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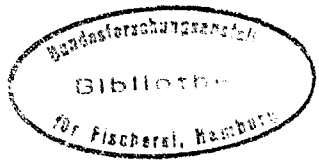

THE FUNGAL PATHOGEN ICHTHYOPHONUS HOFERI IN SEA HERRING, CLUPEA HARENGUS: A PERSPECTIVE FROM THE WESTERN NORTH ATLANTIC

by

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ABSTRACT

Ichthyophonus hoferi, probably the most ubiquitous fungal pathogen of marine fish, has had a particularly devastating effect on herring (Clupea harengus) of the western North Atlantic during the past century. Epizootics, often accompanied by mass mortalities, have occurred repeatedly in herring populations of the Gulf of Saint Lawrence and the Gulf of Maine, beginning in 1898. This summary paper reviews the history of those disease outbreaks, with the specific objective of providing insights about the factors responsible for epizootics, and the effects of disease on abundance of herring.

It seems clear, from the sporadic scientific data available, that outbreaks of the disease can reduce population abundance (as reflected in landing statistics). It seems equally clear that the elements contributing to disease outbreaks are complex and have thus far defied full interpretation, even though some relevant field and experimental data have been assembled and analyzed.

CONTENTS

1. INTRODUCTION

The pathogenicity of the fungal organism presently labeled Ichthyophonus hoferi (Plehn and Mulsow, 1911; Sprague, 1965) has been recognized for exactly 100 years (Hofer, 1893) --first as a menace to wild salmonids in western Europe, then as a danger to fresh and saltwater aquarium fish, then as an introduced but controllable problem in trout hatcheries of the western United States and Australia, then as a potential threat in marine aquaculture, and sporadically during all this time as a persistent and often lethal invader of many other species of marine and freshwater fish (more than 80 species, by one count made several decades ago by Schäperclaus, 1953).

Most recently -- within the past two decades -- Ichthyophonus has been reported at epizootic levels in several demersal species: yellowtail flounder, Limanda ferruginea off Nova Scotia, and plaice, Pleuronectes platessa, and haddock, Melanogrammus aeglefini, off the north coast of Scotland. Earlier, epizootics occurred in pelagic species: the sea herring, Clupea harengus, of the western North Atlantic, and the mackerel, Scomber scombrus, from the English coast.

Additionally, the fungal pathogen, long known in freshwater hatcheries and rearing operations, has emerged recently as a significant problem in marine aquaculture. It was first recognized in cultured marine fish in Japan in 1967 (Kubota, 1967) and is now known as a chronic and usually fatal disease of yellowtail, Seriola quinqueradiata, rainbow trout, Oncorhynchus mykiss, ayu, Plecoglossus altivelis, and other species (Miyazaki and Kubota, 1977a-c; Chien et al., 1979a-d; Miyazaki and Jo, 1985). The disease in yellowtail was recently described by Egusa (1983) as affecting fish primarily during the first year of culture. The disease was thought to be transmitted by feeding Ichthyophonus-infected smaller wild-caught species to the cultured fish. Other cultivated marine fish have been infected by Ichthyophonus, including the sea bream, Sparus aurata, and rainbow trout, Oncorhynchus mykiss, from Greece (Athanassopoulou, 1992).

But this paper has a more specific objective -- it is not to review the impressive global impacts of Ichthyophonus in every aquatic environment, but rather to summarize the scientific information about the history of the pathogen in herring stocks of the western North Atlantic. The published literature is fragmentary, but it contains some important insights about the epizootic phase of Ichthyophonus disease in sea herring. As such, it constitutes a possibly useful but admittedly fragile base for new analyses of ongoing disease-related events in European herring stocks.

2. CHARACTERISTICS OF THE DISEASE IN HERRING AND OTHER SUSCEPTIBLE SPECIES

The disease in herring caused by Ichthyophonus hoferi is systemic, with foci of infection in the heart, viscera, and lateral somatic muscles (Figs. 1-4). A simple sequence of life history stages of the organism, described from herring (Daniel, 1933a; Fish, 1934; Sindermann and Scattergood, 1954), involves multiple germination of heavy walled spores, hyphal invasion of host tissues, formation of conidia-like "hyphal bodies," and sequential germination of these entities. More complex life cycles have been proposed for the parasite as it occurs in other species (Sproston, 1944; Chien et al., 1979b; Okamoto et al., 1985.

The disease in herring has been reported in chronic and acute phases. Acute infections were characterized by massive tissue invasion, necrosis, and death within 30 days. Chronic infections exhibited cell infiltration, progressive connective tissue encapsulation of spores, and accumulation of melanophores (Fig. 5). The disease was rarely arrested completely, however, and deaths occurred in most fish within six months. During the most recent epizootic, infections averaged about 25% of all herring sampled, and many cases were acute (Fig. 5). Enzootic prevalences have been found in several studies to be usually well under 1%, and infections during this phase have been chronic in all cases examined. A number of potential reservoir hosts exist in the western North Atlantic (redfish, yellowtail flounder, winter flounder, alewife, cod, and mackerel).

The pathogen produces systemic infections best recognized by formation of widely disseminated granulomatous lesions containing a variety of life cycle stages (Neish and Hughes, 1980). This so-called "chronic" phase of the disease is progressive, and is found in individuals and species only marginally susceptible to infection. The "acute" phase, found during epizootics in susceptible individuals, is too often deemphasized; it is characterized by rapidly proliferating pathogens with extensive tissue necrosis but minimal host inflammatory response.

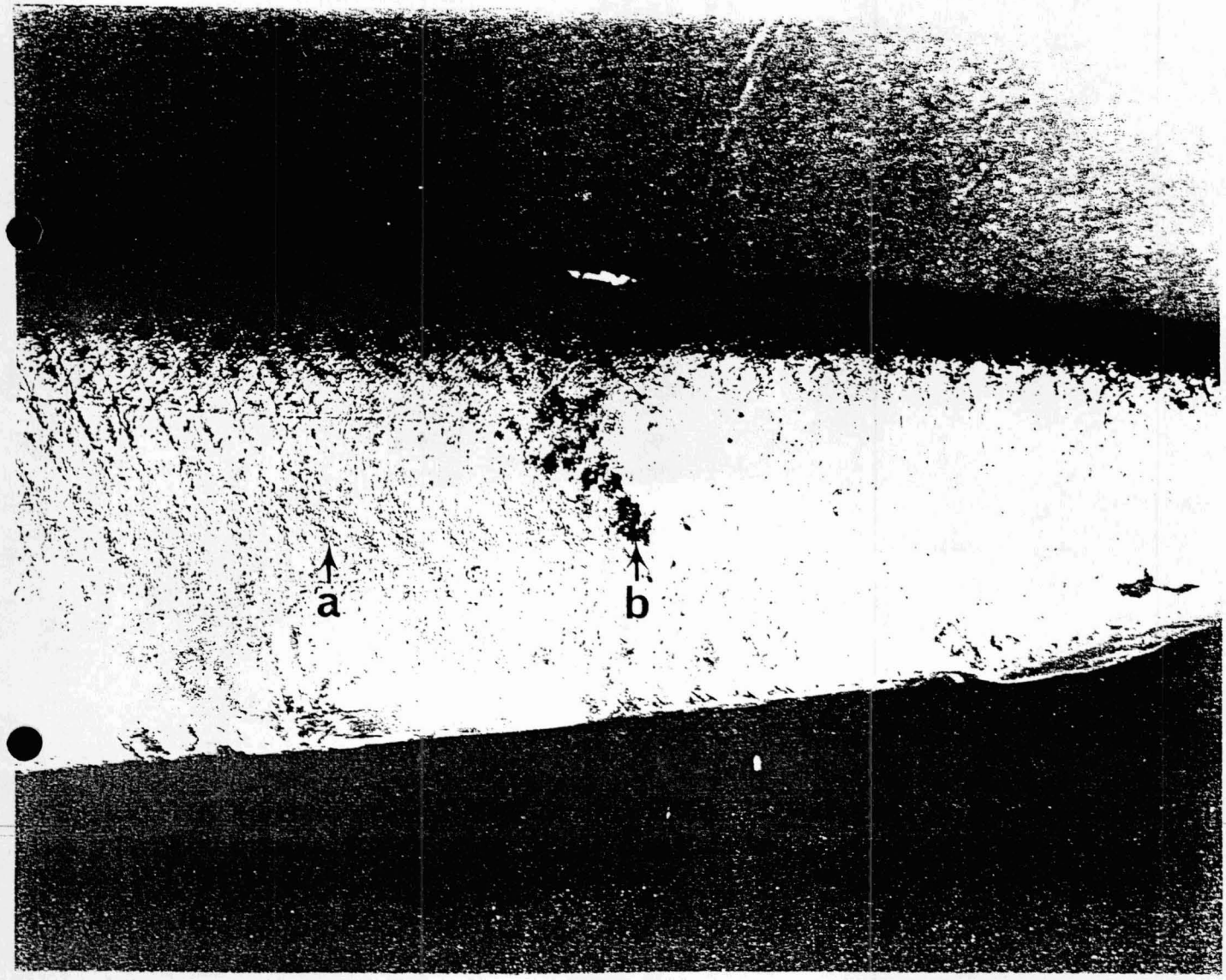

Fig. 1. External signs of Ichthyophonus hoferi infection in Atlantic herring: (a) "sandpaper effect" and (b) ulceration.

