and Drug Administration, and meat and poultry activities remained in USDA to be subject to subsequent integrated growth. In terms of legislation relating strictly to mandatory surveillance of fishery products, the most recent is the Food and Drug Act of 1938 as amended.

That legislation was augmented by the creation of a voluntary fishery products inspection program with the passage of the Agricultural Marketing Act of 1946. That act was focused primarily toward developing voluntary U.S. grade standards and inplant product inspections as a marketing tool to allow processors to use U.S. inspection and grading marks on fishery packages as one way to promote their products as safe, wholesome, and of high quality. In addition to being a voluntary program, that activity was also a fee for service program; that is, the participating processors had to pay for the federal inspection and grading services, a funding concept which is currently receiving renewed political interest. The program was transferred to the Bureau of Commercial Fisheries with the passage of the Fish and Wildlife Act of 1956, and subsequently to NOAA in the U.S. Department of Commerce with Executive Reorganization Plan Number Four of 1970.

Another fisheries surveillance program began in 1926 and was augmented with the passage of the Public Health Services Act of 1946. This was an industry/state/federal program specifically aimed at reducing the prevalance of bacterial disease, particularly Salmonellosis, from the consumption of contaminated raw molluscan

shellfish. In terms of consumer protection relative to fisheries, this was the first program that recognized the role of fishery management as a component in preventing potentially hazardous products from reaching consumers by implementing fishery management prohibitions of harvesting shellfish from suspected contaminated waters. In the late 1950s and early 1960s, this program gained wide acceptance by the participating coastal states, FDA, and industry. At one point it had over 1,000 personnel utilizing 50 labs, 500 boats, and 8 planes participating in some aspect of growing water classifications, shoreline sanitary surverys, patrol of harvest areas, plant inspections, and product evaluations. This former cooperative program was known as the National Shellfish Sanitation Program (NSSP). Unfortunately, this program collapsed due to several weaknesses too detailed to fully discuss here. Increased urbanization of the coastal zone, failure to incorporate changes in contemporary scientific understandings on the role of microbiological standards and acceptance sampling plans, and lack of both uniform enforcement procedures and strict penalties for program violators are among the paramount reasons for the NSSP's demise. That program is currently being revised and modified by the creation of the Interstate Shellfish Sanitation Conference (ISSC) whose purpose, among other things, is to foster and improve shellfish sanitation. This is a new organization, modeled after the Interstate Milk Shippers Conference, that will take some time to become totally effective. During all of this time, still another federal agency was examining and inspecting fishery products—the Department of Defense (DOD). The DOD is one of the largest institutional purchasers of foods in this country, requiring in-plant inspections for fishery products similar to USDA inspections for red meat. In 1977, the Department of Commerce Inspection Program assumed the DOD inspection responsibilities for fishery products with USDA assuming the DOD inspection role for animal proteins.

Therefore, for land-based animal products there is a well established, logical approach to providing consumer protection, which links animal health and human health considerations into what amounts to a resource management scheme under the jurisdiction of a single federal agency cooperating with the states to provide reasonable public health protection in the consumption of redmeat and poultry products. For fresh water and marine animals, such is simply not the case. Rather, retrospection reveals a haphazard evolution of human health philosophies and approaches that give the appearance of a disjointed kaleidoscopic focus by a multiplicity of federal and state agencies developing different standards and compliance schemes based upon antiquated legislative mandates or authorities. Surely it's time for a change, time for a new look at the total problem using contemporary understandings on the interwoven role that animal health and human health considerations play in consumer protection. A complete plan is needed which both provides for public consumption of safe and wholesome products and results in better and more efficient livestock production techniques. One need only to look at the success of USDA in organizing and implementing cooperative programs in resource management and consumer protection to see what can be done given the industry perceived need and legislative mandate.

Lacking that philosophical recognition, about all that any of us in fishery science can do is to continue to use our good science to provide adequate answers to the information needs of our constituents, be they resource managers, public health authorities or seafood industry members attempting either to establish marketing

strategies in a developing fishery or maintaining markets in an established fishery.

The question of a product's safety for human consumption is a primary determinant to its marketability. The lack of safety in a product is the result of any or all of four hazards: (1) environmental natural hazards, such as the presence of ciguatoxin in some reef fishes, (2) environmental manmade hazards, such as pesticides, heavy metals, and PCBs, (3) process-induced hazards, where the processing technology used is either inadequate itself, or is inadequately applied, such as the outgrowth of botulinum in canned fish, and (4) marketing/distribution induced hazards, such as improper handling practices, particulary inadequate storage temperatures, which can quickly render a product unsafe. Situations where products on the marketplace have been found to be unsafe are usually of disastrous consequence to the segment of the industry involved and frequently to the industry as a whole—witness the mercury scare of several years ago. Generally, the seafood industry does not have the expertise nor the resources to provide the research information or analytical capability necessary to avert or recoup from such situations, and necessarily relies on government to provide such. It is also important to recognize that the common property nature of the resource itself is a principal limiting factor on industry ability or desire to invest in such research.

The need for a new look in consumer protection in fishery products may be illustrated by discussing some recent foodborne outbreaks of disease. During the four year period of 1978-1981, there was a total of 2,114 foodborne outbreaks of disease reported to the Center for Disease Control. Of this, the vast majority (61 percent) was attributed to unknown vehicles, 8 percent to red meat, 4 percent to poultry, and 9 percent to fishery products (Center for Disease Control 1981-1983). The significance of these figures assumes added importance when we compare them to the 1983 per capita consumption rates for such products, i.e., red meat 179.2 lbs. (81.3kg), poultry 65.5 lbs. (29.7kg.), and fishery products 16 lbs. (7.2 kg) (National Marine Fisheries Service 1984).

In addition, analysis of the etiological agents associated with foodborne outbreaks of disease attributable to fishery products during the same 1978-81 time frame shows that of the 192 reported outbreaks, 73 percent are due to finfish, with 93 percent of those due to chemical causes (scombrotoxin or ciguatoxin) and 27 percent attributed to shellfish (most of it molluscan), with the vast majority being of bacterial or viral etiology. Simply, progressive fishery management techniques, which examine the cause of these diseases and prohibit the harvest of these affected resources where necessary can go a long way toward resolving public health issues dealing with fishery products.

Finally, the following is an attempt to end on a positive note. Since the mid-1960s, there have been numerous legislative proposals calling for the mandatory federal inspection of fish and fishery products, based upon the concepts employed by USDA for animal proteins. These proposals have failed primarily for two reasons: (1) they did not have industry support, and (2) their cost appeared prohibitive. The industry resistance is changing, and under our current administration the program cost may be switched to the users, with user acceptance.

