A Diagnostic Manual of Eel Diseases Occurring Under Culture Conditions In Japan

William L. Rickards, Editor

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Translated from the Japanese by Fumiko Gregg

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PREFACE

The University of North Carolina Sea Grant College Program has conducted a demonstration eel culture program near New Bern, North Carolina since 1975. During the first year of operation, diseases resulted in a complete loss of the young eels being cultured. At that time, it became painfully evident that the available information concerning parasites and diseases of the American eel (Anguilla rostrata) was extremely limited and of little use to eel farmers because of its technical nature and narrow scope. Therefore, one of the project's objectives became the compilation of literature relating to diseases of all freshwater eels since it is likely that the disease problems encountered in the American eel will involve species of the same genera that infect eels in Europe or Japan. In fact, some of the same species may be involved world-wide.

The eel disease literature search revealed that the Japanese Fisheries Agency had produced the Fish Disease Diagnosis Guide I, authored by Shuzo Egusa and Akio Honma in 1974. One section of this guide contains information on disease diagnosis and treatment for eels, primarily Anguilla japonica, cultured in Japan. Since this information is relevant to culturing the American eel, the North Carolina State University Eel Culture Project decided to fund the translation of the eel section and to have the color photographs reproduced. The translation was completed by Fumiko Gregg, and permission to print the materials was granted by the Japanese Fisheries Agency. Because of restrictions imposed by the copyright owners, distribution of this book is limited to the United States. Also, because the original negatives of the color photographs were not made available, it was necessary to copy the figures directly from the Japanese book. Thus, while some of the color work is not as clear as hoped and there is a faint pinkish banding effect on some figures, the essence of the material should be of value to both researchers and private individuals working with the American eel.

A few other comments regarding this translated material are also necessary. The text contains some suggested antibiotics and chemicals for treatment of particular diseases. The reader should bear in

mind that some of these chemicals have not received clearance from governmental agencies for use on fish being reared for sale as human food. Before any chemical is used, the legality of such an application should be determined so that the crop will not face possible seizure for being contaminated.

Many therapeutic agents are recognized by a variety of names. A listing of currently existing chemicals may be found on pages 321-329 of the book, Disease Diagnosis and Control in North American Marine Aquaculture, by C. J. Sinderman (1977, Developments in Aquaculture and Fisheries Science, Volume 6, Elsevier Scientific Publishing Co., New York). The list includes both generic (scientific) names and trade names for those chemicals suggested for use on eel diseases.

The text contains the names of several eel body parts and structures. For those not familiar with such terminology, definitions may be found in the book, Ichthyology, the Study of Fishes by K. F. Lagler, J. E. Bardach and R. R. Miller, 1962, Wiley & Sons, Inc., New York.

Finally, the terms which are defined below are used in the translated text and require a degree of explanation for clarity.

I. Terms applied to eels:

a) black larval eels - pigmented elvers.

b) whitebait eels - as nearly as can be judged, these are young eels, 1 to 20 grams in weight, which have been in the nursery pond; synonymous with the terms seed eels and fry which are also used in the text.

c) Japanese eel - Anguilla japonica

d) European eel - Anguilla anguilla

e) American eel - Anguilla rostrata

f) Southeast Asian eel - Anguilla bicolor

II. Terms applied to disease conditions and measurements:

a) Catarrhal inflammation - reddening of the mucous membranes.

b) Clavated gill filaments - gill filaments which have become club-shaped.

c) Etiology - the causative agent of a disease; i.e. the pathogen.

d) Foci - points or locations of infection.

e) Hypertrophy - exaggerated growth of a body part.

f) Laparotomy - cutting of the abdominal wall.

g) Necrosis - localized tissue death.

h) μ - micron; equal to 0.001 millimeter.

This book was not intended as a technical text for scientists. It was written primarily for use by the Japanese eel farming industry, and the translation is likewise intended for application to eel culture. Researchers may find it useful in diagnosing or treating diseases in eels being kept for experimental purposes, and we will consider this to be an added benefit from our work.

William L. Rickards

I. RED FIN DISEASE

Red fin disease is a common contagious bacterial disease of eels which has been know for a long time. Eels infected with it can be found the year round, but it is most prevalent between March and June when the water temperature rises, especially during the period when the eels begin to feed. It infects eels at every phase of growth, but it is more widespread among whitebait and black larval eels.

During the months of April and May when the water temperature changes rapidly, red fin disease may affect seed eels and cultivated mature eels a great deal. Generally speaking, both the prevalence and damage caused by red fin disease are less during the period of high water temperature. If an eel becomes infected during this period, it tends to heal naturally in one to three weeks after the onset of the disease.

Etiology

Red fin disease is caused by an infection of the bacterium, Aeromonas hydrophila (A.punctata and A.liquefaciens are synonymous). The characteristics of this bacterium are: 1) it has a single flagellum at its extremity, 2) it secretes oxidase, and 3) it dissolves glucose with an enzyme yielding acid and gas.