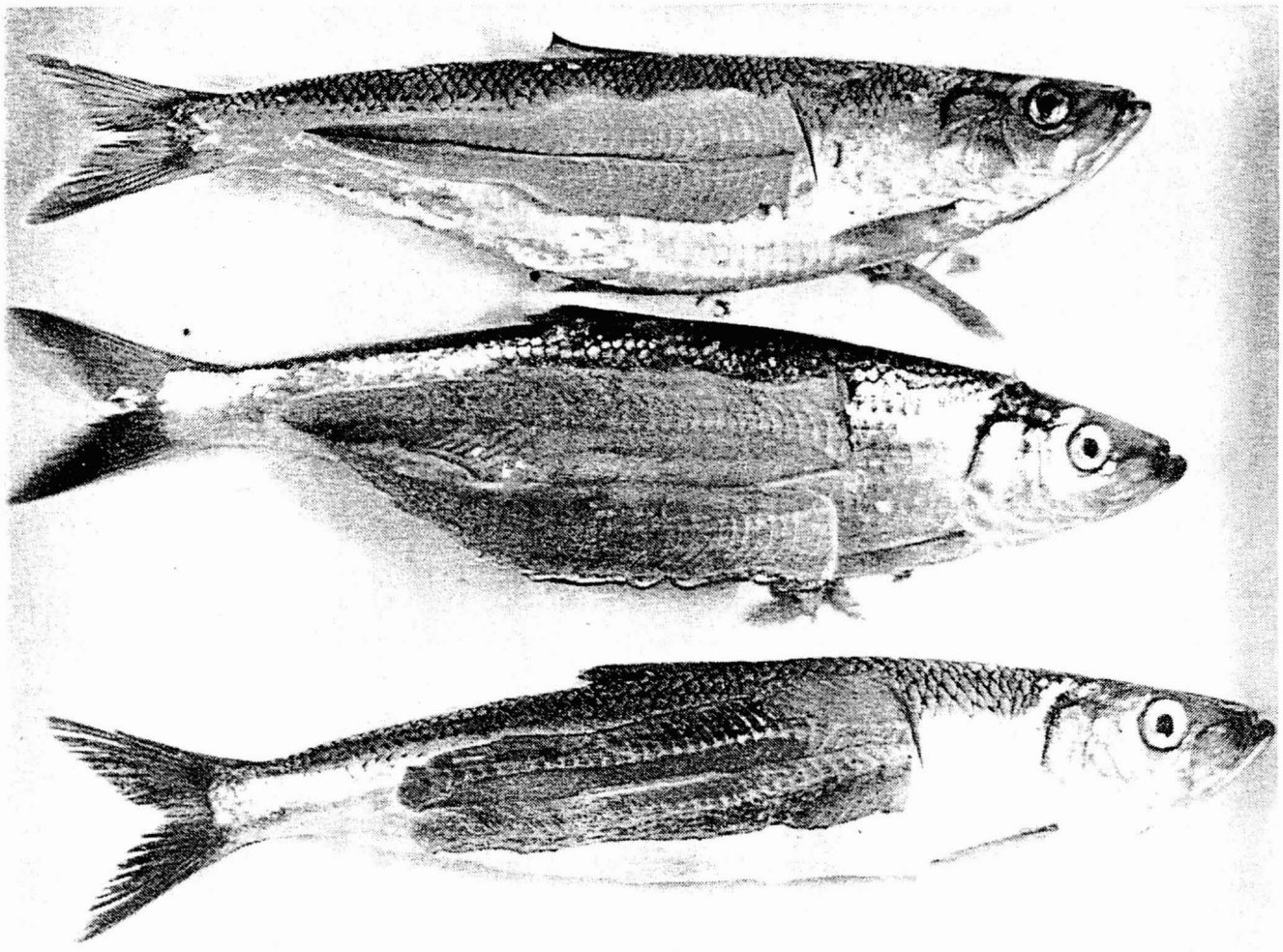

Fig. 2. Effects of Ichthyophonus hoferi infection on body muscles of Atlantic herring: uninfected (top), acute infection with extensive necrosis (center), and chronic infection with pigment deposition around fungus nodules (bottom).

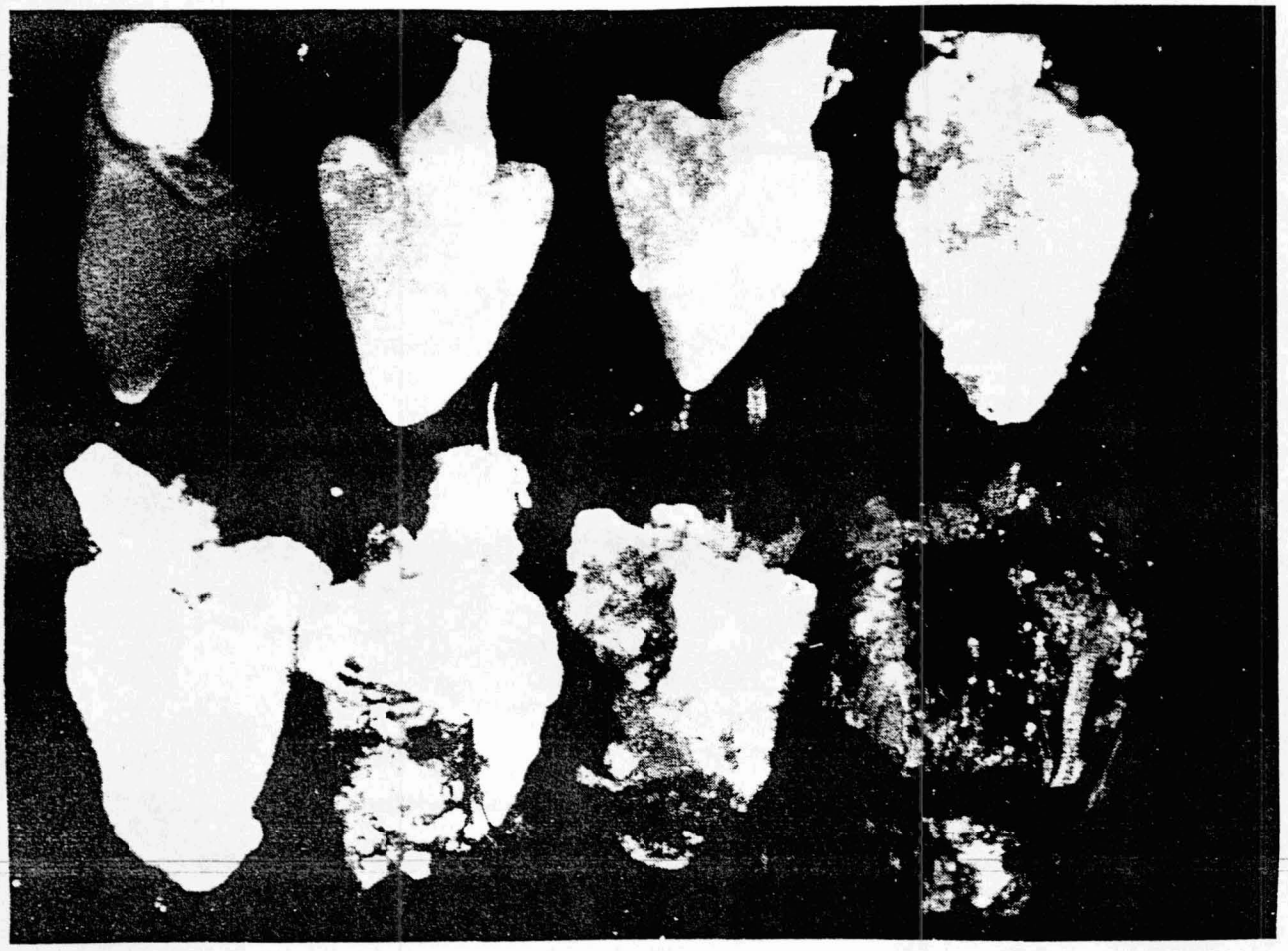

Fig. 3. Hearts of normal (upper left) and fungus-infected Atlantic herring, with increasing extent of invasion.

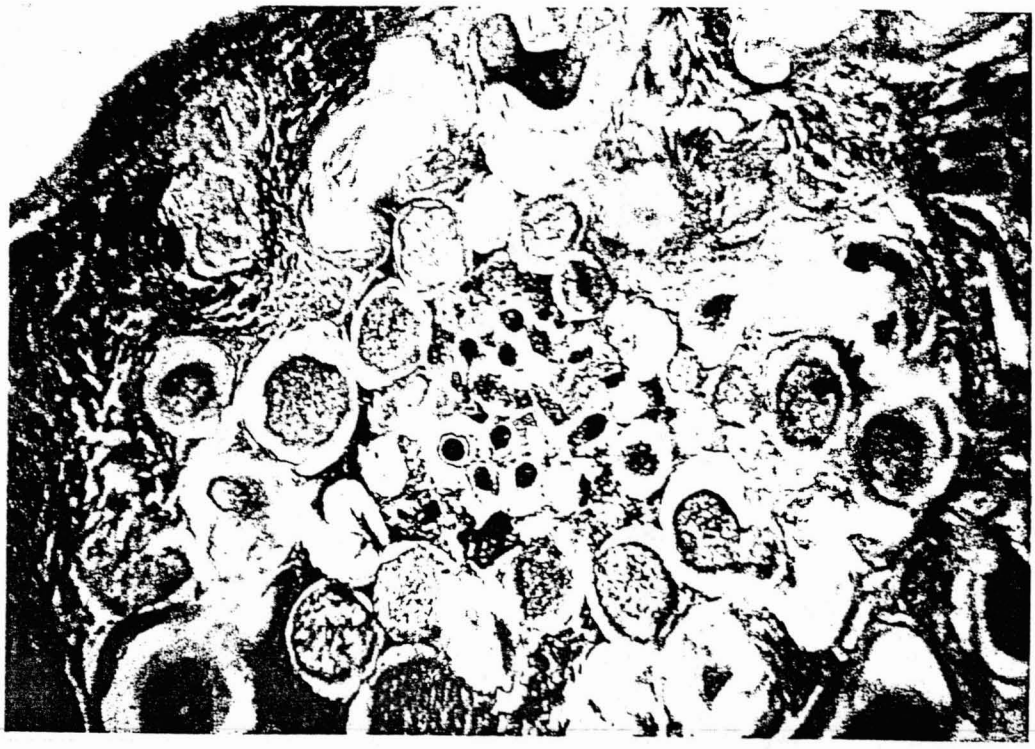

Fig. 4. Hyphal development of Ichthyophonus hoferi in the heart of an Atlantic herring. Note extent of tissue disruption and replacement by fungal life stages.

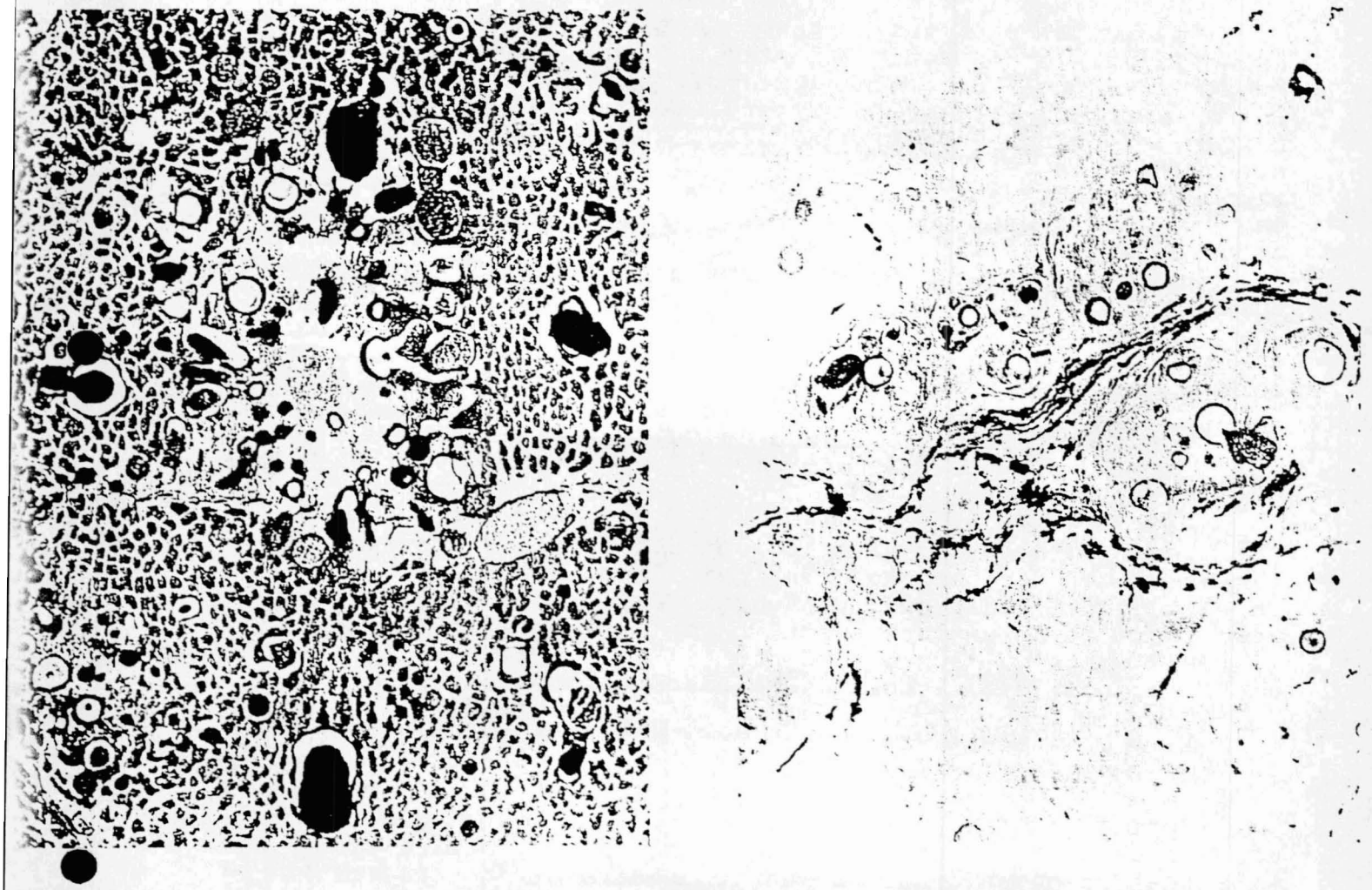

Fig. 5. Histopathology of Ichthyophonus infection in herring -- acute infection (left) and chronic infection (right). The lefthand figure is from a section of lateral musculature of herring dying at Grande Rivière, P.Q. Note spherical and germinating fungus spores in center, necrosis, and lack of tissue response. X70. The righthand figure is from a section of lateral musculature of herring taken in gill net catch, Bay of Chaleur, 1955. Note extensive connective tissue encapsulation, pigment deposition, and degenerating spores, in most cases with only the spore wall remaining. X70.