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Disease Organisms, Economics and the Management of Fisheries

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Introduction

Disease organisms can dramatically alter economic conditions in food producing industries. Recent examples include dramatic fluctuations in egg prices caused by an outbreak of avian flu in Maryland's laying-hen flocks and in citrus prices due to a citrus canker outbreak in Florida. The seafood industry is no exception. One need only observe the recent situation in Maryland oyster production. Oyster spat fall in 1980 was nearly the highest on record, leading to expectations of a boom 1983-84 harvest. Emphasis in research was reoriented from harvest augmentation to marketing and promotion. A protozoan, MSX (*Haplosporidium nelsoni*), infected the oyster stock and destroyed those expectations. Instead of a boom, the 1983-84 Maryland oyster harvest fell below one million bushels, one of the lowest on record.

Increased mortality, as was experienced by the oyster resource, has the clearest impact on the fishing industry, but the subtlety of other impacts should not mask them from our view. Sindermann (1977) offers a pathologist's view of the range of potential economic influences:

> Economic effects of disease in marine fishes may be categorized as reduction in numbers of food fish available to the fishery; weight loss by diseased individuals; rejection of abnormal fish by consumers, and subsequent loss of interest in fishery products as food; (p. 315).

While the Sindermann categorization is quite useful, greater examination of producer and consumer behavior in the presence of disease organisms may prove beneficial.

In this paper, we will explore some of the ways that disease organisms can affect the economic welfare of the fishing industry and consumers of fish. For this study, disease organisms are defined very broadly to include parasites for which fish may act only as a passive host and microorganisms which alter the quality of fish. The first section is a theoretical discussion of how disease affects the supply-side of the market in a deterministic and then a stochastic framework. Examples of lost production and revenues are presented. We emphasize that lost revenues are not particularly useful measures of monetary losses to the industry. This is followed by an explanation of how economists derive monetary estimates of disease-related losses to the producers and consumers of fishery products. The MSX outbreak in the Virginia oyster fishery serves to illustrate our point. The next section deals with the effects of disease organisms on consumer demand. We follow with a discussion of how the understanding of waterborne organisms can improve the welfare of seafood

producers and consumers. The final section contains a discussion of the implications of disease organisms for fisheries management.

Effects of Disease on Fish Supply

Fishermen are faced with a number of short and long-term decisions, which when totaled, result in an industry level of effort and harvest. These decisions include how much labor to apply within a season, and how much capital (e.g., vessel, gear, etc.) to invest or disinvest. There are also individuals who are determining whether to switch from another fishery or occupation, into the fishery of interest. The ultimate decisions depend on economic conditions, which themselves are affected by the population dynamics of the fishery in question. In this section, we explore how disease affects the population dynamics of a fishery and in turn, how this affects fishermen's behavior.

Disease is a direct and major cause of fish mortality. In the 1950s, MSX on planted oyster grounds resulted in 90-95 percent cumulative mortality in Delaware Bay (Ford and Haskin 1982). High oyster mortality from MSX was observed in the early 1960s in the Chesapeake Bay (Haven et al. 1978). *Ichthyophonus*, a disease common to finfish led to a cumulative mortality of 55 percent of plaice in Scottish waters (McVicar 1981), and 50 percent of Gulf of St. Lawrence herring (Tibbo and Graham 1963). Clearly, the presence of disease reduces fishery stocks and hence, production. Table 1 lists some estimated mortalities associated with fish diseases.

Disease organisms can also decrease fish stock size and growth from causes other than direct mortality. Whiting infected with copepod parasites, *Clavella uncinata* and *Lernaeocera branchialis,* experienced significant weight reduction when compared with non-parasitized fish (Van den Broek 1978). *Ichthyophonus* will also lead to emaciation (McVicar 1982). The resulting decreased muscle efficiency makes these fish more susceptible to predation mortality. This was demonstrated indirectly in a 1955-1956 *Ichthyophonus* outbreak in herring that was accompanied by an increase in cod growth, presumably due to the availability of infected herring as prey (Tibbo and Graham 1963). Haddock infected with a coccidian parasite (*Eimeria gadi*) affecting the swim bladder were more suceptible to predation mortality (Odense and Logan 1976). The dysfunction of the swim bladder also prevented the haddock from spawning.

Vaughn et al. (1984) demonstrate how to incorporate disease-related stress into deterministic fishery models. The models range from the simple aggregate surplus production function attributed to Schaefer (1957), to complex bioenergetic models. In the surplus production model, the intrinsic population growth rate parameter can serve to aggregate both the direct mortality and indirect effects of disease on all age classes of the population dynamic processes (i.e., growth, reproduction, etc.).

We will use this simple model to show the intertemporal effects of disease on the fishery. In addition to the biological model, we introduce a fishery investment sector following Smith (1968). The investment sector is characterized by open access. The bioeconomic model then includes a capital stock based on profits and a resource stock based on initial stock and net growth (growth-harvest). A steady-state equilibrium occurs where the level of fishing effort is such that there is no net population growth of fish and no net capital investment in the fishery. No net population growth requires that the harvest level be equal to the natural rate of growth of the

Table 1. Summary of prevalence and mortalities of selected fish diseases.

Disease	Species affected	Location	Prevalence[a]	Mortality Rate[a]	Years	Source
MSX	Oyster	Chesapeake Bay (VA)	0-80%	>50%	1964-1966	Andrews (1968)
		Chesapeake Bay (MD)	4-70%	2-55%	1961-1968	Farley (1975)
		Delaware Bay		37%	1958-1982	Haskin and Ford (1973)
Dermocystidium	Oyster	Chesapeake Bay (VA)	70-90%	17-22%	1952	Haven et al. (1978)
SSO (*Minchinia costalis)*	Oyster	VA Eastern Shore	NA[b]	12-44%	1959-1960	Andrews (1968)
Marteilia refringens & *Bonamia ostreae*	*Ostrea edulis* (Flat oysters)	Brittany	NA	70-90% 80-90%	1970-1976 1980-1982	Grizel (1983)
Ichthyophonus	Plaice	Scotland	2-12%;85%	NA	1976-1980	McVicar (1980)
	Haddock	Scotland	<25%	NA	1976-1980	
	Herring	Gulf of St. Lawrence	NA	50%	1954-1956	Tibbo & Graham (1963)
Blackmat syndrome	Tanner crab	Gulf of Alaska	0-60% (in males)	NA	1981-1982	Hicks (1982)
Eimeria gadi	Haddock	Nova Scotia	32%	NA	1973-1974	Odense and Logan (1976)
Lernaeocera branchiahs	Whiting	Medway estuary	40.4%	NA	1973-1975	Van den Broek (1978)
Vibriosis	Saithe	Norway	NA	NA	NA	Munro et al. (1983)

[a]See source for selected area sampled.
[b]NA = not available.

fish stock. No net investment in the fishery requires that profits resulting from additional investment in the fishery are zero. The bioeconomic equilibrium is denoted as point A in the phase diagram of Figure 1. In this diagram, X is stock size and K is the amount of capital in the industry. The parabola, $F(X,K) = O$, is the combination of X and K at which there is no net growth in the fish population. The function, $I(X,K) = O$, is the combination of X and K where there is no net investment in the fishery. The intersection of these curves at point A is the stable equilibrium point where both these conditions are satisfied. The point of intersection will vary depending on fish prices and fishing costs (Clark 1976).