Besides Aeromonas hydrophila, Paracolobactrum anguillimortiferum (presently under the genus Edwardsiella) is also said to cause red fin disease. In fact, the infectious symptoms of both pathogens are similar, and there are many cases of mixed infections. However, our view is that it is not appropriate to call diseases with different bacterial pathogens by the same name. Therefore, in this book, the name red fin disease will be used only for Aeromonas infections and the Edwardsiella infections will be treated separately under the heading of Paracolo disease. We would also like to add that Vibrio anguillarum infections, which possess disease signs similar to those of red fin disease, were not known until now in Japan. However, we have recently seen outbreaks of this disease in eel grow-out ponds in the Shikoku district.

Diagnosis

The external signs of red fin disease are characterized by reddening of the anal and pectoral fins and of the bases of these fins, the ventral skin, and the anal zone. Bleeding spots on the belly characterize advanced stages of the infection. It sometimes causes necrosis or ulcers on the body. Visceral pathology includes congestion or hemorrhages of the liver, bleeding or dilation of the wall of the stomach, mucus repletion, catarrhal inflammation of the intestinal canal, and congestion or swelling of the kidneys. Although observations from dissections are varied and include exceptions, catarrhal inflammation of the intestinal canal is considered to be an important characteristic symptom of red fin disease. However, abscesses observed in the kidneys and liver, which have hitherto been said to be the main symptom of red fin disease, are not considered by us to be the main symptom of this disease, but rather to be the principal sign of an *Edwardsiella* infection.

Though most of the diagnosis can be done from these internal and external symptoms, it is desirable to conclude the diagnosis only after isolation and culture of bacterial pathogens from infected eels, confirmation of their virulence by injecting eels with cultured pathogens, and other general symptomatic tests. These procedures are desirable because the symptoms of *Edwardsiella* and *Vibrio* infections resemble those of red fin disease, and because the slight reddening of the fins and the belly observable in the first stage of infection is not necessarily peculiar to red fin disease.

Measures for Disease Control

As an introduction to the section on measures for controlling diseases in eels, we would like to mention some basic prophylactic steps. The best prophylactic measure is to stock sanitary ponds with healthy whitebait and seed fish and raise them at the proper density and with the proper diet without subjecting them to undue environmental stresses. Unfortunately, this is easier said than done. In practice, the best way is to employ at least minimum prophylactic measures, and to try to treat an outbreak at the earliest possible opportunity. More

specifically, the procedures for the control of red fin disease are as follow: 1) transfer white-bait and seed fish to ponds only after the fish have been well treated in a bath containing medicine such as Furan agents; 2) avoid adding seed fish to ponds during the fall and winter; 3) disinfect the ponds at least once a year; 4) reduce the amount of feed during the period when the young eels begin to feed and when the water temperature is rapidly changing; 5) strive for early detection of the disease; 6) be especially careful to inspect the bottoms of the ponds for dead fish; and 7) remove dead fish immediately and destroy them properly, i.e. by incineration. If an outbreak of red fin disease is confirmed, after the above-mentioned steps are carried out, administer the appropriate antibiotics. The antibiotics should be selected after performing a simple susceptibility test on isolated pathogens because the susceptibility differs greatly even among individuals of the same Aeromonas hydrophila species. In case the diseased fish will not accept food or if they are being transferred, they should be treated by the medicated bath method. Usually the medicine is mixed into the food and is administered continuously for a certain period of time. Dosage will be omitted here since it is regulated by the kind of medicine, and not by the kind of disease. In addition, the use of any medicine should be performed under the guidance of specialists since matters concerning public health have to be taken into consideration.

Figure I - 1. A healthy eel (top) and a red fin diseased eel (bottom).

Figure I - 2. Reddening of the pectoral and anal fins and of the bases of these fins, and of the ventral skin is conspicous. A group of infected eels gathered in early spring when the disease is most prevalent. Various symptoms can be seen. Among other symptoms, water mildew is often seen among the sick when the water temperature is low (arrows).

(Text by Wakabayashi)

(Pictures by Egusa)