3. EPIZOOTICS IN HERRING STOCKS OF THE WESTERN NORTH ATLANTIC

The fungal pathogen Ichthyophonus hoferi appears to be enzootic in western North Atlantic herring, and is characterized by periodic outbreaks in the Gulf of Maine and the Gulf of Saint Lawrence. It was first reported from Gulf of Saint Lawrence herring by Cox (1916) after an epizootic and extensive mortalities in 1913-14. According to that investigator, a similar outbreak had been observed by fishermen in 1898, but had not been investigated further. The next reported outbreak occurred in 1930-31 in the Gulf of Maine (Daniel, 1933a, b; Fish, 1934) among immature herring. Another outbreak in the Gulf of Maine occurred in 1946-47 (Scattergood, 1948), also among immature herring. Both of the Gulf of Maine epizootics were characterized by widespread fungal infections but no reported mass mortalities such as those which occurred in the Gulf of Saint Lawrence in 1913-14. Then in 1954-55, another epizootic with major mortalities occurred in Gulf of Saint Lawrence herring (Leim, 1955, 1956; Sindermann, 1956, 1958, 1963, 1965; Tibbo and Graham, 1963).

Thus, to keep our distinctions clear, four outbreaks of the disease have been described in the scientific literature -- two in each Gulf. Additionally, hearsay evidence exists for the occurrence of an earlier event in the Gulf of Saint Lawrence (in 1898) that was similar to the 1913-14 epizootic. Information about each episode has been summarized in the following sections.

A. Disease-related mass mortalities of herring in the Gulf of Saint Lawrence

Spring and fall spawning populations of herring occur in the Gulf of Saint Lawrence, but large schools of immature fish -- the kind that so often occur in inshore waters of the Gulf of Maine -- are rarely seen. The fishery, therefore, concentrates on spawning aggregations. Two disease outbreaks, 1913-14 and 1954-55, both accompanied by mass mortalities, have taken place in those spawning stocks and have been described in the scientific literature; an earlier widespread mortality, in 1898, had characteristics similar to those seen in the later outbreaks.

1. Mortalities in 1898

Cox (1916) in his report on mortalities that occurred in 1913-14, referred briefly to herring mortalities in the Gulf of Saint Lawrence in 1898 -- based on recollections of fishermen. His entire statement about the earlier event is:

> "Fishermen recalled the fact, too, that sixteen years before [in 1898] a similar run of diseased fish had visited the coast, and as schools of young herring are very unusual in those waters, it was suggested that the epidemic may be the determining cause of the movement."

No scientific study was conducted; no pathogen was identified; but some characteristics of the episode -- especially a large and abnormal run of diseased small herring -- were similar to those seen later (in 1913-14) in the same waters, and were then associated with a specific pathogen. Because of those characteristics, and the apparently widespread nature of the mortalities, that early event is considered here as a likely disease outbreak -- as it was by Cox (op. cit.).[1]

2. Mortalities in 1913-14

Cox (1916) summarized results of an investigation of herring mortalities in 1913-14 -- mortalities which involved a large and abnormal run of small fish (15-20 cm) and spring spawning adults as well. The pathogen was described by Cox as "microsporidan-like" but it had morphological features similar to an organism described in other fish species as Ichthyophonus hoferi (Plehn and Mulsow, 1911; Neresheimer and Clodi, 1914; Ellis, 1928). Cox' diagrams of the organism bear remarkable resemblance to stages of Ichthyophonus seen by later investigators in herring and other species.

3. Mortalities in 1954-55

The 1954-55 herring mortalities in the Gulf of Saint Lawrence were examined intermittently by scientists from the Fisheries Research Board of Canada, the U.S. Fish and Wildlife Service (at the request of Canadian counterparts), and the Quebec Department of Fisheries (at its Grande Rivière Marine Biological Station). Numerous reports are available (Templeman, 1954; Leim, 1955, 1956; Sindermann, 1956, 1958, 1963, 1965; Ronald, 1960; and Tibbo and Graham, 1963).

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[1]During the field studies of herring mortalities in the Gulf of Saint Lawrence in 1954-55, interviews with fishermen and sporadic perusal of newspaper archives in a few of the larger towns bordering the southern Gulf were made. Several brief accounts of unexplained small-scale herring deaths were recorded for the period of the early 1940s. Associations with disease were not made, however, and in retrospect such localized reports may have resulted from observation of dumped seine catches (which were not unusual, then or now), or stranding of schools of immature herring being driven ashore by predators. Because of the anecdotal nature of the information, the limited areas involved, and the absence of any scientific study, these reports are not included in this summary paper as evidence for a fungal disease outbreak, although it may be tempting to do so, and although they were so included in an early report by an overeager investigator (Sindermann, 1963).

Widespread herring mortalities, particularly of mature spring-spawning fish, were first reported from the Gulf of Saint Lawrence in late spring and early summer of 1954 (Tibbo, unpublished data)[2]. Dead fish were observed floating in shoals at the surface, were washed up on the shores of the Gulf, and were taken in nets of otter trawlers. Mortalities extended from the west coast of Newfoundland to the Gaspé coast, and from Anticosti Island to Northumberland Strait. Deaths were first apparent in mid-May, reached a peak in June, continued at a reduced rate through July, and apparently ceased in August. Abnormal, lethargic, moribund herring could be seen inshore, particularly near breakwaters, throughout the period. Deaths occurred sporadically in such inshore aggregations, the dying fish sinking to the bottom to become part of an extensive litter of herring carcasses. This pattern was repeated in 1955 as far as numbers of fish, abnormal behavior, and time were concerned. Mortalities in 1956 were drastically lower, being confined to the Chaleur Bay region and the Magdalen Islands, and to relatively few fish as compared to 1954 and 1955. No dead herring were found in 1957.

Wherever examined, dead fish were found to be infected with the fungus Ichthyophonus hoferi. This pathogen causes systemic infection, with foci in the heart, liver, and lateral line musculature. The disease affects fish of all ages and is probably acquired in inshore areas. Death of diseased fish seems due to circulatory failure in most cases, although massive involvement of organs other than the heart may be contributory. From examination of the course of the disease in 1955 and 1956, and from experimental work conducted during that period, it appears that the initial and extensive mortalities were caused by an acute phase of the disease, characterized by rapid massive fungal invasion of susceptible fish. Experimentally, this type of infection was terminal within one month from time of exposure. Acute infections, in addition to killing many fish, caused degeneration and necrosis of the body muscles, particularly the lateral line musculature. Such diseased fish were poor for smoking, because they fell from smokehouse racks, and were not suitable for pickling, since they were already partly decomposed. Later sporadic deaths and infections apparent on gross examination were due principally to a subacute or chronic phase, characterized by marked host cellular response, in fish apparently more resistant to the disease organism. Abundant infections, principally of this type, were characteristic of late summer and autumn, after mass mortalities had apparently ceased. These infections were often accompanied by pigment deposition around fungal spores in the body muscles, creating an additional problem for herring picklers.

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[2]Typewritten report by S. N. Tibbo dated August 16, 1954, to the Director of the Biological Station, St. Andrews, N.B. A copy of this report was made available to the senior author by Mr. Tibbo.

Sampling in 1955 and 1956 disclosed a low disease prevalence in the earliest (late April) run of herring, indicating that few heavily infected fish survived from the previous year. Coincident with first reports of mortalities, a rapid rise in prevalence, especially of acute infections, occurred in late May, one month after the herring appeared inshore.

From observations made in 1955, it was estimated conservatively that at least one-fourth of the mature herring of the western Gulf were infected in that year (Sindermann, 1956); this was later supported by the fact that herring landings for the entire Gulf in 1956 decreased one-third from the 1955 landings. There was no evidence that fishing effort had decreased during 1956. Mortalities in some Gulf areas in 1954 were at least as severe as in 1955, suggesting significant population decimation as a result of this outbreak; this was supported by the fact that Chaleur Bay landings, compared to the average for the previous 10 years, were in 1955 only 74%, and in 1956 only 62%, and were lower than any other single year in the previous ten.

Since mortalities in 1956 were greatly reduced, that year may be considered the first post-epizootic year. Disease prevalences in the Gulf, except at the Magdalen Islands, were lower in 1956 than in 1955, and were much lower in 1957 than in 1956. Prevalences averaged 27% in 1955, 22% in 1956, and 10% in 1957. Fungal prevalences in herring sampled in the spring of these years are graphed in Figure 6. Although not included in the graphs, it should be noted that early autumn prevalences in the Gulf of Saint Lawrence were as high or higher than spring-early summer figures. For example, two September samples from Chaleur Bay in 1957 had prevalences of 24 and 30%, much higher than the spring-early summer average of 6%. Also, one sample of trawled herring from the Magdalen Islands in 1957 had a prevalence of 78%, possibly representing a differential deeper water aggregation of infected fish.

B. Epizootics caused by Ichthyophonus hoferi in herring of the Gulf of Maine

Unlike the fishery in the Gulf of Saint Lawrence, which concentrates on adult stocks, herring of the Gulf of Maine are commercially important principally during their second and to a lesser extent their third years of life, when they are processed as sardines. Some adult fish are taken, principally near offshore islands, and mostly in the late summer and autumn months (Sindermann, 1979). Two disease outbreaks have been described in the scientific literature -- in 1930-31 and 1946-47 -- in coastal juvenile stocks. High disease prevalences caused disruption of the fishery because of legal restrictions against processing infected fish, but mass mortalities were not reported.

