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The Role of Disease

*One of the most serious gaps in our knowledge of marine ecology in the study of diseases. To what extent do pathogenic bacteria, fungi, viruses, and other groups affect populations of plants and animals? For this chapter I have assembled a number of examples to illustrate that epidemics * are common occurrences in marine environments, and sometimes have devastating consequences. They may be an important cause of fluctuations, and should therefore have a prominent place in research programs.*

Most scientists agree that understanding of the biology of a marine organism can come only by studying it as a component of an ecological system. This means taking into account the physical characteristics of its environment and the action and interaction of all the competing predaceous and prey organisms composing the system. This is what ecology is and what ecologists seek to understand. However, one aspect of marine ecology which has been peculiarly neglected is disease.

It is true that when biologists draw up ideal programs of ecological research they mention diseases and parasites, but always far down on their list of things-to-study-if-opportunity-permits. And opportunity does not permit. It is an exceptional marine laboratory that provides for a pathologist or epidemiologist on its permanent staff. Marine microbiologists, of whom there are precious few, do

* Strictly speaking, "epidemic" refers to diseases affecting people, and is therefore not properly used in this text. However, it has the advantage over "epizootic" and "epiphytotic" of being a familiar word, and I have therefore indulged in the license of using it in referring to diseases of animals and plants.

not often focus attention on pathogens because most microscopic animals and plants are not pathogenic. Moreover, disease is such a highly specialized subject that one can study it profitably only if he gives it full-time continuous attention and has certain special equipment which marine laboratories usually lack. Hence, the intellectual atmosphere is not very encouraging to the study of marine diseases. Nevertheless, during the past seventy years a few scientists have described a number of pathogenic organisms incidentally to their other studies. They have recorded enough epidemics to suggest that disease might be a much more potent factor in marine ecology than it is usually assumed to be, perhaps as destructive to the animals and plants in the sea as it is to those living in fresh water and on land. If this is true the failure to include disease within the scope of marine biological researches might explain why fluctuating abundance continues to be a mystery.

In the following pages I shall give a few examples to illustrate that diseases do exist in the sea, that they are distributed widely over the world in different classes of organisms, that some of them are deleterious to their hosts, and that they occasionally devastate whole populations.

An example of a well-documented disease is that due to the fungus *Ichthyosporidium* which infects several kinds of fishes in the North Atlantic Ocean. It is endemic to the herring of the western side of the Atlantic, but apparently does not affect the other stocks of that vastly abundant species. American fishermen recognize it by the black specks it makes on the skin of the fish, from which they give it the name "pepper-spot disease." They will tell you they have always known about it, and so had their fathers and their grandfathers before them. They find that the disease is always present. Because the disease sometimes affects the quality of fish to such an extent that canners must cull out infected ones, government biologists have had occasion to keep records of the incidence of infection. The percentage of sick fish fluctuates from year to year and from place to place. It is higher in winter than in summer. It never goes below 1 or 2 per cent, and it may reach 70 to 80 per cent during the height of infection in a season. Certain other fishes, for example the alewife, the winter flounder, and the mummichog, catch the same disease apparently where they frequent waters inhabited by infected herring.

The disease organism is suspected of gaining entrance through the intestine, presumably carried there by some infected food organism or by another kind of parasite. Flatworms may introduce the spores with their borings into the intestinal wall of the fish. By what-

ever means the fungus gets in, it sends forth thread-like processes which produce strong enzymes that break down the proteins of the host tissue on which the fungus depends for its existence. Thus the plant dissolves its way throughout the body of its host, through kidneys, spleen, blood vessels, brain, muscles, and skin. It is inconceivable that a fish so overwhelmed could long survive. During severe epidemics, fish caught in the herring traps skim about at the surface in circles, on their sides, as though intoxicated. When they are in that condition, other kinds of fishes, gulls, and seals find them easy prey.

On the European side of the Atlantic, the same fungus, or at least a closely related species, infects mackerel, hake, sea snail (a fish), flounders, cusk, cod, and haddock, and in lakes on the European continent, trout and other fresh-water fishes! [1] In the mackerel, the disease organism seems to be limited to the viscera, particularly the kidney and spleen. It rarely attacks the muscles as it does in the American herring. Nora Sproston, studying this disease in mackerel at Plymouth, England, in 1940, 1941, and 1942, found the incidence in samples to fluctuate between zero and 100 per cent. Of all the specimens she examined during those three years, the percentages infected were 70, 38, and 69, respectively. She wrote, "The disease is a very serious one, showing no signs of abatement." No one knows what proportion of the mackerel population this disease kills. In any event it is a nuisance to fishermen because the proteolytic enzymes produced by the fungus cause infected specimens to become centers of putrefactive bacteria soon after death, so that the presence of even one in a barrel will quickly spoil the others.

Haddock of the western side of the Atlantic seem to be free of this fungus disease—at least no cases have been reported—while those of the eastern side are not. In them it causes such a discoloration of the muscles that infected fish must be condemned as unfit for human food. Fishermen are familiar enough with them to apply such special names as "spotty haddock," "greasers," "smelly haddock," and so forth.