Our diagram, although not unrealistic, represents a stylized analysis which must be adapted to the particulars of the fishery. Biological quirks, cultural inertia, and institutional anomalies are among the realities which must be recognized when examining a specific disease outbreak. Thus, most disease outbreaks will reduce the biological capacity of the fishery. The degree to which the disease influences the fishing sector depends, in part, on these other factors. With the simple model in hand, we move on to discuss typical variation of it.

Enzootics

Consider first an introduction of disease into the fishery which alters the biological growth characteristics in a constant intertemporal manner. The "long-run" biological effect is seen in Figure 1 as a shift in the biological equilibria from $F_o(X, K)$ to $F_1(X, K)$. The immediate effect of the disease is to reduce fish stock, causing profits to fall and reducing capital. The movement from equilibrium A to A' is

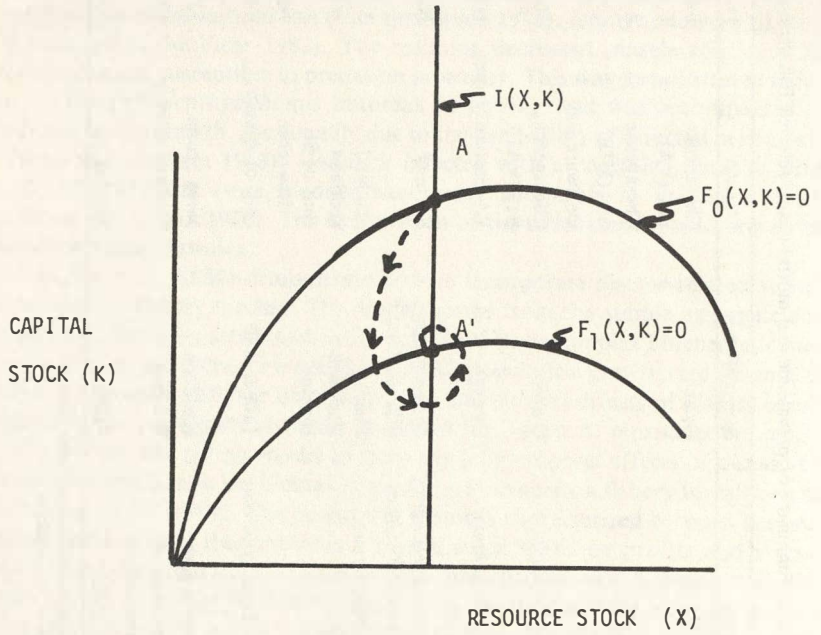

Figure 1. Capital and fish stock movements following permanent introduction of disease.

shown as the dashed line with arrows. The harvest (shown in Figure 2) drops from A to A' after cycling. The periodicity of the cycle is a function of reaction speed of the fish and investors. The quicker the reaction, the tighter is the dashed path and the shorter the period of the cycle.

The above analysis assumed that capital is perfectly malleable so that disinvestment can occur as easily as investment. Bockstael and Opaluch (1983) demonstrated that there may be non-monetary (e.g., cultural) costs in disinvesting from one fishery and investing in another, or out of fishing altogether. Thus, when the disease first occurs to reduce the population to point A', some fishermen may decide to "stick it out," and not disinvest or switch fisheries. If this occurs, the harvest rate may continue to exceed the population growth rate, so that the population declines even further. At the lower population size, profits will continue to decline, eventually below the threshold that was necessary to keep the fishermen in the fishery. Disinvestment finally occurs but, because the resource is so depleted, the time to reach a new equilibrium is lengthened.

Epizootics

Seldom is the disease introduction as simple as depicted above. Immunity, natural selection, and varying environmental conditions can cause diseases to occur during narrow windows of time and sporadically. The MSX and *Ichthyophonus* outbreaks discussed earlier represent such behavior. Sindermann (1956) found that since 1900, North Atlantic herring stocks had experienced six major 1-3 year epidemics of *Ichthyophonus*. The prevalence of the disease was 25 percent during the epidemics and 1 percent in intervening years. MSX in Delaware Bay appears to follow 6-8 year cycles of prevalence, but not in Chesapeake Bay (Ford and Haskin 1982).

Sporadic epidemics can be represented as a series of movements of the biological growth function. An outbreak is seen to shift the function downward (like $F_1(X,K)$ in Figure 1) for a period of time. Once the disease population has run its course, the growth function returns to its original potential, say $F_0(X, K)$. The time path of harvests now depends not only on the "natural" biology of the host and economic

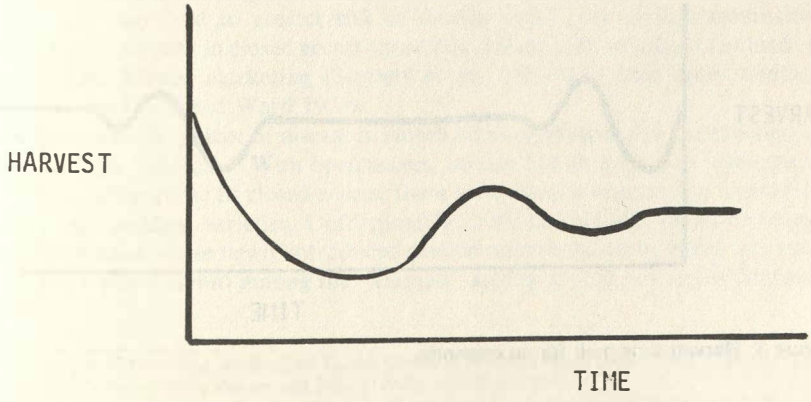

Figure 2. Time path of harvest following disease introduction.

response of the industry but also on the population dynamics of the disease organism. We offer a possible time path of harvest in Figure 3. The illustrated time path shows harvest rising above the initial harvest level. This can occur when the resource population is building rapidly and the investment has not yet responded to the favorable condition. Investment eventually catches up and returns stocks and harvests to the original levels.