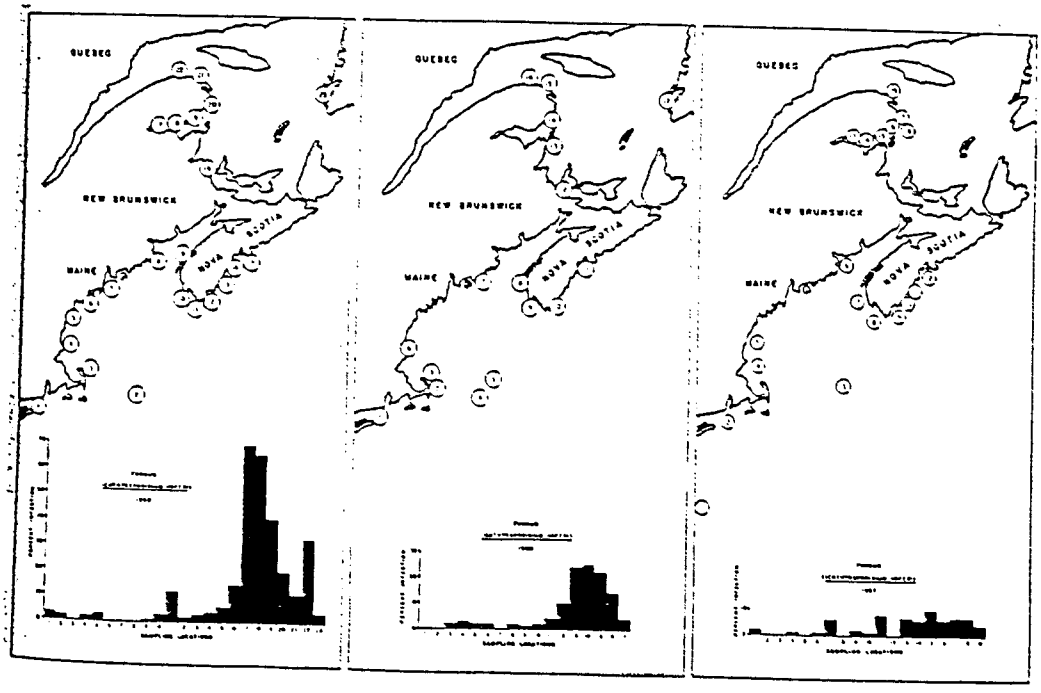

Fig. 6. Fungal prevalences in samples of mature herring of the western North Atlantic, roughly comparable in location and time for 1955, 1956, and 1957.

1. The epizootic of 1930-31

Two investigators examined and reported on the 1930-31 outbreak: Daniel (1933a, b) described the gross and microscopic appearance of the disease, and proposed a life cycle for the pathogen, based on histological observations. He pointed out the systemic nature of the infection, with massive invasion of the heart and liver -- suggesting a high mortality among infected fish.

The outbreak of the fungal disease in 1930-31 was also investigated in the summer and early autumn of 1931 by the U.S. Bureau of Fisheries and the State of Maine, and a comprehensive report was published by Fish (1934). Epizootiological and pathological aspects of the disease were emphasized. Average prevalence in all age groups in winter and spring of 1931 was "about 70%" but fell to 18% by October. Decrease in prevalence was attributed to dilution of heavily infected inshore stocks by a less heavily infected migrating offshore group. Fish further reported that "the unusual scarcity of sea herring in the Gulf of Maine during the past summer [1932] indicates that a large number of fish, infected during the past few years, succumbed to the disease." The investigation also revealed fungal infections in alewives and winter flounders; and flounders were infected experimentally by force feeding with infected tissues from herring. The histo-pathology of infections was described in great detail, with emphasis on the systemic nature of infections and the sequence of connective tissue deposition around the parasites -- but with failure of those defenses to halt the spread of infection. Formation of grossly visible necrotic lesions was the final step in the progression.

2. The epizootic of 1946-47

The 1946-47 outbreak of Ichthyophonus hoferi, the most recent one to occur in herring of the Gulf of Maine, was examined by a succession of biologists employed by the U.S. Fish and Wildlife Service -- but principally by Scattergood, who published a detailed report in 1948, and a follow-up report in 1954. His first paper described the severe economic losses in the sardine industry in 1947 caused by rejection of processed infected fish, and the seeming intractability of the problem (fungal spores of any kind are considered as contaminants by USFDA inspectors). His extensive sampling program, in United States and Canadian waters of the Gulf of Maine, provided information on seasonal and geographic variability in prevalences in 1947 and 1948. He concluded that the highest prevalences were found during winter and spring months, among static populations in bays and estuaries, where transmission of the pathogen was most favored.

4. EXPERIMENTAL INFECTIONS AND EXPERIMENTAL EPIZOOTICS IN CAPTIVE POPULATIONS OF HERRING

The outbreak period in the Gulf of Saint Lawrence in 1954-55 was a time of very low disease levels in Gulf of Maine herring. This offered an admirable opportunity to examine the disease in contrasting phases, and to supplement descriptive studies with experimental work using a susceptible population. Immature Gulf of Maine herring, maintained in the seawater tanks of the Boothbay Harbor Fisheries Laboratory, were used in all experiments.

Knowledge about the effects of increasing infection pressure on occurrence of the fungus disease in susceptible hosts was obtained by experimental exposures and experimental epizootics created in aquarium populations of immature herring. Massive and repeated exposures were necessary to achieve infection in two-year-old fish (Table 1). Beyond a critical dosage level, acute as well as subacute or chronic infections resulted.

With this experimental demonstration of the effect of increasing spore dosage or infection pressure on prevalence and severity of the disease, a large-scale experimental epizootic was created in a laboratory population of 2,000 one-year-old herring. Spore dosage of 2×10^5 spores on each of four consecutive days resulted in infection of 23% of the population -- 8% acute and 15% subacute. The disease was terminal in all acute cases within 30 days. Chronic or subacute infections resulted in death of all but very light cases within six months. As with most aquarium studies of this kind, occasional deaths due to causes other than fungus disease occurred in experimental and control groups during the observation period, but these were infrequent enough so that they would not materially alter the findings. At the termination of the experiment, all surviving individuals were examined; only three lightly infected fish were found.

These experimental results agree with findings during the last outbreak in Gulf of Saint Lawrence herring (Sindermann, 1958). Twenty-seven percent of all fish sampled in that Gulf during the epizootic peak were infected, the ratio of acute to subacute being approximately one to two. Disease prevalences were very low when spawning fish first appeared on the coast in late April, but mortalities due to acute infections began about one month after inshore migration. Deaths continued at a low level throughout the summer, due to subacute infections.

5. EFFECTS OF EPIZOOTICS ON ABUNDANCE OF HERRING STOCKS

It seems that some of the best evidence for an association of disease-caused mass mortalities and changes in abundance of marine fish can be found lurking in the information on herring fungus epizootics. Cox (1916), reporting on the 1913-14 Gulf of Saint Lawrence outbreak, made this statement: "The herring fishery was reduced for several years following the outbreak." Similarly, herring landings in the Gulf of Saint Lawrence were reduced sharply following the most recent outbreak (1954-55). Landings declined to slightly over half their previous level in the years immediately following the epizootic (Sindermann, 1958) (Fig. 7). Outbreaks of the disease in Gulf of Maine herring in 1930-31 and in 1946-47 were not accompanied by observed mortalities, and the fishery did not seem to be immediately affected. However, this fishery concentrates on herring of the 0 and 1 age groups, which were heavily infected, and it is interesting and perhaps significant that in 1950-51, when these fish would have contributed to the spawning stocks, there appeared to be a pronounced reduction of the inshore mature herring population of the Gulf of Maine. It should be noted, however, that little is known of offshore spawning populations (except for the Georges Bank stock), so that this apparent reduction might be due to a shifting of spawning grounds. While other interpretations are possible, these limited data, plus observations of the magnitude of mortalities in 1954 and 1955, tend to support the hypothesis that the fungus disease is an important factor in shaping fluctuations in abundance of herring of the western North Atlantic. Mature as well as immature fish are affected; a significant part of the population is involved during outbreaks; and most of the fish that become infected are killed. It is also interesting and perhaps pertinent that the two most recent outbreaks (the only ones for which we have adequate fishery data) occurred at times of herring abundance, as indicated by landing statistics and general observations.

Results of a major effort to quantify the effects of the 1954-55 epizootic on Gulf of Saint Lawrence herring stocks were reported by Tibbo and Graham (1963). The authors conducted intensive surveys during the post-epizootic years, especially in 1960 and 1961, and made comparisons with pre-epizootic data from 1947 and 1948, concentrating on Chaleur Bay in the western Gulf. Some of their most important findings were these:

(1) "For the 5-year period (1950-54) before the heavy mortalities of herring occurred, average annual landings in Chaleur Bay were 23.9 million lb (10,841 metric tons). For the 5-year period (1955-59) immediately following the start of heavy mortalities average annual landings were 14.7 million lb (6668 metric tons), a decrease of nearly 40%" [italics mine].

Table 1. Spore dosage and experimental infection rate in two-year-old herring. (Experimental groups of 50 fish each, from a laboratory population found previously to be free of fungus disease, were maintained in 250 gallon seawater tanks. Spores were obtained from naturally infected fish and tested for viability by culturing in Sabouraud-serum agar. Spore suspensions were added to food just before the fish were fed. The experiment was terminated at 90 days, and surviving fish examined for gross or histological evidence of disease.)

Exposure and dosage schedule	Observations
2×10^5 spores in single exposure	No gross or histological evidence of disease after 90 days
2×10^5 spores on each of 3 consecutive days	After 90 days, 4 of 50 fish had subacute or chronic infections; spores few and encapsulated; other fish uninfected
2×10^5 spores on each of 5 consecutive days	After 20 days, 1 fish dead with massive acute infection; extensive tissue necrosis; little host response. After 90 days, 10 of remaining 49 fish exhibited subacute or chronic infections of varying severity; 39 fish uninfected
2×10^5 spores on each of 7 consecutive days	After 15 to 30 days, 5 fish dead with massive acute infections. After 90 days, 12 with subacute infections of varying severity; 33 fish uninfected
Control (no spore exposure)	No gross or histological evidence of disease

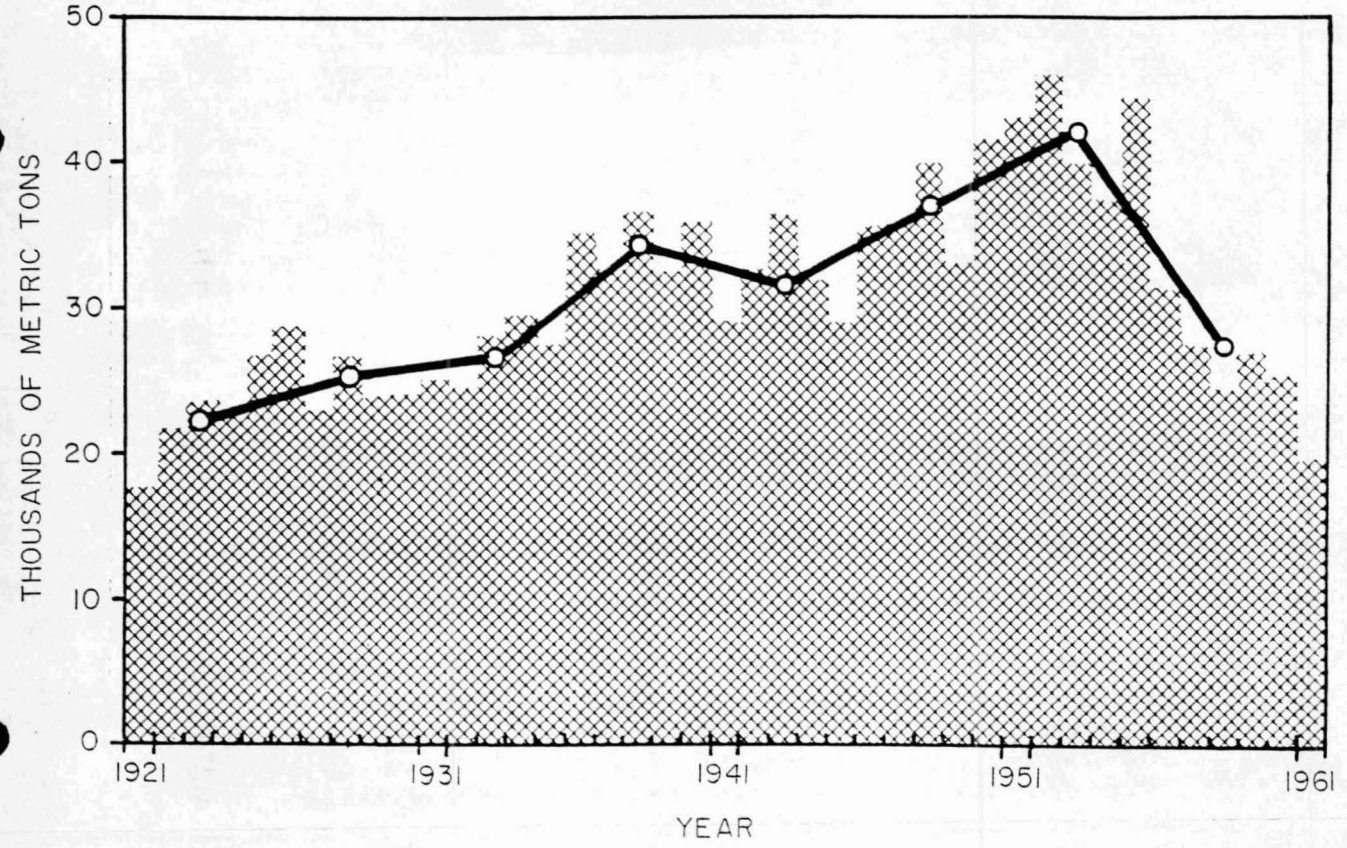

Fig. 7. Annual landings of Atlantic herring in the southern Gulf of Saint Lawrence, 1920-61, showing effects of 1954-55 fungal disease epizootic (from Sindermann, 1963). (Heavy line traces five-year moving averages.)