What transmits this disease is a mystery. Fish do not seem to catch it from each other, at least not immediately, for healthy herring have been kept in tanks full of infected ones for an entire summer and in tanks full of spores of the fungus without becoming infected.* Perhaps the disease is transmitted in food, but no massive infections have yet been observed among the organisms on

* Leslie Scattergood and Carl Sindermann are studying the etiology of this disease at the United States Fishery Station, Boothbay Harbor, Me.

which herring and mackerel feed. Moreover, the experimental fish were fed mysids which had been injected with spores, and copepods which had been exposed to them, and still no symptoms of the disease developed by the end of the summer. The possibility that food is the carrier remains open, however, for the fungus *Ichthyosporidium* has been provisionally identified in a few specimens of copepods in the Mediterranean (in *Acartia*), and in the Clyde Sea area (*Calanus*), showing that it might occur among plankters.[2]

Ichthyosporidium has not yet been reported in the Pacific; but Miss Sproston at Plymouth found in a can of California sardines some particles that looked very similar to spores of the fungus which produces the Atlantic mackerel disease. A primitive aquatic phycomycete, probably related to *Ichthyosporidium*, caused a heavy mortality of rainbow trout in a commercial hatchery in Washington.[3] And a herring packer of Alaska described to me an occasion in 1945 when Pacific herring obviously had something the matter with them, for they swam round and round at the surface as though quite mad, which is what Atlantic herring do when severely infected with pepper-spot disease.*

It is difficult to be certain about the taxonomy of *Ichthyosporidium*, for the plant body varies in form with different hosts. Is it really only one species that attacks the various kinds of fishes in the North Atlantic, as well as trout on the continent of Europe and copepods in the Mediterranean, or are there several species? This is an open question, probably to be most profitably attacked by a team having competence in both biochemistry and microbiology.

Another fungus, belonging to a different group (Saprolegniaceae) was identified with an epidemic disease of a copepod (*Eurytemora hirundoides*) in the northern Baltic in August 1950. Apparently it had not been observed there before. Sten Vallin wrote about it as follows: "Where the mould came from, and why it appeared first in the Bay of Sundsvall, are questions to which we have no answer . . . Probably high water temperature favors the mould. The optimum temperature for the related crayfish disease fungus is 20–25° C., though it can also be virulent in the winter."[4] The disease killed a large though unknown proportion of the local population of *Eurytemora*, which sank to the bottom in large enough quantities to foul fishermen's nets with the sticky substance of the decomposing bodies. The disease recurred in the same area the next year, and though it again killed many *Eurytemora*, the destruction was not so widespread as it had been in 1950. During the summer, *Eurytemora* is normally the commonest copepod in the surface waters of the

* This is a symptom of many other diseases of fishes, however.

central Baltic; and since it is an important constituent of the food of plankton-feeding fishes, any diminution in its numbers probably adversely affects the abundance of herring, among other species.

Parasitic marine fungi must be exceedingly common and widespread. F. K. Sparrow, an American mycologist working at Woods Hole during two summers (1934 and 1935), distinguished seventeen species, two of them new, and described two new genera. Of the seventeen, all but one were parasitic or saprophytic on various species of algae, and one was parasitic on the eggs of a microscopic animal, probably a rotifer.

Sparrow had previously found seven of these fungi on the other side of the ocean, in the Kattegatt, and other mycologists have recorded several in the arctic, the North Sea, the Baltic, the Gulf of Naples, and the Adriatic.

The commonest fungus in Sparrow's Woods Hole collection, *Ectrogella,* is parasitic on certain diatoms * to such an extent that during one month (July, 1934) 88 per cent of these plants were infected. Sparrow describes the importance of the disease thus:

> At no time during the process of disintegration of the host cell could the presence of any other type of organism be detected. . . . The fungus had apparently so completely absorbed the available nutriment that even after infected cells had become broken or had fallen apart, bacteria and protozoa were seldom seen feeding on the residual material.
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> It was very evident that not only could the fungus initiate the infection, but once inside, could bring about, unaided by other agencies, the almost complete disintegration of the contents. Further, the thallus, derived from a single zoospore at maturity, became transformed into a myriad of swimming spores, each of which was potentially able to infect another diatom.
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> . . . a closer examination of this disease might reveal that, in some instances, it is responsible for the partial or complete disappearance of the diatom from a locality. . . . It is not known whether similar epidemics may be produced by *Ectrogella* among pelagic species. In this connection, certain of the peculiar rhythms of "flowering periods" of pelagic diatoms which have been noted in the past and which cannot be attributed to changing physical factors might well be considered from the standpoint of the presence of parasites. Due to the importance of diatoms in their role of "producers" in the sea, such a disease as that caused by *Ectrogella* should be examined in all its aspects, even though at the moment it appears to be confined to littoral algae. Further, it would be well in the future to give closer attention to the examination of plankton samples for evidences of parasitic organisms, especially during the decline of a "flowering period." [5]

Notwithstanding Sparrow's very pertinent exhortation, the only evidence that anyone gave attention to fungus parasites of algae during the next fifteen years is a few pages of brief notes scattered through the scientific literature. Then in 1950 an Egyptian mycolo-