Photo 1

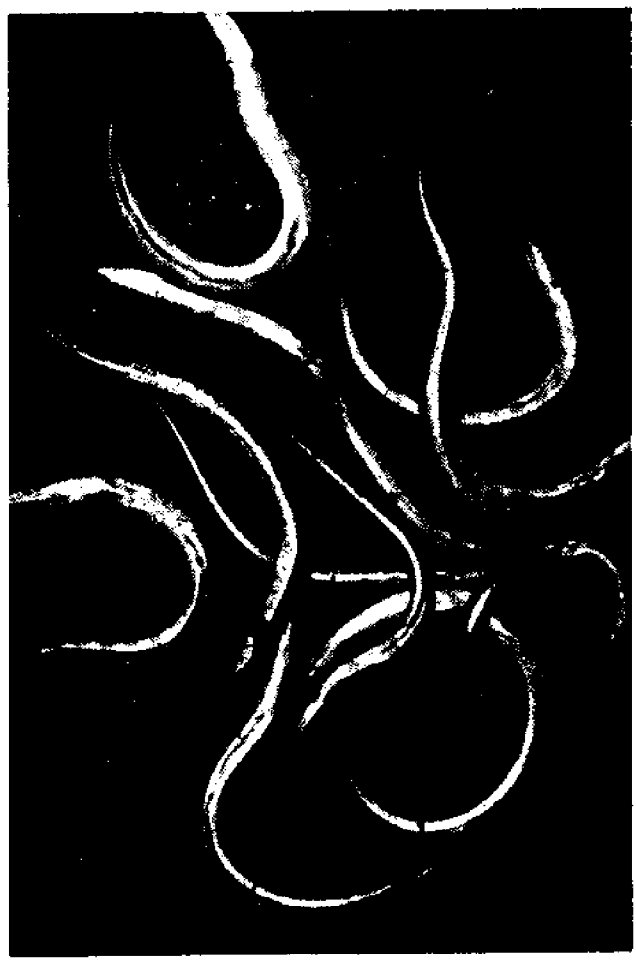

Photo 2

II. RED SPOT DISEASE

Discovered for the first time in eel grow-out ponds in both Shizuoka and Tokushima Prefectures in the spring of 1971, red spot disease is a relatively new bacterial disease in eels. After this incident, red spot disease was confirmed in Kochi, Yamaguchi, and Nagasaki Prefectures. Outbreaks of this disease begin in early spring, declining as the water temperature rises above 25°C, and ceasing in summer. Though it occurs again in the fall, its incidence is not as great as in the spring, and sometimes a fall outbreak is not seen. It is generally recognized that red spot disease tends to occur in ponds containing any amount of salt (usually located in coastal regions) and it tends not to occur in fresh water ponds. It is also said to be true that Japanese eels become infected easily, while European eels are hardly susceptible to this disease.

Etiology

The red spot disease is caused by infections of the bacterium, Pseudomonas anguilliseptica. This bacterium grows in a meat extract culture medium, but its growth is slow. It takes 3 to 4 days before the bacteria form a colony. Its growth is retarded when the temperature is above 30°C, and movement is nearly halted above 25°C. The bacterium is approximately 2μ by 0.4μ in size, and has a single flagellum at its extremity. It is gram negative and produces oxidase, but its principal characteristic is that it does so without using sugar.

Diagnosis

The most prominent characteristic of red spot disease is the presence of conspicuous bleeding spots. Bleeding spots are also often observed in other similar infections, such as red fin disease; but the bleeding of red spot disease occurs under the transparent outer skin (the epidermis) so that the fish looks healthy when the disease is mild. However, close examination reveals that the entire body is covered with small but

vivid spots. In the case of a serious red spot infection, these bleeding spots will have risen to the surface, and a gentle rubbing motion peels off these spots together with the outer skin. Reddening is not usually conspicuous on the pectoral fin, the anal fin, or the anal zone. Internally, swelling of capillary vessels, spotty hemorrhages of the walls of the abdominal cavity, and sometimes hemorrhages of the liver are observed, but they are not severe. Catarrhal inflammation of the intestinal canal is practically nonexistent. It is possible to diagnose red spot disease relatively easily and accurately from the above-mentioned symptoms.

Measures for Disease Control

Although it is fairly easy to diagnose red spot disease, due to the fact that there is about one week of the latent infectious period, by the time symptoms can be seen with the naked eye, the fish are suffering from extreme bacterial hemorrhages, and are so critically ill that medical treatment cannot save most of them. Susceptibility tests show that the culture bacterium, Pseudomonas anguilliseptica, is highly susceptible to antibiotics such as chloramphenicol and tetracycline, and Furazolidone and other Furan agents. However, these drugs are not very effective when actually given to the diseased cels. This is probably because the disease is only detected in its late stages. Therefore, it is necessary to discover the infection before symptoms become visible and to administer medical treatment. Unfortunately, we do not have an effective early detection method at present. A bacterial examination is the only available method today; and, by the time red spot disease is found, it tends to be too advanced to be cured.

By taking advantage of the fact that the bacteria which cause red spot disease do not tolerate high temperatures, it may be possible to arrest red spot disease if the pond can be heated to 26° or 27°C when symptoms of red spot disease are detected.

Figure II - 1. Typical hemorrhages on the head of an eel suffering from red spot disease. The red spots are very vivid because the bleeding occurs very close to the outer layer of the skin. Bleeding spots are observed on the head at a fairly early stage of the disease.

Figure II - 2. Various degrees of hemorrhaging are shown. The eels at the top and bottom of the photograph are suffering from medium degrees of hemorrhaging, while the eel in the center is in a serious condition. If an eel in this serious condition is wrapped in Japanese rice paper, starchy blood stains are transferred to the paper. Thus, the disease used to be called the "bleed if you rub" disease.

Figure II - 3. Bodily bleeding is seen to be quite heavy, but hardly any reddening is seen on the anal zone or the anal fin.