(2) "The spring fishery appears to have suffered most from the mortalities with average landings decreasing 46%, from 15.4 million lb (6985 metric tons) in 1950-54 to 8.3 million lb (3765 metric tons) in 1955-59. The autumn fishery declined 25%, from 8.5 to 6.4 million lb (3856 to 2903 metric tons) over the same period. Since 1959 there has been some evidence of recovery, as average annual catches for 1960 and 1961 were 17.6 million lb (7983 metric tons), an increase of nearly 20% over catches for the 1955-59 period."

(3) "Studies of the effects of disease on herring populations (Sindermann, 1958) and of herring landing statistics suggest a substantial reduction in the total abundance of herring in the Gulf of Saint Lawrence, with the spring-spawning stocks reduced to a greater extent than the autumn-spawning stocks. In the pre-epizootic period spring-hatched herring in Chaleur Bay greatly outnumbered autumn-hatched herring, especially in the spring fishery. For the post-epizootic period the proportions were roughly equal in 1960, but in 1961 there were indications of a partial return to pre-epizootic conditions."

(4) "Comparison of the catches of larvae for periods before (1951-53) and after (1957-61) the disease epidemic of 1954-56 shows that the average catch of spring-hatched larvae decreased from 589.1 to 8.4 per tow whereas the average catch of autumn-hatched larvae increased from 0.03 to 4.6 per tow."

(5) "Studies of the biology of herring in the Chaleur Bay area of the Gulf of St. Lawrence were carried out in 1960 and 1961 and results compared with similar data obtained in 1947 and 1948 (Tibbo, 1957). The comparison shows that in the intervening years major changes occurred in the herring stocks. These changes included a lowering of the level of abundance, a decrease in mean age, a reduction in the number of year-classes represented in commercial catches, an increase in growth rate, a decrease in the production of larvae, and an increase in the relative abundance of autumn-hatched fish."

Other statements in the paper by Tibbo and Graham (op. cit.) contained insights useful from a population dynamics perspective:

- "Commercial landings of herring declined sharply following the disease outbreak with no change in fishing effort. This could only have resulted from a decrease in the availability of herring and while decreased availability does not necessarily indicate decreased abundance it is the only reasonable conclusion that can be reached for this particular situation."

- "The observed decrease in the mean age of herring and the reduction in the number of year-classes represented in commercial catches in the post-epizootic period can also be attributed to the effects of the disease. Assuming that the distribution of age groups at the onset of the disease was normal and that the disease was not specific for any age group, it must be expected that the mean age of surviving fish would remain unchanged. However, the number of recruits to the commercial stocks in the post-epizootic period should be relatively higher and the end result would be a decrease in the mean age of herring taken commercially."

- "In spite of the fact that there has been a general failure to demonstrate a consistent relationship between the size of a spawning stock and the success of a resulting year-class or that factors affecting larval survival are equal from year to year, it is conceivable that the level of abundance of newly hatched larvae might be related closely to the size of the parent stock. The apparent decrease in abundance of adults coincided with the observed decrease in abundance of larvae; while there can be no assurance that this was 'cause and effect,' such a conclusion seems reasonable under the circumstances."

- "The effect of disease on a marine fish population is usually included in estimating mortality rates but seldom is it accompanied by factual information. The present study provides some information on the ecological consequences of an epizootic in herring and continuing efforts to describe the sequence of events in similar situations should be encouraged."

A subsequent effort to quantify the effects of the 1954-55 epizootic on Gulf of Saint Lawrence herring stocks was made by Parsons and Hodder (1975). Those authors suggested that the consequent reduction in predation and competition (since mackerel were affected also) undoubtedly provided conditions that were favorable for larval survival and the production of good year classes in 1958 and 1959, and that the great abundance of these two year classes as juveniles during 1959-62 and then as adults inhibited opportunities for good survival of young for several years thereafter. Poor recruitment of post-1959 year classes to exploited stocks led to a drastic decline in the southern Gulf of Saint Lawrence herring fishery beginning in 1971-72, once the strong 1958 and 1959 year classes had passed through the fishery (Parsons and Hodder, 1975).

6. EFFECTS OF HERRING DISEASE OUTBREAKS ON OTHER SPECIES

An epizootic in one marine species may have pronounced negative or positive effects on related or associated species. The presence of great numbers of a pathogen during an epizootic may create sufficient infection pressure so that members of other species with less susceptibility to the disease organism may become infected. The presence of dying or disabled infected individuals may temporarily increase the food supply for predators or scavengers, resulting in accelerated growth and increased weight of such species. Evidence for such negative and positive effects of an epizootic in one fish species on other species was obtained from the fungus outbreak in Gulf of Saint Lawrence herring. Alewives, which seem less susceptible as a species to the fungus pathogen (Sindermann and Scattergood, 1954), acquired infections and were killed in sufficient numbers to be observed. One mass mortality was observed at Dalhousie, New Brunswick, in 1955 and others were reported by fishery officers and fishermen. Fish (1934), in his report on the 1930-31 outbreak in herring, also reported that alewives were infected, and he was able to infect flounders by feeding them diseased herring.

Mackerel were also reported to be heavily infected by Ichthyophonus during the 1954-55 outbreak in herring, and scattered mortalities were observed. Cod, on the other hand, were not found to be heavily infected, even though samples taken in 1954 and 1955 were frequently found to have herring remains and also the fungus organism in their digestive tracts, suggesting that they were feeding heavily on herring. This was further suggested by the fact that when the herring mortalities began in 1954, the catches of the Gaspe long-line and hand-line cod fisheries decreased sharply, while those of the otter trawl fishery remained unchanged (Ronald, 1960). This also agrees generally with the observation of Cox that during the 1913-14 outbreak cod were found closer inshore than usual, refused bait, but were caught freely in salmon nets, an unusual occurrence. Cod landings in Chaleur Bay, which seemed to be a focus of the disease outbreak in the herring, were in 1956 (the first post-epizootic year) 52% above the average for the preceding eight years, and higher than any single year during this period. The 1957 catch was also exceptionally high. According to Martin (1957), landings of cod in the provinces of Quebec, New Brunswick, and Prince Edward Island in 1956 were the largest on record. Increased landings were due to larger size and not to greater numbers of fish caught. Individual cod were considerably larger and the growth rate was much higher in 1955-56 than in the period 1947-52. Although other factors might be involved, the increase may well be related to the abundance of diseased and dying herring in 1954 and 1955. The relationship of this greater growth of Gulf of Saint Lawrence cod to increased availability of herring as food was suggested by Martin (1956) and is supported by observations made by other investigators. If such a relationship is real, the outbreak period had positive as well as negative components.

The lobster fishery along the northern New Brunswick coast also suffered an almost immediate decline when herring mortalities began, probably because of the abundance of food outside the traps. Recovery of that fishery was rapid, however, in late summer and autumn, after the mass mortalities of herring had ceased (Sindermann, 1956).

Viewed from a broad perspective, disease outbreaks may appear to be catastrophic to the host population, but may be temporarily quite beneficial to predator or scavenger species, some of which may, as in this case, have a higher unit value to man than the affected species.

7. EPIZOOTIOLOGY

One of the most remarkable aspects of the fungal disease of herring in the western North Atlantic has been its history of repeated outbreaks. The two principal areas involved, Gulf of Maine and Gulf of Saint Lawrence, each with discrete populations of herring, have been out of phase during the most recent outbreaks; an epizootic peak in one gulf coincided with low disease prevalence in the other. The comparatively brief interval between some of the outbreaks suggests at best only transient increase in resistance of herring populations to the disease. This hypothesis is supported by relatively low disease prevalence (average, 27%) at the epizootic peak, which may constitute low selection pressure (or high virulence); by the fact that the most recent outbreak in the Gulf of Saint Lawrence stocks was at least as severe as the first recorded outbreak in 1898; and by the determination of mortality rates in experimental epizootics of comparable intensity (Sindermann, 1958).

It should be noted clearly, though, that alternative hypotheses exist. It may be, for example, that the relatively low prevalences during epizootic peaks are merely a reflection of the rapid and lethal course of acute infections during epizootics, resulting in an overly conservative estimate of actual disease impact, if only existing cases are counted. It may also be that the disease persists in other fish species in periods between outbreaks in herring, and reinvades when changes occur in the population and/or the environment. (Ichthyophonus has been reported from a number of other fish species, including some from the areas in which epizootics have occurred in herring.)

The fascinating story of Ichthyophonus outbreaks in herring of the western North Atlantic has had an interesting sequel in the eastern North Atlantic, in waters north of Scotland. Here two other species, plaice and haddock, have been infected at epizootic levels since 1971 and possibly earlier (McVicar and MacKenzie, 1972; McVicar, 1977, 1986), with records of the disease in that geographic area dating back to the early days of the twentieth century (Johnstone, 1905, 1913). A maximum of 25% infection of plaice has been reported in sampling from 1977 to 1981, and up to 85% of haddock were found to be infected during the same period.

No reports of mass mortalities of plaice or haddock due to Ichthyophonus have been published, but annual disease-caused mortalities exceeding 55% were estimated for plaice stocks off northern Scotland (McVicar, 1981). Despite this, plaice catches in that fishing area have been stable since 1974, possibly because of continuous immigration from other areas (McVicar, 1982). The disease seems to have a less severe effect on haddock, despite the reported high prevalences.

The disease in plaice and haddock from northern Scotland demonstrated differences in susceptibility of the two species to the pathogen. Plaice were less resistant, infections were rapidly fatal (within a few months), and fish were emaciated. Haddock from the same area seemed more resistant (as measured by inflammatory and encapsulation responses) and pathogenicity was assumed to be low. Prevalences in some samples reached 85% in haddock and 25% in plaice, but effects on plaice were considered more severe because of greater susceptibility to the pathogen (McVicar, 1982; Munro et al., 1983; McVicar and McLay, 1985). Using prevalence data and the presence of precipitating antibodies to the pathogen in serum of plaice, McVicar (1981) estimated an annual Ichthyophonus-related mortality of 55% for one fishing area off northern Scotland.