* *Licmophora* and *Striatella.*

gist, A. A. Aleem, reported observations which he had made at Banyuls in the Mediterranean during one September, which were comparable to those of Sparrow at Woods Hole.[6] He found eleven species of fungi parasitic on diatoms and algae, not only along shore but also in deep offshore water. Three of them gave evidence of damaging their hosts, the commonest of these being *Ectrogella*, which was in several species of diatoms. Regarding the importance of these marine fungi, Aleem says:

> . . . . It is clear that the marine Phycomycetes are a very widely distributed group, capable of activity, able to survive great variations in physical and chemical conditions. Their tolerance to variations of salinity and temperature is shown by the presence of the same species simultaneously at Banyuls and on the Scandinavian coast. The role which these organisms play in the organic decomposition in the sea cannot be over-estimated. . . . Moreover, they produce great numbers of zoospores, but these are so minute that people have given them little attention. Yet they may be very important as food among the microfauna. While examining algae under the microscope, I have on several occasions seen them devoured by rotifers and other animals which live with the algae.*

A primitive fungus, *Labyrinthula*, is credited with responsibility for the pandemic of a wasting disease which, in 1931 and 1932, all but destroyed the eelgrass (*Zostera marina*) of the North Atlantic.[7] This marine flowering plant lives close to shore from North Carolina to southern Labrador, in Hudson Bay, in Greenland, and in Europe from northern Scandinavia to the Mediterranean. In the Pacific it occurs from Lower California to Bering Strait, and on the Asian side southward at least to southern Japan.

It is exceedingly important as a check to bottom erosion, as a niche to several species of larval and small fishes, shellfishes and other aquatic organisms, and as a food to aquatic birds, which feed on its seeds, underground root stocks, and leaves. People use eelgrass in various ways for insulating, packing, and upholstering material, and for fuel and fertilizer.

The pandemic started along the American Atlantic coast in the summer of 1931. The first symptoms were a peculiar streaking and blackening on the leaves of the eelgrass, usually down near the roots. Soon the blackened area disintegrated, the leaves broke off, the roots died. By the end of the first year of the pandemic, this wondrously abundant plant which had formed vast meadows along our Atlantic shore was virtually wiped out, and the beaches were piled high with windrows of dead leaves that had washed ashore. Soon

* A free translation.

after it started in America the disease appeared in France, England, and Scandinavia, with almost equally disastrous effect.

The conclusion of this devastating episode was by no means as abrupt as its beginning. Time and again the eelgrass would seem well started towards recovery, only to be struck down again. After more than ten years of such ups and downs, the stands eventually have again grown to their former state of abundance and well-being. It is puzzling that *Labyrinthula* still lives among the eelgrass, but now apparently as a harmless commensal. It might be that *Labyrinthula* has lost its virulence, or that the eelgrass has gained in resistance. Or it might be, as some authorities think, that it really was not *Labyrinthula* at all that did the damage, but some other organism still undiscovered—a bacterium, perhaps, a virus, or another fungus.

Late in the 1930's eelgrass became affected on the Pacific coast. The disease might have been carried there with eastern oysters, which are regularly planted in California bays, or with the water ballast of ships coming from the Atlantic. By whatever means the disease was introduced into the waters of California and Oregon, eelgrass declined very gradually beginning about 1938 until by 1940 only 25 to 40 per cent of the normal stand was left in some bays.[8]

The ecological effects of the disappearance of eelgrass from the Atlantic coast were severe. Shore birds which depend on the grass, notably the brant, became greatly reduced in numbers. It is also often asserted, but unfortunately without quantitative evidence, that certain coastal fishes and mollusks, crustaceans, and other small invertebrates also diminished in numbers. The effects on the Pacific coast were less striking than on the Atlantic for the disease was not so widespread or so devastating, and birds were evidently able to find other food during the time of scarcity.

In October 1938 an epidemic broke out among sponges in the Bahama Islands, rapidly spread throughout the West Indies and thence to the northwest coast of Florida, to Mexico, and to British Honduras. The orderly geographic progression of this disease corresponded strikingly with the prevailing system of currents, suggesting that the whole epidemic started from one point. Within two years, the disease killed 30 to 90 per cent of the adult populations of commercially valuable sponges. Such worthless species as loggerhead and mumjack remained unaffected, while in some places velvet, grass, and yellow sponges were almost completely wiped out. The disease organism was a fungus, tentatively identified as *Spongiophaga.*

The symptoms of the disease were identical in all the varieties of sponges, the first visible sign of it being a bald patch on the jet black horny skin of the affected specimen. On cutting such a sponge, a considerable portion of it was found to be already dead, the tissues rotted away and often of a greenish color. In many instances only a thin core of living tissue remained of a large wool or velvet sponge, the interior of which was still occupied by the skeleton. . . . After the peak of the mortality had passed, specimens were often found with large lesions showing the inroads of the malady.[9]

People engaged in the sponge fishery were aroused for a while to encourage research into the origin and nature of this blight which affected their livelihood, but their interest subsided as the sponges showed signs of recovery.