(Text by Wakabayashi)

(Pictures 1 and 3 by Wakabayashi, 2 by Egusa)

Photo 1

Photo 2

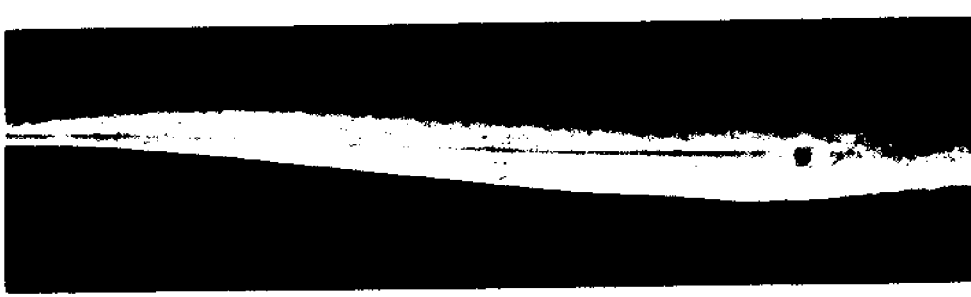

Photo 3

III. PARACOLO DISEASE

(Chōman (= swelling), Red Fin Disease)

Paracolo disease has been known for a long time in the Yoshida area in Shizuoka Prefecture. The disease called "Chōman" by eel breeders is usually Paracolo disease. They call it "Chōman" probably because the areas around the anus and the liver swell to an unusual degree. However, such a swelling symptom is not peculiar to Paracolo disease alone. This disease is most prevalent in summer, but it is not surprising for it to occur in late spring or in early fall. Paracolo disease has not been reported anywhere except in Shizuoka Prefecture, and its geographical extent is not clearly known.

The cause of Paracolo disease is an infection of the bacterium, Edwardsiella tarda, but since symptoms of Paracolo disease can seem similar to those of the disease caused by the bacterium, Aeromonas hydrophila, both of these diseases have been called red fin disease even in scientific circles. However, it is not appropriate to call diseases with different causes by the same name. Furthermore, since pathological differences between these two bacterial infections have become clear, it is desirable both scientifically and practically to give them separate names. We believe it is appropriate to call the Aeromonas infection by the name of red fin disease, and we shall call this infection Paracolo disease, deriving this name from that of the Paracolobactrum bacterium.

Etiology

The pathogenic bacterium, Edwardsiella tarda, belongs to the family of intestinal bacteria. It is gram negative and is a hairy and short bacillus. Its principal characteristic is that it does not use lactose. The range of temperatures in which the bacteria reproduce is about 15° to 40°C. This bacterium used to be called Paracolobactrum anguillimortiferum, but this generic name is no longer used in bacterial taxonomy. Thus, it has been given the name Edwardsiella tarda.

Diagnosis

Strictly speaking, a bacterial test is necessary to diagnose Paracolo disease. However, it is quite safe to diagnose Paracolo disease on the basis of a dissection alone, because many bacterial infection points of various sizes are likely to be observed in either the kidneys, the liver, or in both. In eels with advanced symptoms, the tissues of these organs are melted and ulcers are formed. As can be seen from the figures on the next page, most diseased eels show the characteristic external symptoms as well. Of course, there are a variety of symptoms which differ from individual to individual. Inflammation of the intestines is the principal symptom of Paracolo disease, rather than pathological changes in the kidneys or the liver. Extreme reddening of the skin and/or the fins sometimes bears a close resemblance to red fin disease. Occasionally, as the result of points of infection in the muscles of the body, especially of the belly, reddening appears on the skin above these infected muscles which resembles the redness produced by red fin disease. These cases can be diagnosed accurately only by bacterial tests. There are also cases which manifest complicated symptoms as the result of a mixed infection of red fin disease bacteria and Paracolo disease bacteria.

Measures for Paracolo Disease Control

The susceptibility of Paracolo disease bacteria to chemical treatment differs from case to case. Therefore, appropriate medicines should be prescribed according to their directions after a susceptibility test has been administed. The method of transmission is not clearly known, but infection by mouth is believed crucial. Therefore, it is recommended that as soon as signs of a Paracolo disease outbreak are seen, a Furan agent (nf --- 180, Furazolidone) should be added daily to the feed in the amount of 0.02% of the feed for 1 to 2 weeks in order to prevent the infection from spreading.

Figures III - 1 & 2.

The usual appearance of eels suffering from Paracolo disease. The anal zone is dark red and distended. The body is often swollen with the anal zone protruding due to the swelling of the main section of the kidneys. Also, the body surface above the infected kidneys frequently manifests pathological changes. (1) As the result of the swollen liver, the anterior part of the belly is often swollen and the skin on that part shows pathological changes and sometimes becomes ulcerated. (2) Most fish suffering from Paracolo disease exhibit redness on the anal fin and also extreme redness on wide areas of the belly, symptoms which closely resemble those of red fin disease. There are, however, fish which die showing few external symptoms.

Photo 1

Photo 2

Figure III - 3. Dissection

(A) The number of cases of this type is small. Inflammation of the intestines is severe and the intestines are swollen. A large number of hemorrhages can be seen on the peritoneum. No abnormality exists in either the kidneys or the liver. This case is difficult to distinguish from red fin disease.

(B) The main part of the kidney is enormously swollen. A large ulcer caused by the collapsed focus located at the anterior of the kidney (←1) can be seen as well. A dark red malodorous fluid flowing out of the ulcer can also be seen. Although not visible in this picture, there are many discolored and half collapsed foci of infection in the kidneys. There are also foci (←2) in the body muscles. There is no abnormality in either the digestive tract or the liver.