The literature on Ichthyophonus hoferi outbreaks in herring provides some limited information on prevalences during inter-epizootic periods -- indicating a rapid decline in pathogen abundance following an epizootic peak; an interim period of very low prevalences, then a gradual increase in prevalences before the next outbreak. Information from the Gulf of Maine investigations is instructive. Fish (1934) reported that the pathogen was seen in a sample from 1926, was common in 1929, and reached epizootic levels in 1930 and 1931. Scattergood (1948) reported the following:

> "For several years prior to 1947, the Maine sardine industry had been concerned over the apparently increasing incidence of the fungus in the herring."

Comparable fragmentary information suggests a gradual decrease in pathogen abundance after an epizootic peak. As an example, Scattergood's coastwide sampling in 1948 (the first post-epizootic year following the 1946-47 outbreak in the Gulf of Maine) disclosed substantial reductions in prevalences when compared to 1947, although they were still relatively high (between 3 and 9%). Then Sindermann and Scattergood (1954) found that prevalences in the Gulf of Maine during the period 1951 to 1954 had declined still further -- to less than 1% of all samples.

Beginning in the mid-1960s, gross examination for the presence of the fungus was incorporated into the protocol for collection of statistical data from Gulf of Maine herring for stock assessment purposes. Samples were checked for disease for seven years (1963-69) by the second author of this paper.

Prevalences of the disease, as determined by gross examination of the heart, were zero in all samples during that entire period (Table 2). After a hiatus during the 1970s and 1980s, when disease examination was not included in the assessment protocol, samples obtained in August and September 1992 were scrutinized for evidence of infection. Eleven samples (50 fish each) of immature and adult herring from nine locations on the New England coast were examined by Ms. Eileen Brewer of the Maine Department of Marine Resources, without finding any gross indication of Ichthyophonus infection (Table 3).

Some very limited information is also available about pre- and post-epizootic prevalences before and after the 1954-55 outbreak in the Gulf of Saint Lawrence herring. One definitive piece of data, supplied by Scattergood (1948) was that during the peak of the epizootic in Gulf of Maine herring (1947) samples of spring spawning fish from the southern Gulf of Saint Lawrence "contained no infected fish." Samples from that area in spring of 1948 were also free of Ichthyophonus infection. These data suggest lack of intermixing of herring stocks of the two Gulfs, and provide some evidence for absence of the disease in the Gulf of Saint Lawrence in 1947 and 1948. Scattergood's observations are important, since the disease apparently progressed from extreme scarcity or absence during the period of his sampling (1947 and 1948) to an epizootic level in 1954 -- only six years later. According to Sindermann's (1963) report, prevalences reached 27% in 1955 (an epizootic year in the Gulf of Saint Lawrence) and then declined to 1.2% in 1960, five years after the epizootic peak (Fig. 8). Subsequent prevalences, reported incidentally as parts of broader surveys, were zero in limited samples for 1980 to 1984 (Morrison et al., 1986) and zero in 136 herring sampled off Nova Scotia in 1986 and 1987 (Rand, 1992).

But the information is not definitive, and the story is not simple. For example, Forster (1941) did extensive sampling at Mount Desert Island, Maine, for diseased fish during the summers of 1939 and 1940 (midway between the Gulf of Maine outbreaks of 1930-31 and 1946-47) and reported an average prevalence of 6% for all fish examined -- with a range of 1.3 to 30%. He was undecided whether this figure indicated that the disease had declined to very low levels following the 1930-31 epizootic (prevalences were 15-20% in 1931) and was again increasing, or whether the disease had persisted at an intermediate level since the outbreak.

To be included also as "inconsistent findings" are data from Sindermann (unpublished report to Maine Sardine Council, September 1955) on prevalences in herring from the Nova Scotia coast in August 1955. One thousand fish were examined in samples collected from Halifax southward to Yarmouth and Digby. Overall disease prevalence averaged 4.2% with a range in the nine samples of 1.0 to 12.0. It will be recalled that 1955 -- the year of sampling -- was an epizootic year in the Gulf of Saint Lawrence, but these figures from the lower Nova Scotia coast do not suggest participation by those Nova Scotia spawning stocks in the Gulf epizootic, even though some infections were found in every sample.

Table 2. Annual prevalences of fungal infections in herring from the Gulf of Maine, 1963 to 1969.

Year	No. examined < 23.9 cm	No. examined > 23.9 cm	No. with gross evidence of disease
1963	203	0	0
1964	16,290	2,052	0
1965	16,989	272	0
1966	7,805	545	0
1967	7,038	1,592	0
1968	8,804	401	0
1969	7,933	696	0

Table 3. Examinations of adult herring for fungal infections in 1992 (data supplied by Eileen Brewer, Maine Department of Marine Resources).

Date	Location	No. examined	No. with gross evidence of disease
August 6	Wooden Ball Is.	48	0
August 8	Matinicus Is.	50	0
August 18	Mt. Desert Rock	50	0
August 24	Mt. Desert Rock	50	0
August 25	Jeffrey's Ledge	50	0
August 25	Metinic	50	0
August 25	Monhegan	50	0
September 1	Seal Is.	50	0
September 1	Seal Is. (2)	50	0
September 5	Matinicus Is.	50	0
September 8	Seal Is.	50	0

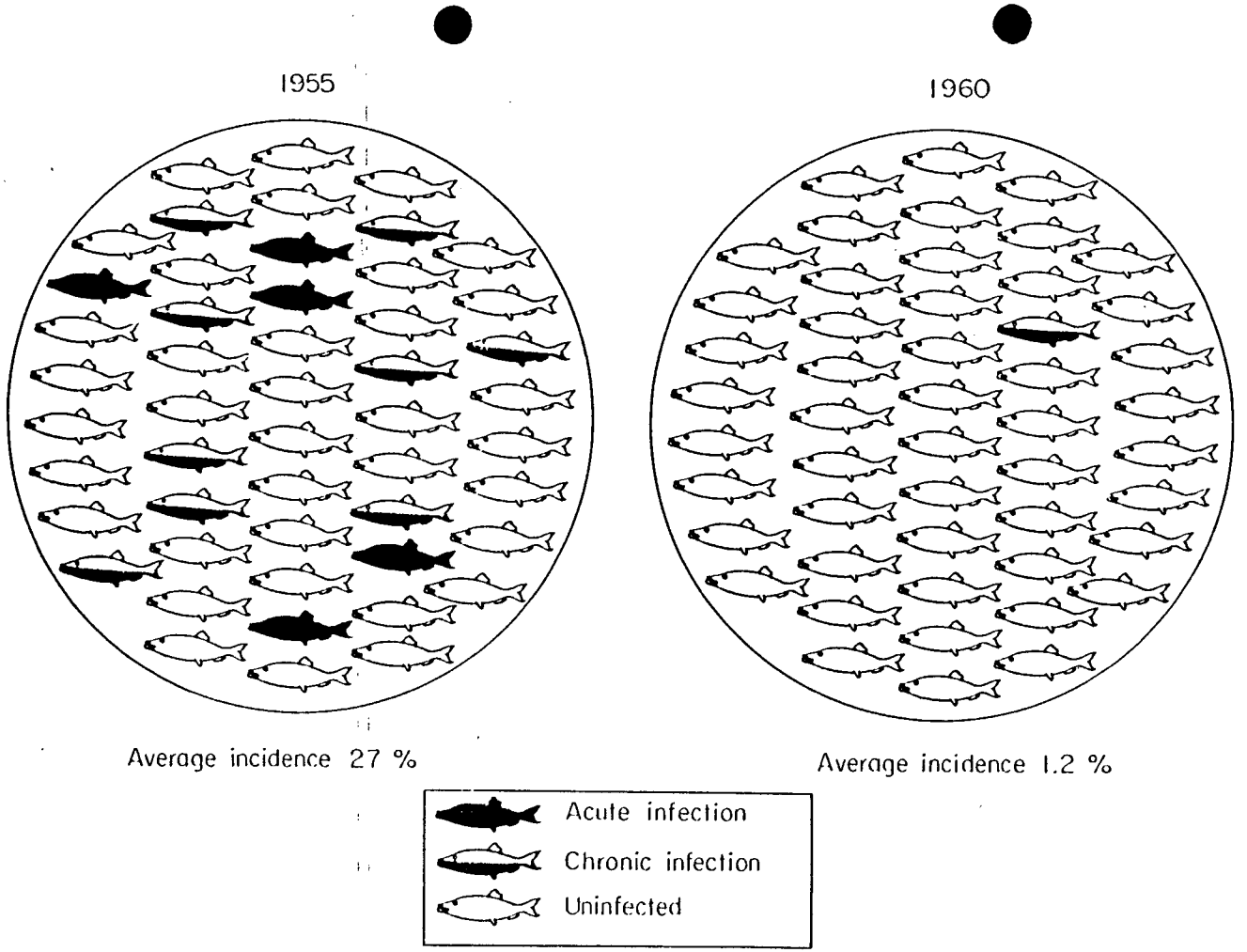

Fig. 8. Relative frequencies of infected individuals in samples of Atlantic herring from the Gulf of Saint Lawrence during (1955) and after (1960) the last epizootic of the fungus Ichthyophonus hoferi (from Sindermann, 1963).

One distinct possibility is that the pathogen, during inter-epizootic periods, becomes very rare in herring stocks or may disappear altogether -- being maintained in reservoir or alternate hosts such as yellowtail flounder, redfish, haddock, mackerel, or other species known to be susceptible to Ichthyophonus hoferi (Fig. 9). As an indication of this possibility, an epizootic was reported in yellowtail flounder from offshore Nova Scotian banks in the mid-1960s with prevalences averaging 25% on Sable Island Bank and 57% on Western Bank (Powles et al., 1968; Ruggieri et al., 1970). By 1986-87, the average prevalence in that species from the same general area was only 0.83% (range 0.4 to 13%) (Rand, 1992). Any relationship with the disease in herring is uncertain, but intriguing.

Before we leave this section on epizootiology, there should be a small niche for speculations -- which are always useful in the absence of factual information. Here are a few of our favorites:

- Disease outbreaks in herring may be consequences of interactions with populations of other susceptible species, which may be at any point on a maximum-minimum scale of disease abundance.

- During the period of scientific observation, we can see only a very short segment of a time line that can include fluctuations in disease abundance that may be measured in decades or even centuries.

- We are dealing with a disease of juvenile herring -- but one that may spill over into adult populations when seasonal geographic distributions overlap temporarily during epizootic periods.

What we may have here in repeated epizootics of Ichthyophonus in herring is a small glimpse of much larger and long-term interactions of a virulent pathogen and several susceptible species with widely varying habitats. Reported outbreaks (in addition to those in herring) have occurred in mackerel off the coast of England in the early 1940s (Sproston, 1946), in yellowtail flounder off the Nova Scotia coast in the late 1960s, and in plaice and haddock off the north coast of Scotland in the 1970s and early 1980s. We are looking at phenomena with broad geographic ranges -- on both sides of the North Atlantic -- and with broad host ranges as well. Other species, including invertebrates, may eventually be found to be susceptible, and epizootic fungal infections (but not with Ichthyophonus) are known to occur even in planktonic crustacean populations.

The pathways to understanding major disease events in the sea can be obscure, especially when the controlling variables are uncertain. Despite minimal research investment, the studies of herring epizootics have provided some insights of potential value in assessing the population impacts of disease.