Fungi also parasitize eggs of marine animals. ZoBell has found malformation in eggs of Pacific sardine to be often attributable to fungus infection.[10] This might be a significant cause of failure of sardine crops.

Eggs of the blue crab in Chesapeake Bay become infected with the fungus *Lagenidium callinectes* and either die or hatch into abnormal larvae which fail to develop further. This fungus penetrates the eggs while they are being carried by the mother. Fortunately it affects only those eggs near the surface, and rarely goes deeper than a few millimeters into the egg mass before the eggs hatch and are scattered. This mechanism may prevent the fungus from ever overwhelming the population of crabs, unless the fungus can also attack the free swimming larvae. Unfortunately no one has studied the ecology of this disease enough to examine that possibility or to measure the annual fluctuations in the incidence of the disease and determine its contribution to the causation of fluctuations in abundance of the crabs.

It requires a rather elaborate procedure to establish the etiological role of microbes, particularly bacteria and viruses. It is necessary to isolate bacteria, which are often highly fastidious, from the infected fish. However, this is a difficult thing to do because many kinds of bacteria are normally present on fish; and though they are not normally pathogenic, they may act as secondary invaders. Most of them grow on ordinary culture media, and unless the laborious testing to fulfill Koch's postulates is carried out to conclusion, these harmless organisms can easily be confused with the actual agent of the disease in question. Proof of the pathogenicity of a fungus also depends on laboratory infection of lots of fish and the exclusion of bacteria and other possible agents.

Marine epidemics are attributed more often to fungi than to any other type of organism. This need not mean that fungi are the

most destructive pathogens of the sea, but only that they are the easiest for a nonspecialist to detect. They generally are relatively large and often have a peculiar form that readily distinguishes them as fungi. Other microbes require special stains and tests which must be based on their morphological, physiological and biochemical characteristics. Unfortunately these characteristics have been studied for only a few species, and therefore there is very little in the literature on which to base diagnoses. Some microbiologists think fungi are usually only secondary invaders, attacking after an infection of protozoan, bacterium, flagellate, or worm has weakened the host. Certainly there are enough records in the literature to prove that those organisms occur in the sea. It would be most peculiar if there were not many more than we know about, and if they had no influence on mortality rates of fishes, and if their virulence did not fluctuate.

People notice that an epidemic is in progress when the symptoms are startlingly obvious. They do not report it until large numbers of fish have died; then they send a few specimens to a fishery laboratory. By the time the specimens arrive, they have become so full of putrefactive organisms that it may be impossible to determine what killed them.

Let us turn to a few examples of pathogens other than fungi. There is a large group of protozoans having the form of stalked cysts, which attach themselves to certain appendages of copepods. These seem to be harmless. There is another group of protozoans, however, belonging to the Cnidosporidia, a subclass of the Sporozoa, which are not harmless. These are parasitic only on invertebrates and cold-blooded vertebrates, and often produce epidemics among commercial marine fishes. Nigrelli [11] has observed more than 700 species of Cnidosporidians in more than 1,000 species of fishes during the course of his work at the New York Aquarium (see Table 10-1). He found various species infecting the skin, connective tissue, muscle, bone, cartilage, eyes, gills, heart, nervous tissue, body cavity, gastrointestinal tract, liver, gonads, spleen, kidney, air bladder, gall bladder, and urinary bladder.

Fish become infected by eating infected fish, or by ingesting the spores of the parasites, which have been released into the water with intestinal or urinary wastes. Some of these parasites have been found infecting fish eggs.

These parasites produce all sorts of symptoms, which vary with species and according to the degree of infection. They may cause no more serious reaction than formation of connective tissue cysts; or they may cause an acute or chronic disease similar to cystitis, ne-