(C) Most of the front half of the liver is eroded (←3) and the position of the swollen back section has shifted to the front. Fully developed foci (←4) in the body muscles posterior to the kidney are affecting the skin. No particular abnormality exists in either the digestive tract or the liver.

(D) Nearly half of the liver is eroded and the remaining section is dark brown. There is a large hole in the belly skin right below the liver (←5). This hole is caused by pathogens from liver foci of infection which have infected the abdominal wall whose tissue has then disintegrated. Most of the collapsed liver tissue is believed to have flowed out through this hole. The red lesions (←6) on the wall of the abdominal cavity

and the reddening of the alimentary canal chorion were caused by an infection of the disease bacteria flowing out of the liver lesions. No abnormality exists in the kidneys. Redness and distension of the anal zone and swelling of the body around the anus are not observed in diseased eels which have mainly liver infections.

Figure III - 4. Dissection

Although there is no abnormality in the main parts of the kidneys, the front left section is swollen and in it there are large and small white dot-like foci (←7) and ulceration can be seen. The body muscles directly touching these ulcers also have large and small holes in them. Dark red fluid has collected in these holes, and it produces a bad odor. Since there was no abnormality in any other organ, this infection was discovered only after removing the alimentary canal and the air bladder. Diseased eels with only the front section of the kidneys infected, as described here, manifest hardly any external symptoms.

(Text by Egusa)

(Pictures by Egusa)

Photo 3

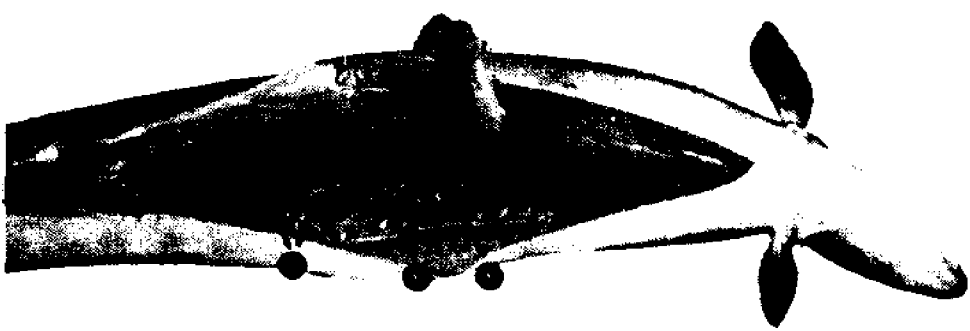

Photo 4

IV. GILL ROT DISEASE

(Gill Disease, Columnaris Disease)

Gill rot disease was first observed in eel grow-out ponds in Yoshida, Shizuoka in the summer of 1966. Since then, this disease has broken out in various localities every summer when the water temperature rises. Moreover, the frequency of outbreaks of this disease and their resultant damage have been increasing every year.

Gill rot disease is caused by a myxobacterium infecting the gills. Infected fish do not show any particularly severe symptoms externally. However, when the gills are exposed, one can often observe (with the naked eye) a yellow substance adhering to the gills. Also, the tips of the gill filaments are missing. In the initial stage of this disease, this loss is only partial and the color of the gills is bright red. As the diseased condition advances, the decay affects all of the filaments, filth sticks to the gills, the color of the gills turns from pink to white, and the fish manifests an anemic condition. As the anemia progresses, the color of the viscera fades, the liver becomes milky yellow, and the body becomes almost grayish white. The sick eel thrashes about weakly at the water surface throughout the evening. Diseased eels are very easily affected by a lack of oxygen or a change in water quality and their appetite decreases.

Gill rot disease has a high frequency of occurence in grow-out ponds containing adult eels, but it rarely breaks out in ponds containing seed eels. The fry in such ponds are especially immune. Generally speaking, the occurrence of gill rot disease is higher in densely populated grow-out ponds. The daily mortality rate is not very high. Usually a few dozen diseased eels die every day over a long period of time.

Gill rot disease is most common in summer when water temperatures are high. It rarely occurs in water colder than 15°C.

Etiology

The pathogen of gill rot disease is the myxobacterium, Chondrococcus columnaris, which parasitizes the gills. This bacterial agent is a long, gram-negative

bacillus, 3 to 8μ x 0.5μ in size. It moves in the zig-zag, slithering manner characteristic of myxobacteria and tends to form column or dome-shaped clusters of bacteria.

Diagnosis

Diagnosis should include dissection of the opercula of diseased eels to expose the gills and examination of the gills to determine if any destruction of the gill filaments has occurred, and also to see if yellow mucous clots of bacterial concentrations have formed. Furthermore, microscopic examination (using a magnification of 400x) of smears from gill lesions should reveal masses of bacteria forming columnar structures.

Disease Control Measures

Since the development of lesions is limited to the outer surfaces of the gills, diseased fish may be treated by dipping them in a medicated bath containing a nitrofurazone-type medicine. The pathogen of gill rot disease is very infectious, but at the same time it is quite sensitive to antibiotics. Furthermore, because the lesions are limited to the body surfaces, a preventive medicated bath is very effective. It is possible to prevent any occurrence of gill rot disease by treatment in a medicated bath when the fish are being transferred to grow-out ponds, provided that there is a separate water source available.