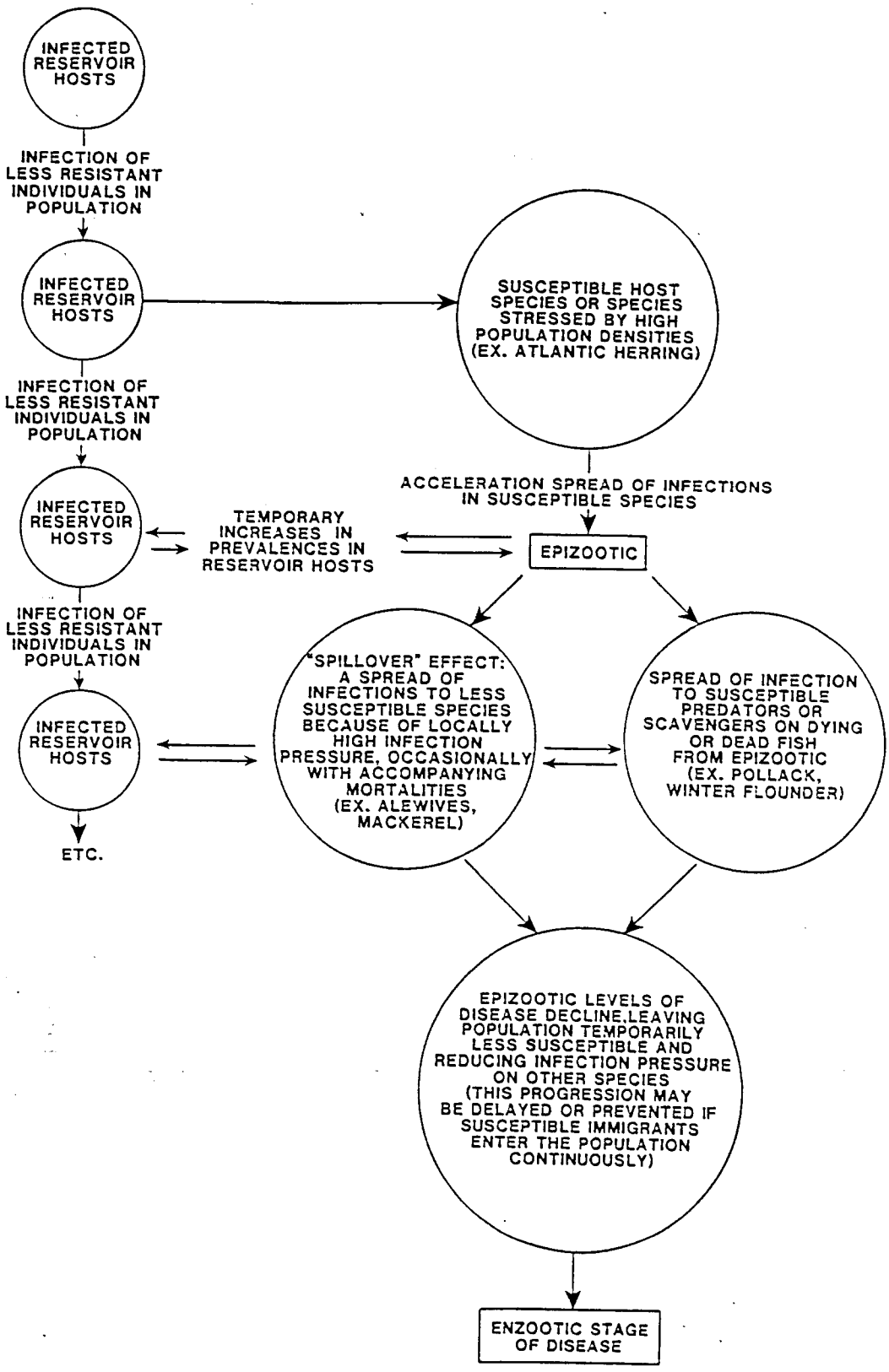

Fig. 9. Proposed dynamics of Ichthyophonus infections in western North Atlantic species (from Sindermann, 1990).

8. DISCUSSION

No outbreaks of Ichthyophonus have been reported in herring of the western North Atlantic since 1954-55 -- an interval of almost 40 years. During this period, occasional infections have been seen in other species (Hendricks, 1972), and an epizootic was reported in yellowtail flounder (Limanda ferruginea) in the mid-1960s off Nova Scotia (Powles et al., 1968; Ruggieri et al., 1970). Looking at the array of factors, extrinsic or intrinsic to herring populations, that could be involved in the onset of an epizootic, one factor that has changed significantly in this interim period is stock abundance -- which has been depressed substantially in recent decades, in part at least because of repeated year class failures accompanied by the application of more efficient fishing methods and greatly increased fishing effort. Other than this, nothing is known about such factors as the current level of population resistance to the pathogen, or the presence of environmental stressors that may increase susceptibility to disease. Earlier experimental studies suggested that changes in virulence of the fungus may not be critical in the short term, but infection pressure may be. From extremely limited gross examinations, present levels of the disease in herring stocks of the Gulf of Maine seem very low, but sampling is inadequate for any detailed statement, and no published reports of recent Ichthyophonus disease prevalences in Gulf of Saint Lawrence herring stocks are known to the authors.

Infections of small immature herring have characterized four of the five epizootics included in this report. In the Gulf of Saint Lawrence, where the fishery concentrates on adult fish, Cox (1916) referred to abnormal early spring inshore runs of diseased immature herring, 15-20 cm in length in both the 1898 and the 1913-14 outbreaks. In the Gulf of Maine, where the fishery concentrates on the earliest age groups (as a source of "sardines"), fish of these sizes were heavily infected, but mass mortalities were not reported for either the 1930-31 or the 1946-47 outbreaks. During the most recent and most intensively studied outbreak -- in the Gulf of Saint Lawrence in 1954-55 -- mortalities involved adult fish, and few immature individuals were seen. (It should be pointed out, however, that no data for those years about possible early runs of immature fish exist, to my knowledge.) The significance of the early abnormal runs of immature fish in the Gulf of Saint Lawrence is elusive, but it is clear that all age groups are susceptible to the pathogen.

In reviewing the literature on Ichthyophonus infections in herring and other species, several problem areas become apparent. One has to do with the taxonomy and identification of the organism. The genus Ichthyophonus has not been adequately circumscribed from cultural, morphological, biochemical, and genetic studies, as was pointed out by Johnson and Sparrow (1961) and Lauckner (1984). Thus, it is best described as a "species complex" possibly consisting of a number of species or variant strains. Culture studies of the genus have been infrequent;

diagnosis is usually made on the basis of morphology within the fish (which may vary with host species) and on the basis of differential staining reactions. Furthermore, other parasites, especially mycobacteria and microsporeans and even larval worms, may produce granulomatous lesions in marine fish which may be and undoubtedly have been confused with effects of I. hoferi infections, as was also suggested by Lauckner.

Another problem encountered quickly in attempts to understand the epizootiology of Ichthyophonus is the lack of continuity of observations. Administrative support for disease research is normally available only during epizootics, when the problems are acute. Events that precede or follow outbreaks rarely get recorded, because funds and research people are diverted to other more pressing crises. Such discontinuities can prevent the emergence of a coherent picture of the interactions of pathogen, host population, and environment that are so critical to any comprehension of disease effects.

9. CONCLUSIONS

Four geographically extensive epizootics have occurred in herring stocks of the western North Atlantic within the past century -- two in the Gulf of Saint Lawrence (1913-14 and 1954-55) and two in the Gulf of Maine (1930-31 and 1946-47). Each outbreak was linked by scientific investigators with high prevalences of a protistan pathogen having morphological characteristics similar to the phycomycetous fungus Ichthyophonus hoferi. A fifth and similar event, with mass mortalities, had occurred earlier (in 1898) in Gulf of Saint Lawrence herring, according to information from fishermen; their descriptions suggest a similar disease etiology, even though no scientific study was made at that time.

Studies of the disease of herring have been sporadic, and confined usually to the waning days of outbreak periods. Despite this severe limitation, information has accumulated from field and laboratory studies.

Conclusions reached from field investigations are:

1. Gross external and internal manifestations of infection of the herring by I. hoferi can be differentiated from other types of infection by careful examination. Externally, the "sandpaper effect" of multiple tiny papules, followed by ulceration and sloughing of the skin is characteristic. Internally, white nodules on and in the heart, liver, and lateral body musculature distinguish the disease.

2. Transmission of the disease appears to be direct, from fish to fish, and infection is probably effected by invasion of the digestive tract.

3. The disease is systemic. Hyphal activity is responsible for localized dissemination within the herring; while spore stages which may be demonstrated in the circulating blood may account for the generalized nature of the infection -- involving all areas and organs of the fish. Spores lodge in the smaller vessels and germinate, setting up foci of infection. Proliferation of the fungus seems to occur principally by formation, enlargement, and germination of hyphal bodies.

4. Heart, liver, and lateral body musculature of the herring are invaded most heavily. In advanced infections, much of the tissue of these organs is replaced by fungus. Involvement of the nervous system of the herring, although not severe, is also characteristic of the disease.

5. The disease does not usually exist in a dormant condition. Once invasion is effected, systemic involvement proceeds. Gross signs of infection become apparent in as little as 30 days following feeding of infected material. No large "pool" of lightly infected fish which do not exhibit gross symptoms of infection has been observed in herring of the western Atlantic.

6. From gross and histological examination of 0-age group herring, it appears that infection occurs in inshore waters. In one study in the Gulf of Maine, such fish were uninfected at the time of their first inshore movement in the spring, and during the first summer of life. The first evidence of infection was found in early September, when the herring were about one year old.

7. It is difficult to determine with any precision the exact reasons for periodic increases in fungal infection to epizootic proportions. Extrinsic factors such as extremes of environmental conditions or a disease outbreak in reservoir hosts, or intrinsic factors such as changes in population resistance or population density may be contributory.

Conclusions reached from results of experimental studies are:

1. The disease can be transmitted by feeding spores removed from infected fish. Exposure of 50 age-group 1 herring to 2×10^5 spores in a single dose resulted in no infection, but the same dose given on three successive days to another experimental group of 50 fish resulted in infections in four (8%).

2. Experimental groups of 50 age-group 1 herring fed increasing numbers of fungus spores (above the dosage in (1) above) exhibited increasing incidence of infections of the subacute or chronic type up to a certain level of dosage (usually four exposures to 2×10^5 spores on four successive days); above this level, acute infections and mortalities also occurred.

3. Acute infections occurred in experimental fish within one month from time of exposure to a sufficiently large number of spores. These infections were characterized by massive invasion of the heart and necrosis of tissue adjacent to spores, with little host cellular response. Subacute or chronic infections, characterized by pronounced host cellular response, occurred after one month from time of exposure. Such infections were progressive and terminal in most experimental fish.

4. An experimental epizootic, produced in 2000 age-group 0 herring by exposure on four successive days to doses of 2×10^5 spores each, resulted in infection of 23% of the population -- 8% acute and 15% subacute. The disease was terminal in all but light chronic cases within three months. Mortalities from acute infections occurred from 14 to 30 days, and from heavy chronic infections after that time. Control fish were negative for the disease throughout the experiment.

5. From percentages of infections obtained, age-group 0 herring appeared to be more susceptible to identical dosages of spores than age-group 1 fish.

6. The fungal organism from Gulf of Saint Lawrence herring was similar to that from Gulf of Maine herring in its effects on experimental fish. Organisms from the two Gulfs did not differ in culture morphology or growth rates.

When experimental results are related to observations of the outbreak in nature and to previous studies, a somewhat coherent picture of the disease in epizootic form begins to emerge, although there are still large gaps in our knowledge. It does appear that infection pressure and host susceptibility play major roles here, as they have been found to do in other epizootics.