TABLE 10-1. SOME MYXOSPORIDIA AND MICROSPORIDIA FROM MARINE FISHES

Host	Parasite	Site
	Myxosporidia	
Alosa finta, thwait shad	*Mitraspora caudata*	Kidney
Anguilla vulgaris, eel	*Myxidium giardi*	Kidney
Atherina hepsetus, silverside	*Letotheca hepseti*	Gall bladder
Bairdiella chrysura, silver perch	*Myxoproteus cornutus*	Urinary bladder
Brevoortia tyrannus, mossbunker	*Chloromyxum clupeidae*	Muscle
Clupea harengus, herring	*Chloromyxum clupeidae*	Muscle
	Ceratomyxa sphaerulosa	Gall bladder
Clupea pilchardus, sardine	*Ceratomyxa truncata*	Gall bladder
	Sphaeromyxa balbianii	Gall bladder
Cynoscion regalis, squeteague	*Myxidium glutinosum*	Gall bladder
Gadus morrhua, common codfish	*Myxobolus aeglefini*	Cartilage, bone, cranium and eye
	Myxidium oviforme	Gall bladder
	Zschokkela hildae	Urinary bladder
Hippoglossus hippoglossus, common halibut	*Unicapsula muscularis*	Muscle
Melanogrammus aeglefinus, haddock	*Myxidium gadi*	Gall bladder
	Myxobolus aeglefini	Cartilage
	Zschokkella hildae	Urinary bladder
Merluccius merluccius, European hake	*Ceratomyxa globulifera*	Gall bladder
	Leptotheca elongata	Gall bladder
Merluccius capensis, South African stockfish	*Chloromyxum thrysites*	Muscle
Microgadus tomcod, tomcod	*Zschokkella hildae*	Urinary bladder
Molva molva, ling	*Myxobolus aeglefini*	Bone
	Leptotheca informis	Gall bladder
	Sphaeromyxa hellandi	Gall bladder
Mugil cephalus, mullet	*Myxidium incurvatum*	Gall bladder
Mugil chelo (?), mullet	*Myxobolus exiguus*	Stomach, spleen, Kidney, etc.
Oncorhynchus keta, chum salmon	*Henneguya salminicola*	Under skin
Oncorhynchus kisutch, silver salmon	*Henneguya salminicola*	Under skin
Oncorhynchus nerka, red salmon	*Chloromyxum wardi*	Gall bladder
Paralichthys albiguttus, gulf flounder	*Ceratomyxa navicularia*	Urinary bladder
	Leptotheca glomerosa	Urinary bladder
	Sinuolinea brachiophora	Urinary bladder
Paralichthys dentatus, fluke	*Ceratomyxa drepanopsettae*	Gall bladder
	Ceratomyxa navicularia	Urinary bladder
	Sinuolinea capsularis	Urinary bladder
	Leptotheca lobosa	Urinary bladder
Pleuronectes platessa, plaice	*Sphaeospora platessae*	Otic capsule
Pollachius virens, pollack	*Myxidium gadi*	Gall bladder
	Myxidium bergense	Gall bladder
Pomolobus aestivalis, summer herring	*Chloromyxum clupeidae*	Muscle
Pomolobus mediocris, hickory shad	*Chloromyxum clupeidae*	Muscle
Pomolobus pseudoharengus, alewife	*Chloromyxum clupeidae*	Muscle
Pseudopleuronectes americanus, winter flounder	*Ceratomyxa acadiensis*	Gall bladder
	Myxobolus pleuronectidae	Muscle
Scomber scombrus, mackerel	*Leptotheca parva*	Gall bladder
	Leptotheca renicola	Kidney

TABLE 10-1. SOME MYXOSPORIDIA AND MICROSPORIDIA FROM MARINE FISHES (*Continued*)

Host	Parasite	Site
	Myxosporidia	
Sebastes viviparus, redfish	*Leptotheca macrospora*	Gall bladder
Sphaeroides maculatus, common puffer	*Ceratomyxa navicularia*	Urinary bladder
...	*Sinuolinea capsularis*	Urinary bladder
...	*Zschokkela globulosa*	Urinary bladder
Tautogolabrus adspersus, cunner	*Chloromyxum clupeidae*	Muscle
Thrysites atun, snake mackerel	*Chloromyxum thrysites*	Muscle
Urophycis chuss, codling	*Ceratomyxa acadiensis*	Gall bladder
Zeus capensis, John Dory	*Chloromyxum thrysites*	Muscle
Zoarces americanus, eel-pout	*Ceratomyxa acadiensis*	Gall bladder
	Microsporidia	
Acanthocottus scorpius, European sculpin	*Plistophora typicalis*	Muscle
Callionymus lyra, dragonet	*Glugea destruens*	Muscle
Clupea pilchardus, sardine	*Glugea cordis*	Heart muscle
Lophius piscatorius, goosefish	*Nosema lophii*	Nervous system
Macrozoarces americanus, ocean pout	*Plistophora macrozoarcidis*	Muscle
Melanogrammus aeglefinus, haddock	*Nosema branchiale*	Gills
Osmerus eperlanus, Baltic smelt	*Glugea hertwigi*	Intestine
Osmerus mordax, American smelt	*Glugea hertwigi*	Intestine
Pollachius virens, pollack	*Glugea punctifera*	Eye muscle
	Plistophora spp.	Muscle
Pseudopleuronectes americanus, winter flounder	*Glugea stephani*	Intestine

SOURCE: Ross F. Nigrelli, "Cnidosporidiosis in Marine Fishes" (unpublished manuscript, 1952).

phritis, hepatitis, or enteritis such as occur in warm-blooded vertebrates. Others may induce tumors of the infected organ or surrounding tissue. Still others may cause hyalin degeneration of the tissues (see Table 10-2).

Cnidosporidia are responsible for the condition known as mushy or wormy halibut and the milky disease of Australian barracuda. The halibut fishery of the eastern Pacific has been subject to a considerable annual loss because a large percentage of the fish are so badly infected as to be unmarketable. Apparently no figures have been compiled to indicate the frequency of this disease. In Australia, 5 per cent of the barracouta (*Thrysites atun*),[12] and in South Africa,[13] 76 per cent of the John Dory (*Zeus capensis*) and 70 per cent of the stockfish (*Merluccius capensis*) taken in trawls have all been found infected with the same species of Cnidosporidian, namely, *Unicapsula thrysites*. About a quarter of the catch of John Dory has been in such bad condition that the fish were unfit for filleting. Nigrelli comments as follows on these diseases.