Figure IV - 1. The pathogen, Chondrococcus columnaris, has spread over the gills. Disintegration of the gill filaments is evident. The yellow areas are aggregations formed by the pathogens.

Figure IV - 2. The tip of the gill is missing. Bacterial aggregations have formed only along the missing part, and the rest of the gill is in relatively good condition. Although it cannot be seen clearly from the picture, the inner gill lamella has been attacked more severely than the external one. It is characteristic of gill rot disease that the inner gill lamellae are attacked more often than the external ones. This should be kept in mind when performing examinations.

Eels infected by gill rot disease either 1) suffer from sudden death as the result of rapid infestations of the gills by the pathogen (as seen in Photo 1), 2) recover from lesions which occur only on the tips of the gills (as seen in Photo 2), or 3) die later of complications from secondary causes. Which course infected eels will follow depends on their physiological condition and environmentally influenced variables. Also, it greatly depends on the virulence of the pathogen infecting the fish. Generally speaking, in eel grow-out ponds the latter course is more common than the former, causing small numbers of diseased fish to die every day.

(Text by Wakabayashi)

(Photographs by Egusa)

Photo 1

Photo 2

V. TAIL ROT DISEASE

(Columnaris Disease)

Tail rot disease is caused by the same pathogen as the one responsible for gill rot disease, Chondrococcus columnaris. The characteristic of tail rot disease is that lesions occur on the fins and the body, the tail being most susceptible to the infection. Tail rot disease, like gill rot disease, occur most often in summer when water temperatures are high, though its frequency of occurrence is much lower than that of gill rot disease. Outbreaks of tail rot disease are rare in ponds stocked with cultivated adult eels, but are more frequent in seed eel ponds.

In the initial stage of the infection, small yellow or yellowish white sticky spots appear on the edges of the fins and on the body surface. These expand gradually and develop into congestions around lesions and into necrosis of epithelial tissues. The fin rays, especially those of the caudal fin, disintegrate. This is followed by fraying and sloughing of the fins as the lesions advance to the anterior sections of the tail. Severe infections cause the muscles anterior to the tail to rot away exposing the bone structure. Tail rot disease, if developed in May, June, or October when water temperatures are relatively low, tends to be complicated by water fungi.

Etiology

The pathogen of tail rot disease is the myxobacterium, Chondrococcus columnaris, which can be identified relatively easily from its characteristic movements and formations of aggregations. Yellow viscous smears from lesions, examined using a 400x microscope, reveal innumerable long bacteria, 3 to 8 x 0.5 in size. One should notice that most of the bacteria secure themselves to a surface by one end and move their other end as though they were shaking their heads. After a short while, the innumerable bacteria gather together to form a dome or column-shaped colony.

Diagnosis

It is easy to diagnose tail rot disease from its characteristic external signs. It is recommended that

the existence of the pathogen be confirmed by microscopic examinations of smears from lesions of diseased fish in the first stages of the disease. Tail rot disease in its last stages, or when the eel is on the way to recovery, is sometimes difficult to distinguish from other infections such as those due to water fungi and in cases where the pathogens have disappeared. Because there are other similar myxobacteria apart from Chondrococcus columnaris in the grow-out ponds, isolation and culturing are necessary for accurate identification of the pathogen. For culturing of this type of bacterial agent, a special culture medium like cytophagus medium is required.

Measures for Disease Control

Same as for gill rot disease.

Figure V - 1. This is a relatively mild case of tail rot disease, yet the tail is rather extensively infected (the infected area is indicated by arrows).

Figure V - 2. This is a relatively advanced case. The tip of the tail has been sloughed off. Lesions are expanding and the area around the lesions is discolored (the discolored area is indicated by arrows). Bacterial aggregations can be seen on the second dorsal fin at the area around the lesions (arrows).

Figure V - 3. Lessions are localized along the dorsal fin. The tail tissue has rotted away exposing the muscles. Bacterial aggregations are not present, so the eel should recover unless a secondary infection, such as a water fungus, is contracted.

(Text by Wakabayashi)

(Pictures: Photo 1 & 3 by Egusa,
2 by Wakabayashi)