New scientific awareness of the importance of disease in marine populations exists, and epizootics in herring stocks may be useful as models for studies of disease impacts on abundance of marine fish. An objective for the future could be a long-term international research effort on herring disease in the North Atlantic, as a new ICES initiative.

10. LITERATURE CITED

Athanassopoulou, F. 1992. Ichthyophoniasis in sea bream, Sparus aurata (L.), and rainbow trout, Oncorhynchus mykiss (Walbaum), from Greece. J. Fish Dis. 15: 437-441.

Chien, C.H., T. Miyazaki, and S. Kubota. 1979a. Studies on Ichthyophonus disease of fishes. IV. Comparative study on naturally infected fishes. Bull. Fac. Fish., Mie Univ. 6: 129-146 (in Japanese, English summary and plate legends).

Chien, C.H., T. Miyazaki, and S. Kubota. 1979b. Studies on Ichthyophonus disease of fishes. V. Culture. Bull. Fac. Fish., Mie Univ. 6: 147-151 (in Japanese, English summary and plate legends).

Chien, C.H., T. Miyazaki, and S. Kubota. 1979c. Studies on Ichthyophonus disease of fishes. VI. Artificial infection. Bull. Fac. Fish., Mie Univ. 6: 153-159 (in Japanese).

Chien, C.H., T. Miyazaki, and S. Kubota. 1979d. Studies on Ichthyophonus disease of fish. VII. Morphology and life cycle. Bull. Fac. Fish., Mie Univ. 6: 161-172 (in Japanese, English summary and plate legends).

Cox, P. 1916. Investigation of a disease of the herring (Clupea harengus) in the Gulf of St. Lawrence 1914. Contrib. Can. Biol. Fish. 1914-1915: 81-85.

Daniel, G. 1933a. Studies on Ichthyophonus hoferi, a parasitic fungus of the herring (Clupea harengus). I. The parasite as it is found in the herring. Am. J. Hyg. 17: 267-276.

Daniel, G. 1933b. Studies on Ichthyophonus hoferi, a parasitic fungus of the herring (Clupea harengus). II. The gross and microscopic lesions produced by the parasite. Am. J. Hyg. 17: 419-501.

Egusa, S. 1983. Disease problem in Japanese yellowtail, Seriola quinqueradiata, culture: a review. Rapp. P.-V. Reun. Cons. Int. Explor. Mer 182: 10-18.

Ellis, M. F. 1928. Ichthyophonus hoferi Plehn and Mulsow, a flounder parasite new to North American waters. Trans. N.S. Inst. Sci. 17: 185-192.

Fish, F.F. 1934. A fungus disease in fishes of the Gulf of Maine. Parasitology 26: 1-16.

Forster, R.P.. 1941. The present status of the systemic fungus disease in herring of the Gulf of Maine. Bull. Mt. Desert Is. Biol. Lab. 1941: 33-36.

Hendricks, J.D. 1972. Two new host species for the parasitic fungus Ichthyophonus hoferi in the north-west Atlantic. J. Fish. Res. Board Can. 29: 1776-1777.

Hofer, B. 1893. Ein Salmoidenkränkung. Allg. Fisch. Ztg. 8: 168.

Johnson, T.W. and F.K. Sparrow. 1961. Fungi in Oceans and Estuaries. Cramer, Weinheim.

Johnstone, J. 1905. Internal parasites and diseased conditions of fishes. Rep. Lancashire Sea-fish. Lab. 14: 151-185.

Johnstone, J. 1913. Diseased conditions of fishes: a phycomycetous fungus in a mackerel (Scomber scomber). Proc. Trans. Liverpool Biol. Soc. 27: 28-33.

Kubota, S. 1967. On diseases of cultured marine fishes in Mie Prefecture. Gyobyo Kenkyu 1: 78-84.

Lauckner, G. 1984. Agents, pp. 89-113. In Kinne, O. (Ed.), Diseases of Marine Animals, Vol. 4, Part 1. Biol. Anst. Helgol., FRG.

Leim, A.H. 1955. Herring mortalities in the Bay of Chaleur in 1955. Fish. Res. Board Can., Prog. Rep. Atl. Coast Sta. 62: 30-32.

Leim, A.H. 1956. Herring mortalities in the Gulf of St. Lawrence, 1955. Misc. Spec. Publ., Fish. Res. Board Can. 607: 1-9.

Martin, W. 1956. Change in growth of Gulf cod. Fish. Res. Board Can., Annu. Rep. Biol. Sta. 1956: 63-66.

Martin, W. 1957. Canadian researches: high cod landings. Int. Comm. N.W. Atl. Fish., Annu. Proc. 7: 25.

McVicar, A.H. 1977. Ichthyophonus as a pathogen in farmed and wild fish. Bull. Off. Int. Epizoot. 87: 517-519.

McVicar, A.H. 1981. An assessment of Ichthyophonus disease as a component of natural mortality in plaice populations in Scottish waters. Int. Counc. Explor. Sea, Doc. C.M.1981/G49, 7 pp.

McVicar, A.H. 1982. Ichthyophonus infection in fish, pp. 243-269. In Robert, R.J. (Ed.), Microbial Diseases of Fish. Academic Press, London.

McVicar, A.H. 1986. Fungal infection in marine fish and its importance in mariculture. Spec. Publ., Eur. Aquacult. Soc. 9: 189-196.

McVicar, A.H. and K. MacKenzie. 1972. A fungus disease of fish. Scott. Fish. Bull. 37: 27-28.

McVicar, A.H. and H.A. McLay. 1985. Tissue response of plaice, haddock and rainbow trout to the systemic fungus Ichthyophonus, pp. 329-346. In Ellis, A.E. (Ed.), Fish and Shellfish Pathology. Crown, London.

Miyazaki, T. and Y. Jo. 1985. Studies on Ichthyophonus disease of ayu. Gyobyo Kenkyu 20: 45-48.

Miyazaki, T. and S.S. Kubota. 1977a. Studies on Ichthyophonus disease of fishes. I. Rainbow trout fry. Bull. Fac. Fish., Mie Univ. 4: 57-65.

Miyazaki, T. and S.S. Kubota. 1977b. Studies on Ichthyophonus disease of fishes. II. Yearling rainbow trout -- chronic infection. Bull. Fac. Fish., Mie Univ. 4: 57-65.

Miyazaki, T. and S.S. Kubota. 1977c. Studies on Ichthyophonus disease of fishes. III. Life cycle of Ichthyophonus affected rainbow trout. Bull. Fac. Fish., Mie Univ. 4: 57-80.

Morrison, C.M., G. McClellend, J. Cornick, and D. Marcogliese. 1986. Parasites and diseases of some marine finfish off Nova Scotia. Can. Tech. Rept. Fish. Aquat. Sci. 1424: 1-36.

Munro, A.L.S., A.H. McVicar, and R. Jones. 1983. The epidemiology of infectious disease in commercially important wild marine fish. Rapp. P.-V. Reun., Cons. Int. Explor. Mer 182: 21-32.

Neish, G.A. and G.C. Hughes. 1980. Fungal diseases of fishes, pp. 1-159. In Snieszko, S.F. and H.R. Axelrod (Eds.), Diseases of Fishes, Book 6. T.F.H. Publ., Neptune City, NJ.

Neresheimer, E. and C. Clodi. 1914. Ichthyophonus hoferi Plehn and Mulsow, der Erriger des Taumelkrankheit der Salmoniden. Arch. Parasitenkd. 34: 217-248.

Okamoto, N., K. Nakase. H. Suzuki, Y. Nakai, K. Fujii, and T. Sano. 1985. Life history and morphology of Ichthyophonus hoferi in vitro. Gyobyo Kenkyu 20: 273-285.

Parsons, L.S. and V.M. Hodder. 1975. Biological characteristics of southwest Newfoundland herring, 1965-71. ICNAF Res. Bull. No. 11, pp. 145-160.

Plehn, M. and K. Mulsow. 1911. Erreger der "Taumelkrankheiten" der Salmoniden. Z. Bakteriol. Parasitenkd. Infektionskr., Abt. 1, Orig. 59: 63-68.

Powles, P.M., D.G. Garnett, G.D. Ruggieri, and R.F. Nigrelli. 1968. Ichthyophonus infection in yellowtail flounder (Limanda ferruginea) off Nova Scotia. J. Fish. Res. Board Can. 25: 597-598.

Rand, T.G. 1992. Distribution of Ichthyophonus hoferi (Mastigomycotina: Ichthyophonoles) in yellowtail flounder, Limanda ferruginea, from the Nova Scotia shelf, Canada. J. Mar. Biol. Assoc. U.K. 72: 669-674.

Ronald, K. 1960. Herring mortalities in the coastal waters of the Gaspe Peninsula. Rep. Minist. Fish. Prov. Que. 1954, 5 pp.

Ruggieri, G.D., R.F. Nigrelli, P.M. Powles, and D.G. Garnett. 1970. Epizootics in yellowtail flounder, Limanda ferruginea Storer, in the western North Atlantic caused by Ichthyophonus, an ubiquitous parasitic fungus. Zoologica (N.Y.) 55: 57-62.

Scattergood, L.W. 1948. A report on the appearance of the fungus Ichthyosporidium hoferi in the herring of the northwestern Atlantic. U.S. Fish Wildl. Serv., Spec. Sci. Rep.--Fish. 58: 1-33.

Schäperclaus, W. 1953. Fortpflanzung und Systematik von Ichthyophonus. Z. Aquarien-Terrarienver. 6: 177-182.

Sindermann, C.J. 1956. Diseases of fishes of the western North Atlantic. IV. Fungus disease and resultant mortalities of herring in the Gulf of St. Lawrence in 1955. Maine Dept. Sea Shore Fish., Res. Bull. 25: 23 pp.

Sindermann, C.J. 1958. An epizootic in Gulf of Saint Lawrence fishes. Trans. North Am. Wildl. Conf. 23: 349-360.

Sindermann, C.J. 1963. Disease in marine populations. Trans. North Am. Wildl. Conf. 28: 336-356.

Sindermann, C.J. 1965. Effects of environment on several diseases of herring from the western North Atlantic. Spec. Publ.--Int. Comm. N.W. Atl. Fish. 6: 630-610.

Sindermann, C.J. 1979. Status of northwest Atlantic herring stocks of concern to the United States. U.S. Dept. Commerce, NOAA, Natl. Mar. Fish. Serv., Tech. Ser. Rep. No. 23, 449 pp. Sandy Hook, N.J.

Sindermann, C.J. 1990. Principal Diseases of Marine Fish and Shellfish, Vol. I. Academic Press, New York.

Sindermann, C.J. and L.W. Scattergood. 1954. Diseases of fishes of western North Atlantic. II. Ichthyosporidium disease of the sea herring (Clupea harengus). Maine Dept. Sea Shore Fish., Res. Bull. 19, 40 pp.

Sprague, V. 1965. Ichthyosporidium Caullery and Mesnil, 1905, the name of a genus of fungi or a genus of sporozoans? Syst. Zool. 14: 110-114.

Sproston, N.G. 1944. Ichthyosporidium hoferi (Plehn & Mulsow, 1911), an internal fungoid parasite of the mackerel. J. Mar. Biol. Assoc. U.K. 26: 72-98.

Templeman, W. 1954. Herring mortality. Rep. Newfoundland Fish. Res. Sta. 1954, App. No. 14: 25-26.

Tibbo, S.N. 1957. Herring of the Chaleur Bay area. Bull. Fish. Res. Board Can. 111: 85-102.

Tibbo, S.N. and T.R. Graham. 1963. Biological changes in herring stocks following an epizootic. J. Fish. Res. Board Can. 20: 435-449.