No determination of what role they play in the mortality of fishes under natural conditions has yet been made. There can be no doubt, however, that affected fish become susceptible to secondary invaders, such as fungi and bacteria, and that the hosts succumb more readily to changes in the physical and chemical conditions of the water, e.g., to sudden changes in temperature, pH, salinity, or to pollution.[14]

There are parasitic dinoflagellates which live in the body cavity or in the gut of copepods. Parasitologists seldom examine copepods —that is to say, not more than half a dozen have reported doing so in the last 50 years—but when they do, they find these organisms. Thus they have recorded their presence in the Clyde Sea area, in the

TABLE 10-2. CAUSES OF DEATH FROM PARASITIC AND INFECTIOUS DISEASES OF FISHES IN THE NEW YORK AQUARIUM

	1940	1941
Diseases of skin and gills		
Bacterial	8	23
Oodinium (Dinoflagellate)	11	—
Trichodina (Ciliate)	20	24
Myxosporidia (Cnidospordia)	—	3
Epibdella (Trematoda)	99	2
Microcotyle (Trematoda)	44	14
Diplectanus (Trematoda)	2	—
Argulus (Copepoda)	2	—
Livonica (Isopoda)	1	—
Diseases of skin and internal organs		
Lymphocystis	9	—
Diseases of digestive system		
Enteritis and Stenosis due to Acanthocephala	5	5
Diseases of circulatory system		
Pericarditis due to Echinostome infection	—	1
Total	201	72

SOURCE: Ross F. Nigrelli, "Causes of Diseases and Death of Fishes in Captivity," *Zoologica*, XXVIII (1943), 203–16.

North Sea, in the English Channel, in the Mediterranean, and in the Arabian Sea.[15] Specimens of 25 species of copepods in the Arabian Sea and 16 in the Mediterranean were found infested with several kinds of dinoflagellates of the genus *Blastodinium*.

Some parasitic dinoflagellates spend the first part of their life cycle in the water as free-living flagellates. When one finds a suitable host, it penetrates the body and there changes into an amoeba-like form having several nuclei. At that stage it is called a plasmodium. With *Peridinium*, one of the more pathological dinoflagellates, the plasmodium is a delicate network of protoplasm which gradually spreads among and over the organs, pushes its way

between the body muscles, and goes deep into the nervous tissue. When the parasite has filled all available space, it gathers into one or two spherical masses which penetrate the gut and leave the host through the anus. In the sea they transform to flagellate spores, and the cycle begins again. This type of parasite may completely destroy the sex glands of its host or at least upset the balance of sex hormones so profoundly as to cause hermaphroditism or sterility. Not only that, but the egress of the parasite is nearly always fatal to its host. If it does not break the body of its host into pieces, it leaves it enfeebled beyond recovery.

There are records of dinoflagellates infecting tunicates, diatoms, pteropods, siphonophores, annelids, and the eggs of copepods. One species, *Oodinium ocellatum*, lives on the skin and gills of several kinds of marine fishes, with consequent dermatitis and suffocation. This disease has been a frequent cause of death in the aquaria in London, San Francisco, and New York. In London, the disease seems to originate among specimens from Bermuda, in San Francisco, among Hawaiian fishes, and in New York, among those from Sandy Hook Bay. Many kinds of fishes are susceptible—jacks, pilot fish, bluefish, striped bass, porgy, weakfish, spot, croaker, puffer, boxfish and sea robin.[16] A similar disease, evidently also caused by *Oodinium*, affects pink salmon in Puget Sound.[17]

Bacterial diseases have been observed more often in aquaria than among feral populations of marine fishes. Tuberculosis caused by acidfast bacteria is the most fully described. It is also relatively easy to diagnose, thanks to well-established specific staining techniques. This disease causes tubercles in the spleen and liver, sometimes also in the gills, kidneys, roe, pericardium, eye, and intestine.[18]

A bacterium belonging to the genus *Gaffkya* was responsible for an epidemic disease of lobsters in the summer of 1946 along the coast of Maine and the maritime provinces of Canada. The symptoms were a diminution in number of blood corpuscles and in viscosity of the blood. From 20 to 50 per cent of the lobsters held in ponds and tanks died. In some provinces in Northumberland Strait losses approached 100 per cent.[19] The same disease appeared in the Netherlands in 1955 and again in 1957, causing numerous deaths among lobsters held in ponds.

In the early 1930's a dermatitis caused by the bacterium *Achromobacter ichthyodermis* broke out in the laboratory aquarium at Scripps Institution of Oceanography. Killifish, blennies, gobies, smelt, and other species of fishes became infected. At the same time, several specimens of killifish (*Fundulus*) sick with this dis-

ease were observed in the sea. The effect on the wild populations was not determined, but in the laboratory, mortality reached close to 100 per cent.[20]

There is a good deal of visible evidence that kelp on the Pacific coast of America is parasitized, but the diseases of these valuable plants have not been given much study. Sometimes, when water temperatures become abnormally high, extensive acreage of kelp off southern California and in the adjacent Channel Islands region is destroyed by a disease called "black rot," which is believed to result from a bacterial infection.[21]

Erling Ordal of the University of Washington has studied fish diseases at the State Department of Fisheries Station at Bowman's Bay. There he has observed several diseases of salmon in sea water. He writes of these in a letter (1957):

Some studies have been carried out on diseases of fish from sea water. Particular attention has been given to salmon in sea water at the State Department of Fisheries Stations at Bowman's Bay and Hoodsport.