Disease and Uncertainty

Thus far, the discussion has been couched in a world in which the investors make myopic decisions based on the level of profits received at one point in time. Clearly, this is a simplification (see Berck and Perloff [1983] for a more complex model). Most investment analysis suggests that investors not only consider the current level of profit but also the amount of fluctuation in profits over time. Investors are considered to be risk-averse, preferring low variation in profit to high variation.

In Figures 2 and 3, we see that the disease has introduced not only lower catches but also greater fluctuation in catches. Implicit in these harvest fluctuations are fluctuations in profit. Thus, the introduction of disease, at least in our model, introduces greater fluctuations in the economic environment.

Although it is difficult to obtain good cost/profit information, we believe disease organisms also introduce uncertainty in existing fisheries. A disease such as vibriosis, for example, may affect fish so rapidly that the disease is not detected, although significant mortalities have occurred (Munro et al. 1983). The sudden appearance of an unidentified fungal disease at high prevalence rates in some North Carolina and Virginia-caught menhaden is an excellent current example of disease-related uncertainty.[1] Lesions indicative of the disease have been found mainly on young-of-the year menhaden. The industry is unsure how this disease will affect recruitment to the offshore fishery one or two years hence. The industry would be in a better position to plan their investments if they had projections of recruitment. These examples demonstrate that, at least in some species, disease may be a significant contributor to the variability of the stock size, and the uncertainty of fishermen and investors.

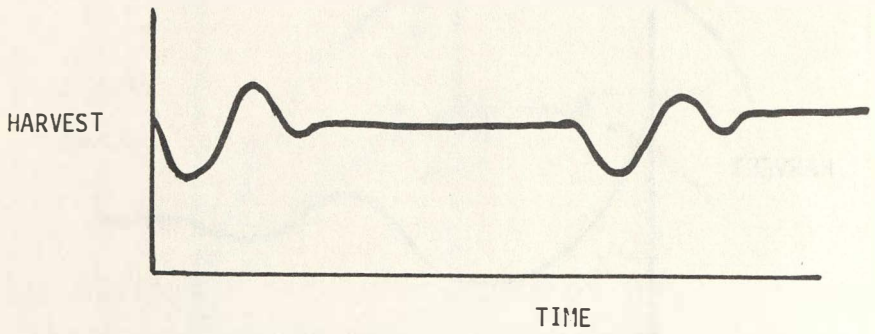

Figure 3. Harvest time path for an epizootic.

[1]Personal communication, Dr. J. V. Merriner, NMFS, Beaufort Lab., N.C.

The studies of fishing under uncertainty are extensions of the deterministic model presented earlier.[2] This model is modified to incorporate a stochastic element in stock growth over time. Assuming that fishermen and investors in fishing are risk-averse, they would be willing to invest more in a fishery where the stock size was known with certainty than another fishery with the same average stock size, but where the stock size varies from year to year due to disease.[3] Thus, the level of investment is less in the stock with disease. The corresponding harvest is smaller, providing harvest is not being exploited beyond maximum sustainable yield (MSY). If it is beyond MSV, then the presence of disease strangely enough increases the harvest level.

Another change in fishing behavior brought on by the presence of uncertainty in the stock size is that of risk-spreading. This is manifested by fishermen investing in the appropriate gear to allow them to switch to other fisheries when a disease outbreak occurs in the primary fishery. It is a characteristic of many inshore fisheries.

Disease in a Closed Access Fishery

The discussion of open access fishing is not always appropriate. Many capture fisheries and aquaculture have characteristics which alter some of our previous arguments. Whereas fishermen in open access have little incentive to defer production, those in a closed access fishery may actually allow or be forced to defer current harvest with the expectation of returns in future harvest. However, when the uncertainty of mortality from disease increases, these fishermen and managers have an incentive to allow more harvest, as well as catching smaller, faster growing fish. This behavior is demonstrated by the change in harvesting strategy of Delaware oystermen in response to MSX (Haskin and Ford 1983). These oystermen reduced the marketing age of oyster to allow only one summer's growth. This permits harvest before MSX related morality could occur. Plantings were also made in new areas where conditions resulted in poor growth, but were free of MSX. In France, shifting from the flat oyster (*Ostrea edulis*) to another variety (*Crassostrea Gigus*) occurred following disease outbreaks (Grizel 1983).

In many instances, the closed access fishery leads to greater problems of disease. If "farming" is practiced, greater density of stock is likely to occur. The increased density itself may lead to greater risk of disease and to its rapid transmission. Examples of outbreaks in closed access situations include salmon culture (Eklund et. al 1984), live lobster marketing (Stewart et at. 1975) and blue crab shedding operations (Newman and Ward 1973).

Another potential source of disease in closed access systems is the introduction of disease through "exotics." With open access, no one has an economic incentive to introduce new varieties. In closed access, there are potential returns from introducing new, high yielding varieties. Unfortunately, there are possible dramatic losses, especially in areas where newly introduced disease organisms can be easily transmitted (by the water column) among the "farmed" stocks. Oyster culture in Brittany,

[2]See for example: Smith (1980), Dudley and Waugh (1980), Andersen (1982), Bockstael and Opaluch (1983), Andersen and Sutinen (1984), Ploude and Bodell (1984), and Yohe(1984).

[3]It has been shown empirically that New England fishermen are on average risk averse (Bockstael and Opaluch 1983).

France, is reported to have introduced several disease organisms (Grizel 1983) from importation of a Japanese and North American oysters.

Revenue Losses

A few studies which discuss the economic effects of fish disease usually measure these effects by losses in total revenue. Sieling (1971) estimated that Delaware Bay losses due to MSX totaled $3 million per year in lost revenue. He showed, however, that in Virginia and Maryland, the lower production due to MSX was offset by higher dockside prices. Total revenues in Virginia were unaffected, while revenues in Maryland actually increased. Oyster disease in Brittany lowered revenues from *Ostrea edulis* culture from 131.6 million francs to 65.2 million francs (Grizel 1983). This loss was somewhat offset by increased culture of *Crassostrea angulatus*. The industry use of labor declined from 742 thousand man-hours to 492 thousand man-hours.

An Illustration of Profit Losses

While the lost revenues associated with disease outbreaks are somewhat indicative of the lost profits to producers, they are by no means a precise measure. As has been documented (Haskin and Ford 1983), producers can shift to production of other items to offset their losses. Moreover, many costs are directly related to production levels. As production and revenues fall, so do costs. Thus, lost revenues from harvest, at best, represent the worst possible losses to the harvesting sector. Most often these are not the only lost profits from the disease.