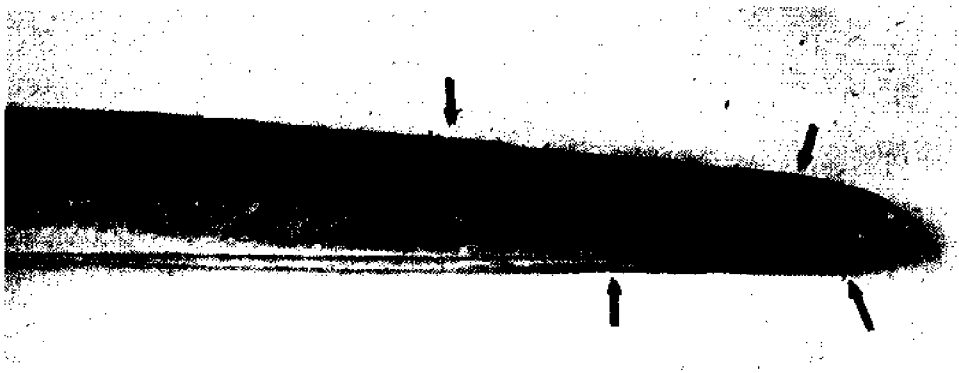

Photo 1

Photo 2

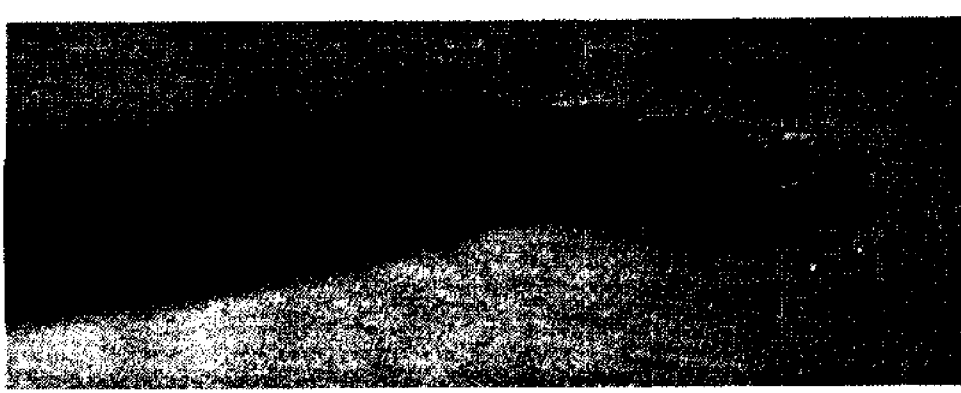

Photo 3

VI. MYXOBACTERIAL GILL DISEASE

Myxobacterial gill disease occurs frequently during periods of low water temperatures, especially in October and November as water temperatures drop. Characteristics of this disease include 1) extreme congestion or hemorrhaging of capillary vessels of the gill membranes, and 2) adhesion of the gill membranes producing clavated gill filaments or necrosis of the filaments. A myxobacterium can practically always be isolated from the gills of diseased fish, especially in the initial stage of the infection, and this myxobacterium is considered to be the pathogen of the disease. This pathogen is said to be identical to that which causes bacterial gill disease of salmon and trout in the United States because both of the pathogens possess the same characteristics. However, there are still problems in the classification of this type of myxobacteria, and its generic name has not yet been established.

Myxobacterial gill disease used to be included in the so-called "gill nephritis". It is now distinguished from the nephritis because congestion or hemorrhaging of the gills is excessive and treatment with brine dips is not effective. However, we still face the problem of distinguishing between chronic myxobacterial gill disease or a subsequent disease and critical "gill nephritis". Furthermore, even though examinations of living specimens or of tissue specimens suggest strongly that a myxobacterium is the pathogen, re-infections from the cultured pathogen are either difficult to obtain or the results of the re-infections are uncertain. Therefore, one has to conclude that the virulence of this particular bacterium depends strongly on its environment. This evidence has led some to regard myxobacterial gill disease as a secondary infection of gill nephritis. Further study of both of these diseases and their mutual relation is necessary.

Etiology

The pathogen of myxobacterial gill disease is a slender bacillus, about 5 x 0.5μ in size which has the slithering motion peculiar to myxobacteria. Compared with *Chondrococcus columnaris*, which is similar

and better known, the pathogenic bacterium of myxobacterial gill disease is of obviously different biochemical properties possessing a lower optimum temperature for growth and virulence. Other characteristics of this bacterium are 1) its length changes easily, being shorter in conditions of high temperatures or when found in tissues, 2) its slithering motion is more obvious, 3) it forms a thinly spread-out colony if cultured on a flat plate culture medium, and 4) when parasitizing the gills, it does not form conspicuous columnar aggregations. It infests the gills over their entire surface so that the aggregate formations are not visible to the naked eye while those of gill rot disease are quite apparent.

Diagnosis

Myxobacterial gill disease exhibits no discernible external disease signs. Lightly parasitized opercula, when cut open and examined, appear dark red, and the surfaces of heavily infested opercula are sticky because of hemorrhages. Examinations of smears from gill tissues using a 400x microscope often reveal slender bacteria moving in a way peculiar to myxobacteria, but finding these slender bacterial pathogens is more difficult than it is for gill rot disease. Moreover, the pathogen cannot be found in the chronic condition of myxobacterial gill disease. Therefore, diagnosis is difficult. Bacteriological or histological tests are essential for accurate diagnosis.

Disease Control Measures

The proper measures for controlling myxobacterial gill disease are not known due to a lack of knowledge about the causes of the disease. Both brine dips and administration of medicine are said to be ineffective. Tests performed by the (Japanese) authors of the susceptibility of cultured pathogens to about ten kinds of antibiotics showed that the bacteria were sensitive to tetracycline and especially to Nalidixic acid. They were either insensitive to all the rest or the degree of sensitivity varied a great deal depending on the particular bacterial strains used.