One of the most striking of the diseases occurring in young salmon in sea water may be characterized as a hemorrhagic septicemia. Outbreaks of this disease have occurred many times at Bowman's Bay and Hoodsport and heavy mortalities have sometimes resulted. The pathology in affected fish is strikingly similar to that reported by Foerster in the course of a disease of unknown etiology in pilchards along the shores of Vancouver Island some years ago.[22]

Similar disease in pilchards with very heavy mortalities had been noted in American waters shortly before the Canadian outbreak occurred.[23]

More recent studies have shown it to be endemic in herring in lower Puget Sound. Outbreaks of the disease in herring with characteristic pathology occur almost every year in this area. Bacteriological studies have shown that a number of marine vibrios, which can be distinguished serologically or culturally, are responsible.

An outbreak of disease characterized by initial destruction of the eyes followed by septicemia and death occurred in salmon, cod, and bottom fish held in sea water. Again a marine vibrio was found to be the etiological agent. It is possible that this disease is identical to "Augenkrankheit" which was found in cod in the North Sea by Bergman.[24] Earlier, Bergman had shown that "red disease" of eels in the Baltic Sea was due to a marine vibrio which was given the name *Vibrio anguillarum*.[25]

Several marine myxobacteria have been isolated during outbreaks of disease in young salmon held in sea water at Bowman's Bay and at Hoodsport. One particular type, which has been isolated several times, produces a disease which resembles "columnaris disease" in fresh water. However, the etiological agent is different and is a highly fastidious, halophilic myxobacterium.

Another disease occurring in three year old silver salmon held at Bowman's Bay was characterized by muscular boils and abscesses. The disease was slow and chronic but ultimately killed the entire population. The agent in some respects resembled the diplobacillus of kidney disease, but was easily cultivated and gave entirely different symptoms on experimental infection of several lots of fish.

In addition, during the past few years we have been investigating kidney disease, primarily in chinook salmon. In the course of these investigations we have taken various lots of the fish to Bowman's Bay for study of the disease in sea water. We found that it persisted in sea water and the majority of the fish were eventually lost, though the mortality rates varied in different lots of fish. One lot introduced into sea water in January was lost by the end of August. In another experiment we found that the mortality rate was reduced when the fish were fed a full natural diet, but the disease persisted and eventually most of the fish perished. The stocks of Columbia River origin suffered a higher mortality rate than those from the Green River Station.

During the summer and fall of 1952 further studies were carried out on kidney disease of salmon in the Columbia River stocks of salmon. As part of our study we looked for evidence of it in adult salmon migrating into the river from the sea. A considerable number of salmon taken at Bonneville, Oregon showed gross lesions of the kidneys. Upon microbiological investigation we found enormous numbers of tubercle bacilli in the lesions. Many of the tubercular fish were smaller than normal fish. Some tubercular females, though normal in size, showed partially developed ovaries. The question then arose as to the source of the tuberculosis. Were the fish originally infected before going to sea, were they infected in the marine environment, or did the infection occur after they again returned from the sea? Upon examination of salmon taken in the commercial fishery near Astoria, it was found that considerable numbers of fish were tubercular. This finding coupled with the disparity in size of some of the tubercular fish makes it unlikely that the fish were infected on return from the sea. Much more work needs to be done before it can be established with certainty that the disease was contracted in the sea, during the initial period in fresh water, or in both habitats.

Pathologists suspect that viruses are very destructive to freshwater fishes. At least one, the lymphocystis disease, occurs in the sea. This is characterized by the fact that connective tissue cells in the victim grow to gigantic size and become surrounded by a thick transparent membrane. The disease has been recorded among flat fishes in Europe, perch in the Baltic Sea, *Sargus* in the Mediterranean, striped bass in Connecticut, New York, and New Jersey, angelfish and hogfish from Key West, Florida, and orange filefish from Atlantic City.[26]

Worms—flatworms, roundworms, and leeches—as well as crustaceans, parasitize all kinds of marine animals. These have not been studied much, but being quite visible to the unaided eye have been more frequently noticed, named, and recorded in scientific literature than have the microscopic pathogens. In general the metazoan parasites seem not to destroy their hosts. They reduce their vitality, they sterilize them, they emaciate them, and they make them susceptible to other diseases. But there are no accounts of mass mortalities attributed to them.