To illustrate this point, we explore the events surrounding the initial outbreak of MSX in the Chesapeake Bay. Beginning in Virginia's 1959-60 oyster season, MSX began causing mortality in adult oysters. Mortalities ranged from 20 percent in the first year to over 50 percent in later years (Andrews 1968). By the end of the 1966-67 season, mortality from MSX stabilized and in some areas of Chesapeake Bay, oysters showed signs of resistance (Farley 1975). Using commercial data provided in Haven et al. (1977), we derive estimates of lost profits and behavioral changes for this period. These numbers are provided mostly for illustration and should not be treated as complete estimates. A more intense research effort would be required to increase their accuracy. The process of estimation, however, is useful for illustration. Although profit losses by watermen are observed, our major concern is the losses to private growers from the MSX. Planters were most affected by MSX because the seed purchased by them suffered severe mortality from the protozoan invasion.

The planter makes profits by buying seed oysters, planting them, and then harvesting them several years later. The expected profected profit for a two year cropping pattern is given by:

$$(1) \qquad E\left[\left(f(Q_s)\,(P_{t+2}) - CH_{t+2}\right)(1+r)^{-2}\right] - (p_t + c_t)\,Q_s$$

Discounted Expected Net Returns in $t+2$ — current costs

where:

$E[.]$ is the expected value operator;

$f(Q_s)$ is the output of marketable oysters (bushels from the planting of Q_s bushels of seed);

P_{t+2} is the market oyster price in year $t+2$;

CH_{t+2} is the harvest costs per bushel of marketable oyster in year $t+2$;

r is the rate of interest;

p_t is the price per bushel of seed in year t; and

c_t is the transportation and planting cost of seed in year t.

The epidemic associated with MSX reduced the marketable oysters produced from seed by an amount $(1 - \%MSX_{t+1})\ (1 - \%MSX_{t+2})$ where $\%MSX_{t+1}$ is the percent mortality associated with MSX during year $t + 1$. Thus, as the mortality rises, expected net revenues will fall, decreasing the damand for seed oysters. It is in the seed oyster market that we estimate the losses to growers. Following Just et al. (1982), changes in demand for an essential input (in this case oyster seed) can be used to estimate profit losses to individuals "up" the market chain. We calculate changes in profits for the period 1959 to 1974 that we can attribute to MSX. The "welfare" changes are shown for both the seed harvesters and the growers.

In order to make equation (1) operational, we assume the grower expects the market oyster price and harvest costs in subsequent years to be equal to the current values. We also make the assumption that the mortalities suffered in the contemporaneous season are expected for the subsequent growing seasons. Finally, we postulate that after 1966-1967 growing season, the growers required a "premium" above normal profits in order to absorb the newly acquired risk of subsequent MSX outbreaks in the industry. Our derived demand for oyster seed becomes:

$$(2) \qquad QD_t = g\,(P_t - CH_t)\,(1 + r)^{-2}\,(1 - \%MSX)^2,\ p_t + c_t,\ R_t)$$

$$= g\,(DNR_t,\ p_t,\ R_t)$$

where R_t is a binary variable, equal to one for all seasons following 1966-67.

To complete the seed oyster market, we specify that the quantity supplied is as follows:

$$(3) \qquad QS_t = k\,(P_t/p_t,\ Q_{t-1}).$$

This is the simple partial adjustment model (Maddala 1977). We assume seed tongers are responsive to the relative prices of market and seed oyster price (they can harvest either seed or market oysters) but that their responsiveness is not complete after one year. If the relative price of market to seed oysters rises, one expects less production of seed oyster. Fishermen direct effort to tonging market oysters. The partial adjustment model captures the possibility that not all of the movement to the market oyster is made immediately (in one season). Fishermen may slowly adjust. Data for MSX mortalities is provided by Andrews (1968).

An instrumental variable regression analysis produced the following estimated equations:

$$(4) \qquad Q_t = \underset{}{4.24} - \underset{(-2.22)}{1.73\,p_t} + \underset{(1.26)}{0.24\,DNR_t} - \underset{(2.10)}{0.91\,R_t}$$

$$(5) \qquad Q_t = \underset{}{0.58} - \underset{(1.52)}{0.19\,(P_t/p_t)} + \underset{(10.74)}{0.97\,Q_{t-1}}$$

where the t - ratio is shown in parenthesis below coefficients. The signs of all coefficients are as expected, although the standard errors of coefficients associated with discounted net revenue and relative price variables are relatively high. The coefficient of 0.97 on lagged seed suggests that if tongers respond to price, they do it

in a very slow fashion, about 3 percent per year. This is not too difficult to believe, however. The risk premium (R) for the additional MSX risk was approximately $.50 per bushel of seed. In other words, planters after the 1966-67 season required an additional profit of $.50 per bushel of seed to bear the additional risk of planting.

Using the coefficients in equation (4) and (5), we calculate tonger and planter losses from the MSX episode. For the seasons 1959-60 through 1974-75, the loss in profits to seed harvesters and planters averaged around $2.2 million per year (1967 dollars). The high was around $4 million and the low around $1 million. The harvesters of the seed suffered about 40 percent of the losses whereas the planters suffered around 60 percent of the losses.

There are without doubt oversights and exigencies inherent in our analysis. For example, we chose to use an *ex ante* approach to the welfare change. By looking at the grower supply of market oysters, we may have been able to examine *ex post* losses. No consideration has been given to the welfare of marketing agents in the wholesale and retail trade. Finally, the consumer is ignored.

Effects on the Demand for Fish

Thus, far, the attention has been on the harvesting sector. There are, however, potential dramatic effects of disease organisms on consumers of fisheries products. Our guess is that consumer losses are of equal or greater magnitude with those of fishermen and processors. Unfortunately, data do not exist to substantiate this claim, though we can still discuss the manner in which consumers are affected by disease organisms in fish.

Price Effects

The most obvious manner in which consumers are affected is the lost production discussed in the previous section. Consider the effects of decreased production in a community which does not import seafood. Because one cannot consume a product which has not been produced, consumption cannot exceed production. Since production has fallen, consumption also must decrease. Clearly someone will go without the item and others will pay more to consume the same amount.

The dollar loss to the consumer can be measured by the change in household expenditures necessary to keep the household at the same standard of living given the higher price. While this might be small for each household, the total effect when all households are totaled may be considerable. The losses should be greatest for those items for which consumers do not easily substitute other goods. Outbreaks of gaffkemia (*Aerococcus viridans homari*) in the lobster industry likely will reduce consumer welfare more than an outbreak of similar proportion of *Ichthyophonus* in haddock. There are simply more substitutes for haddock than for lobster.

Quality Effects

In addition to higher prices, there can be other effects on consumers from quality changes which arise because of disease organisms. These effects can be divided according to whether the disease organism changes the taste/product form or consumers' health. Each will be considered separately.

Taste and Product Effects

Perceived Changes. The analysis of product quality has been a major research area for economists for the last twenty years. Early work on differentiated agricultural products (e.g., Waugh 1929) was generalized into a theory of consumer demand for quality (e.g., Lancaster 1966). The theme of the analysis is that consumers consider quality as well as price when they buy products. It is now commonplace in economic analysis to consider quality as well as product price in the demand for goods (e. g., Hanemann 1982).

The manner in which fish disease effects are perceived by consumers can be quite straightforward. The following excerpt is somewhat illustrative:

> *Ichthyophonus* infected haddock have been recorded from commercial landings in Scotland . . . resulting in [discard] . . . due to flecked appearance, rubbery texture and obnoxious smell. (McVicar 1980, p. 3).

These particular quality differences are so overwhelming that the fish is immediately discarded by the merchants. They know consumers will not buy spoiled fish. In this case, the haddock suffer from economic mortality rather than physical mortality. Similarly, processors are reluctant to accept tanner crab infected with a fungal disease known as black mat syndrome (Sparks 1982, Hicks 1982). Black tar-like material on the crab's exoskeleton can break into the crabmeat resulting in an inferior product. The effect of discard culling on consumers is the same as with physical mortality increase: lower consumption and higher prices.

Unperceived Changes. Not always is the consumer able to make accurate quality judgements on their fish purchases-and their learning experience may actually be more troublesome than higher prices. Take, for example, a homemaker who purchases an infected fish that has escaped culling. They prepare it and find during the meal that the fish is rancid. Not only is the preparation time lost, but there is no easy alternative for the meal. Having seen the situation, the authors are aware the higher prices may not be the most costly effect to the consumer of disease organisms in fish and shellfish.

Scenarios such as the above, form our "experience" (Nelson 1970) about fishery products. After enough time, the consumer has an expectation of the quality of the product. One or two rancid fish expereinces lower expectations of quality and therefore reduce demand. These reactions lead to lower welfare to the consumers from the unstandard quality. Exactly how the experience is formed and how many bad experiences are required before demand is affected is not well understood.

It is understood, however, that the absence of enforceable minimum quality standards in fish may contribute to losses suffered by consumers and the industry (Bockstael 1984). The reason derives from the rational strategy for a fish monger. If each producer's output is not differentiated (i.e, a fish from Giant is the same as one from Safeway), then the producers do not receive monetary incentives from maintaining quality. The average quality of fish is not as high as when the consumer knows the difference between products. The welfare of consumers and producers is lower because of the undifferentiated quality. While it is not always true that products are undifferentiated—consumers often remember that Store X has good fish and Store Y does not, the fact remains that because Store Y has poor quality fish, consumers who go to Store Z where the quality is unknown, are less likely to buy fish than a Perdue chicken. Greater quality standards and enforcement of

existing ones can potentially improve both the welfare of consumers and producers in this case.

Do disease organisms in fish lower the quality of fish/shellfish in a manner which cannot be perceived by the consumer? The answer is yes, although the extent of the problem is not clear. *Icthyophonus* is considered responsible in part for the undesireability of slowly marketed fish. "Proteolytic enzymes released during the growth of *Itchyophonus* . . . contributed to the rapid decay in the muscle." (McVicar 1982). The disease organisms can also spread into uninfected fillets, even at temperatures as low as 8 degrees Centrigrade (McVicar 1982). Herring exhibits similar degeneration if infected with *Itchyophonus*. On the West Coast, Pacific whiting, milky halibut, sole, flounder, and salmon all potentially have their quality affected by myxosporidian spores. Upon harvest of the fish, the parasite releases an enzyme which diffuses away from the cysts. The texture of the flesh softens and the quality of the product diminishes. (Patashnik and Groninger 1964, Patashnik et. al. 1982).

Probably more disconcerting to consumers, however, is the potential for nematodes (worms) in seafood. Not always can they be perceived:

> A cod, caught on ocean side of Cape Breton Island, Nova Scotia, was filleted and purchased from a fisherman at Ingonish Beach, Nova Scotia on 5 August 1972. Within an hour, one of the fillets was cooked outdoors over a gasoline campstove. While consuming the fish, one of the structures taken for "veins" was seen to move; it was recognized as a worm, and since other such "veins" had been ingested, this specimen was kept in charcoal transport medium intended by one of the party for bacteriological sampling. Enquiry of local residents revealed that pollack was considered to be of good quality although cod was recognized to be wormy during the summer. During the evening of 11 August the 22-year-old woman who had eaten the infected cod (filleted 6 days earlier) felt a "tingling" sensation in her throat and extricated the apparent cause—a live nematode. (Kates et. al.).

The incident suggests nematodes (in this case *Phocanema*) are perceived by some and not perceived by others. Without some rules of thumb (e. g., Nova Scotian cod is wormy in the summertime), the consumer is left to question the quality of the product. Evidence that the roundworm problem for consumers is not specific to Nova Scotia/Gulf of Maine comes from reports of similar incidents throughout the country (e.g., Juels, et al. 1975, Dailey et al. 1981). Research during 1976 has shown that roundworms occurred in 10-20 percent of the fish (edible portions) caught in Washington, Oregon, and California waters during 1975. West Coast shellfish did not show the presence of roundworms (Myers 1979).

To believe roundworms reside only in finfish, however, would be a mistake. Scallops, shrimp, and surf clams are among the commercially valuable shellfish hosts of nematodes (Norris and Overstreet 1976, Sawyer et al. 1983). An interesting episode occurred in the surf clam industry during 1975. Dark roundworms *Sulcascaris sulcata)* suddenly began appearing in surf clams. These were noticeable to processors who were quite concerned about the impact on their market from the obvious presence of worms. As it turned out, the worms may have been present in surf clams for some period and only became obvious when a haplosporidan protozoa *(Urosporidum spisuli)* also invaded the clam, discoloring the worms. Since then, it has been claimed that this nematode "may parasitize a wide range of molluscan hosts" (Sawyer et al 1983).

Human Health Effects

Although the unpercieved quality change may reduce demand and consumer welfare, a far more serious problem arises when the unperceived quality is linked with human disease. The distinction between whether or not human health is involved has important economic as well as public safety ramifications.

There appears to be growing concern about the human health effects of fish consumption. Myers (1979) explains:

> The anisakine nematodes attracted attention during the early 1960s because their presence reduced the commercial value of fish (8). Large anisakines, such as *Phocanema* sp. larvae, were easy to detect in both the edible and nonedible muscle of a large variety of fish, especially the cod. Extensive studies were conducted in Canada on this so-called "codworm" but the parasite was thought to be merely unappetizing. Work was directed toward removing it or reducing the number of nematodes visible in fish fillets (8). That this anisakine could be a human pathogen was not considered. During the past 10 years, however, there have been an increasing number of reports that these nematodes can infect humans (6).