I will end this collection of evidence bearing on the importance of diseases and parasites in marine ecology by telling of the natural

history of one copepod parasite to illustrate how it links the life conditions of two very diverse groups of fishes:

This copepod, *Lernaeocera branchialis,* begins life as an independent, free-swimming member of a plankton community. It moults several times in the course of growth. When it reaches a point of development at which it must take up its parasitic life, it seeks a flounder, and if chance favors its coming upon one, it goes into the mouth and attaches to the inner wall of the gill cover. Hundreds of young copepods may live thus in a single fish, doing what damage no one knows. Presumably they inhibit its respiration to some degree. In any case, in the flounder they mature and fructify, the male dies, and the fertilized female grows in size about threefold. Then it leaves the flounder, lives a free-swimming existence for a short period while seeking another fish, this time a member of the cod family. There, as in the flounder, it takes up residence in the gill cavity. But whereas there had been hundreds of infant *Lernaeocera* in the first host, there are never more than three, and usually only one, in the second. Now an extraordinary change takes place. The female copepod, which so far has looked enough like a crustacean to be easily recognized as such, develops into a grotesque object, a mass of growing tissue bearing no resemblance to its free-living relatives. Leaving its posterior part exposed in the gill cavity, the parasite eats its way and grows through the flesh of its host towards the heart, sometimes actually reaching the pericardium. This activity stimulates the formation of tumors in the host tissue that probably impede circulation and may contribute to shortening the life of the host. Even after the host dies, the female copepod continues to live until the eggs hatch. Then the cycle of generation of the parasite begins again. In Cuxhaven in 1949 and 1950, the percentage of infected fish among samples of fishes examined were: haddock, 10 per cent; whiting, 80 per cent; and cod, 20 per cent. Infected fish were below normal in weight, whiting by as much as 23 per cent, cod and haddock by 5 to 15 per cent.

Now to attempt some conclusions:

Marine animals and plants, at all stages of life from egg to adult, are hosts to a large assortment of disease organisms, which vary in their effects from mild irritation to deadly virulence. The resulting diseases often occur in epidemics which may be limited to particular localities or may travel around the world. Some disease agents are severely fastidious, held by their physiological constitution to living on a single species; others have a much greater nutritional tolerance. Diseases in the sea come and go as mysteriously as do those on

land. Apparently it is not merely fertility of the water or population density that controls them, for there are examples of epidemics in poor as well as in rich areas, and among stocks of fishes at low levels of abundance as well as among those at high levels. Apparently epidemics occur in cycles of rather long period, on which are superimposed irregular fluctuations and seasonal cycles. Very likely they, too, are subject to diseases which may occur in cycles. Thus, epidemics seem to be controlled partly by climate, but much more by something else. That something else may be a complex of fluctuating influences, including fluctuating virulence in the pathogens, or fluctuating resistance in the host species. What mechanisms control these influences is a mystery.

A very large proportion of marine biological research programs is devoted to measuring abundance of fishery stocks and determining how this is affected by rates of mortality and replacement. "Mortality" is the resultant of death from man's fishing and death from natural causes. "Replacement" refers to restoration of a fished stock by growth of survivors and by addition of new generations. The interrelation among all these quantities should be, and sometimes is, one of the most important considerations in designing regulations for conservation purposes. Unfortunately, mortalities and replacements are so very elusive and difficult to measure that fishery administrators often cannot wait for the determinations. Behind fishery administrators' policies is often the unconscious assumption that natural mortality at the commercially useful ages is always negligible compared to fishing mortality.

Behind fishery research programs is this line of reasoning: Natural mortality is highest during the infancy of a generation; it probably reaches its peak soon after the larvae have absorbed their yolk and must begin to find their food supply in the water. It then declines rapidly to a relatively low level where it remains stable for the rest of the life of the generation. As a consequence of fluctuating infant mortality, the number of survivors in broods of fish varies from year to year, and this greatly affects abundance and therefore the fortunes of fishermen. It would be valuable both to administrators and to fishery industrialists to predict the size of generations as far as possible in advance of their entrance into fisheries.

The commonest assumption about infant mortality is that it results chiefly from unfavorable hydrographic conditions which are lethal to the brood or to its food organisms. Biologists therefore attempt to associate changes in various features of the environment with changes in distribution and size of broods of fish, and with the density of plankton. They sample the water and its contents as

thoroughly as the speed of their ships and their equipment permit, measuring such qualities of the sea as are likely to prove useful—temperatures, salinity, oxygen content, phosphates and nitrates, and turbidity. This has been going on for years in many parts of the world with few, if any, persistent correlations so far. Could it be that they are examining only a part of the environment and overlooking one of its most important features?

If diseases did weigh heavily among causes of infant mortality, fishery biologists would hardly ever know it, because they do not usually search for diseases of eggs and larvae. They preserve their plankton samples in formalin sometimes for months before they examine them to identify and tabulate their contents. It would be close to impossible to detect bacterial or virus infection in such material! Nor can any very useful progress be made toward exploring this field until scientists are attached to marine laboratories for the particular purpose of conducting continuous research into disease-producing organisms and their effects on the marine world. This is not a job for the jack-of-all-trades. A laboratory interested in establishing a pathology unit should not be content with less than a team consisting of a bacteriologist, a mycologist, a parasitologist, a virologist, a pathologist, and a biochemist.