In essence, what was believed to be a taste/form effect has been linked to human health. The nematodes are just one of numerous potential causes of human disease/reaction arising from consumption of fish and shellfish.

Outbreaks of typhoid fever, for example, have been related to U. S. oyster consumption since an outbreak in Connecticut in 1893. In 1910, the annual report of Virginia's Board of Fisheries claimed "The scare of 'polluted oysters' has cost the workers of Virginia three to four million dollars a year for three or four years." (Capper et al. 1983). A 1924-25 typhoid epidemic in Chicago and several eastern cities caused around 150 deaths and was linked to indigestion of raw oysters harvested from Raritan Bay in New Jersey. Over the years, however, better sanitation facilities and water quality requirements of oyster harvesting areas have largely eliminated problems of health hazard caused by bacteria.

Viral infection has not been as easily addressed. The reason, according to Metcalf (1979), is that the water quality criteria for closure of shellfish areas is based on fecal or total coliform count. While these may be reasonable indicator organisms for bacteria, they are shown to have little relation to the presence of viruses (Goyal et al. 1979). Enteroviruses, reoviruses, adenoviruses, and heptatitus A are common viruses which are likely to be transmitted by shellfish. The shellfish apparently act as passive hosts for the virus (Chang et al. 1971) and enter humans through consumption of raw seafood.

Numerous examples of gastroenteritis outbreaks exist but the one following a meeting of a Northeast shellfish sanitation association highlights the problem. The meeting, held in New Haven, Connecticut, featured a social hour during which raw clams were served. Seventeen of the 19 persons who consumed the raw clams developed gastroenteritis, symptoms of which included nausea, vomiting, fever, and diarrhea. The possible cause of the clam contamination was a power failure in Norwalk, Conn. The outage resulted in partially treated sewage overflow into waters upstream of clam beds. The beds were subsequently closed to shellfish harvest, (*J. of Infectious Disease,* 1969, pp. 265-66).

Ciguatera is slightly different in causing human reaction to seafood consumption.

It is a toxin found in certain fishes in the Caribbean and around Hawaii. It apparently arises when fish consume dinoflagellates and humans consume the fish, even cooked. There is no way to identify these hazardous fish, but individuals who eat them experience nausea, vomiting, severe diarrhea, and abdominal cramps. It has even been known to cause death *(San Jose Star Magazine,* October 4, 1981, p. 3-6). Deaths are also known to be caused by botulism in smoked fish.

The wide range of health hazards resulting from fish/shellfish consumption ultimately influences the fishing industry and other users of the water. We first discuss the consumer reaction and then examine the fishing industry and related industries.

Consumer Response. Consumer response to human disease organisms in fish is conceptually similar to the quality changes discussed previously. The main difference, however, might be a substantially greater response. Disliking or throwing away a dinner is not as traumatic as vomiting, stomach cramps, hospitalization, or death. Risk averse people will likely avoid fish more if their health is involved. Weighing against this argument is the fact that most human disease organisms can be eliminated by a thorough cooking. People can avoid the nuisance by changing the form in which the seafood is consumed. Thus, if cooked seafood is nearly as preferred as raw seafood, there may be little consumer reaction to the disease organism.

To the authors knowledge, little is known about consumers preference between raw and cooked seafood. Data normally are not collected on prices and quantities consumed of different product forms. Our best guess is that cooked seafood is not a perfect substitute for raw seafood. Behavior to avoid the nuisance (Shulstad and Stoevener 1978, Swartz and Strand 1981) would result in a loss to the consumer from human disease organisms and lower demand for seafood.

Because the data are not available to study this problem empirically, we can only present results from related research and speculate on potential losses. It is known that consumers respond to quality characteristics of seafood. Bockstael (1977), for example, found that, for New England groundfish, "The relative price of fresh and frozen fish has been such that only in relative gluts, or when existing fresh fish channels were full, would it be worthwhile to divert domestic catch to the frozen market" (p. 38). Consumers are generally willing to pay a higher price for fresh fish. Moreover, it has been shown that consumers of shucked oysters were unusually responsive to potential health effects arising during the Kepone closure of Virginia's James River (Swartz and Strand 1981). Although there is not overwhelming evidence, we conclude that consumer reaction to disease-related quality in fish is a serious problem for the industry.

Because there is so little information on consumer response, it is impossible to obtain estimates of consumer welfare losses from disease-realated organisms. Nevertheless, figures regarding the medical treatment expenses associated with botulism from canned salmon cost are revealing. Treatment of one individual cost approximately $65,000. This offers some guidance as to the medical expenses for an individual who eats diseased fish.

Theoretically, there is a difficulty in measuring welfare losses when human health is involved. Typical welfare measures [such as those previously used] only offer reliable guidance if large changes in the individuals' state of welfare are not involved. They, in some manner, are based on a compensation to offset a change in

an indiviudual's state (situation). When dramatic changes in an individual's state are involved, the measures sometimes cannot be used. The extreme example is the case in which no amount of money can compensate the individual for the change of state. Death might be the clearest example but surely one has heard the expression " I wouldn't go through that again for anything." In these cases, typical welfare measures might not be very useful and one is left in an unsatisfactory position regarding loss estimation.

Production Losses. Perhaps as a result of the potentially extreme losses when human health is involved, federal and state governments have interceded in an attempt to reduce the potential for human health effects from fish with disease organisms. The apparent expression of policy comes in the prohibition of harvest from particular waters. Because there is an available resource in these areas which is prohibited from use, the possibility exists for a negative impact on producer and consumer welfare. But, because the disease organisms are present, harvest is not undertaken and production is lost.

Shellfish production is an obvious example of production losses. In 1980, nearly 15 percent of the Nation's estuarine water were classified as prohibiting shellfish harvest (Verber 1981). Atlantic Coast estuaries had 96 percent of all U. S. waters under shellfish harvest prohibition.

Unfortunately, it is not possible to determine the value of the lost production. First, is is often possible to harvest shellfish from prohibited areas if the purpose is replanting. Here, the industry does not lose production, prohibition merely raises the costs of harvest. Secondly, it is never clear whether stocks from a closed area would be harvested if the prohibition was not in place. The presence of shellfish is not sufficient for their harvest. Costs and prices must be considered. Finally, these prohibited areas may act as sanctuaries for the shellfish. Because of their existence, reproduction might be enhanced.

Ending on a positive note, the shellfish prohibition figures show that acreage with shellfish prohibition has begun to decrease. National prohibited acreage went from 3.79 million acres (1.53 million ha) in 1974 to 2.89 million acres (1.17 million ha) in 1980, a 25 percent decrease. Some of the improvement is due to water quality improvements and some to alternate methods of regulation.

Other Related Losses

The issue of improved water quality brings up an important component of loss from disease, the opportunity costs incurred to assure populations of disease organisms are held "reasonably" in check. Historical review notes that the reason Baltimore built a state-of-the-art sewage treatment plant in 1912 was primarily to protect the good name of Maryland oysters (Capper et al. 1983). The increased expense to Baltimore citizens for the purity of wastewater discharge was due solely to the disease potential of oysters. These expenses, then, convey income losses to Baltimore citizens from the existence of the organisms.

The Back River Disposal Plant in Baltimore is representative of the technologies we use to avoid greater presence of disease organism in our waters. Between 1973 and 1984, the federal government awarded $32 billion in construction grants for sewage disposal. Obviously not all of this money was directed to suppressing disease organisms in seafood. However, our society allocates a substantial portion of resources each year to assuring the cleanliness of our estaurine systems. A fair

portion of that allocation is undoubtedly attributed to the presence of disease organisms in fish and shellfish.

Management Implications

In this paper, fisheries management is not considered synonymous with harvest restriction. National Marine Fisheries Service and other fisheries agencies have broad responsibilities beyond the direct control of harvest. In what follows, we examine the implications of the previous discussion on several of these missions, combining them under the general label of management. In particular we consider research direction, input controls (prophylaxis), and harvest management.

Research Direction

Much of the literature cited in this paper primarily addresses the life history as well as etiology of enzootic and epizootic occurences. In this role, the biological researcher is like an historian of the microscopic world, recording and interpreting events. The value of the biological research derives not only from the information itself but also from how it is used. The research on MSX, for example, may have provided planters with valuable information on expected mortality. Armed with this, planters may have made better decisions and not sunk valuable time and resources into dying oysters.

Government sponsorship of the research is important because no one planter is likely to have the incentive or capital to obtain the information. Moreover, when the information is publicly supplied, it is available to all, including future generations. Its potential for pay-off therefore may be high. It might even provide a classic circumstance from which valuable insight can be drawn.

To measure the "social returns" from the provision of this information is difficult if not impossible. One would have to determine the circumstances (e.g., industry profits) without the information and then determine the conditions with the information. The difference would reflect social returns. The impact of information, however, might be far-reaching. In their pursuit of generalization, scientists often require an understanding of many cases before a pattern emerges. Thus, beside the more direct gains from information, the research can have a long-lasting effect on science in general. Typical methodology to account for the more general is not likely to yield measures of the returns that are above reproach.

One area for which research might provide valuable information is in the area of consumer preferences for raw seafood. Our review noted little to no knowledge of the consumer preferences for raw fish. Yet this is a critical piece of information both for the industry and for the government. Without an understanding of the consumer trade-offs between raw and cooked fish, there are only indirect methods for determining losses to consumers from the disease organisms. A directed research effort along these lines could be quite valuable. If one found out, for example, that consumers were largely indifferent between raw and cooked fish, efforts could be directed to telling/requiring people to cook the fish. On the other hand, if they preferred raw fish to cooked fish strongly, efforts might be directed to assuring that products were free of parasites.

Input Control (Prophylaxis)

Another important use of basic research is in the prevention of disease outbreak. It may be possible to restrict the spread of disease through intervention. The literature suggest three methods for input control:

1. Direct protection-measures which alter the host so that infection can not occur;
2. Eradication—measures which eliminate the pathogen after it is introduced;
3. Exclusion—measures which prevent the introduction of pathogens into an area.

Sindermann (1985) has discussed the relevance of these methods for extensive (in a common water column) aquaculture and intensive (in private water column) aquaculture. We would like to discuss the role of government in the two settings.

With extensive marine aquaculture, there is a serious potential for entire industry effects from one producer. That is, if one aquaculturist introduces disease into a common water body, then many aquaculturists are affected. There is a strong tradition, even in our free market, to reduce or eliminate these external effects. The obvious way is through control of transfer or exclusion. Here, the role of government is regulation.

On the other hand, the government's role in intensive aquaculture may be substantially less. If an individual wishes to risk his entire enterprise by using an exotic, he is only going to affect himself. In a free market, he should have this freedom without government regulation. There may be a research role for the government, however. Just as the U. S. Department of Agriculture provides research on agricultural production and marketing, so too is it appropriate in aquaculture. The public good nature of information provides a high potential pay-off from centralized research.

Harvest Management

Rational fisheries management should make allowances for the existence of disease organisms in fish and shellfish. The traditional goal of fisheries management is a suppression of current harvest so that future harvests are improved. Because disease organisms can either directly or indirectly influence harvest, the clever manager will understand their effects and use them to an advantage. Without the understanding, the manager's policies can be enhanced or shunted in an apparent whimsical fashion. A general picture of disease/harvest interaction currently is unavailable, and worthwhile effort would be to expand the first section of this paper into a complete model. We offer only a sketch, the portrait remains unfinished.

There are two observations which might indicate the complexities of the general model. First, it is entirely possible that disease organisms in fish can improve future production while limiting current production—a manager's delight. The mechanism making this possible is consumer demand. Deaths from typhoid fever in the 1920s, for example, probably drove down the price of oysters and "economically" restricted their output. Here, the harvest reduction probably led to greater stocks and greater subsequent production. This is, at least, informed speculation and supported by some circumstantial evidence.

More troublesome, however, is that we do not even know whether disease induced mortality will and *should* lead to greater or lesser current harvest. One could

argue that harvest should increase because the infected specimens will just die anyway. On the other hand, one could argue that the increased mortality endangers the stocks, and reduced catch is necessary to prevent a complete collapse of the fishery. Our guess is that the correct answer depends on the parameters of the system and that circumstances dictate the appropriateness of the policies. Which particular parameters and circumstances result in recommendations for greater or lesser current harvest is the question. Greater understanding of these might prove valuable to fisheries managers.

Conclusions

Our intention in this paper is to explore the relationship between fish disease organisms and economics and then relate it to fisheries management. We had no predispositions before the effort. We found, to our surprise, that problems of fish disease were ubiquitous, significant, and possibly increasing. They existed for numerous species and for most types of fishermen. Moreover, the economic impacts of disease organisms were wide-ranging—from altering producer expectation to reducing demand for products. We document many cases and attempt to show how one would measure losses from existence of disease organisms.

We also begin to explore why the situtation is important to managers of fish. Admittedly, our efforts are restricted. While there is a long history of government intervention to prevent the spread of disease in agriculture, the role of government in relationship to fish diseases is relatively new. We hope that it could be eliminated but, in fact, believe it may become far more important in the future. If we are right, research along the lines developed in this paper may prove valuable.

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