Figure VI - 1. The typical appearance of an infected gill. The entire gill looks dark red and is sticky. The mucous substance seen in the center is filth which is not directly related to myxobacterial gill disease.

Figure VI - 2. As seen in this picture, partial whitening of the gills probably occurs when the disease becomes chronic. This is often seen from January through March. However, most of the time, the pathogen cannot be isolated from these lesions, and attempts at reinfection using cultures have been unsuccessful. Thus, the diagnosis of the whitish gills presented here is still only conjectural.

(Text by Wakabayashi)

(Photographs by Wakabayashi)

Photo 1

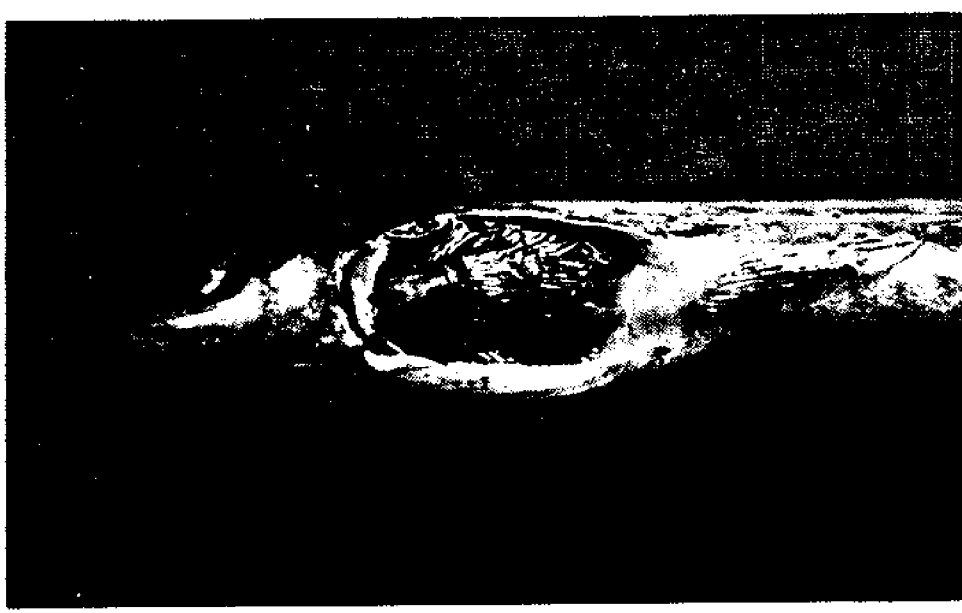

Photo 2

VII. COTTON-COVERED DISEASE (1)

(Seed eels, Adult eels)

(Water Fungal Disease, Aquatic Microbial Disease)

Rapidly developing outbreaks of cotton-covered disease occur mainly between the end of March and the early part of May and result in mass mortalities. Fungi in concentrated aggregations invade the head, tail, body, and gills. Explosive outbreaks of this disease take place several days after cold fronts from the Asian Continent pass by Japan causing the water temperature to drop sharply. Massive outbreaks can also occur during the coldest time of the year, i.e. January through March. In addition, this disease can be found after eels have been transferred to a new pond or after seed eels have been added in late fall and winter. In any case, this disease occurs during the season of low water temperatures and ceases to occur in May when water temperatures rise to close to 20°C.

Etiology

The parasitic fungus which causes cotton-covered disease is mainly the Phycomycete, Saprolegnia parasitica, or Saprolegnia sp. which is very similar to Saprolegnia parasitica but in which indications of sexual reproduction cannot be observed.

The disease involves hyphal invasion of the epidermis followed by penetration through the dermis into the muscles, and it causes necrosis and erosion of these tissues. At the same time, massive formations of hyphal infestation occur on the skin where germination and propagation of various fungal spores is observed. The optimum temperature for propagation of wandering spores is in range of 10 - 20°C, but propagation is feasible down to about 4°C. The reproductive activities of the spores decline in water above about 25°C, and the spores die above this temperature because they cannot compete with other microbes in the water.

Fungal infections result from the attachment of wandering spores to the skin and their subsequent germination. The spores invade the fish only after its

resistance has been lowered and it has been made vulnerable because of some other causes, or its local resistance to infection has been lowered because of abrasions, foci of infection on the skin, or some other pathological changes. Among the many possible primary causes, the infection by the bacterium, Aeromonas, is most important. This infection causes systemic weakness and makes germination of fungal spores on the body surface possible. Besides the Aeromonas infection, fasting in winter also contributes to systemic weakness. It is correct to assume that the vitality of eels is at the lowest at the beginning of spring. Sudden drops in the water temperature at the beginning of spring, which often happen, increase the vulnerability of eels to fungal infection. There are also cases where changes of water and administration of excess amounts of sulfa drugs have induced attacks of cotton covered disease. We suspect that there exists an infectious agent, which has not yet been identified, which causes local necrosis on the epidermis and reduces local resistence to infection.

Diagnosis

It is necessary to confirm the existence of fungus hyphae using smears taken from the filthy cotton-like lumps which stick to the head, tail, body, or gills. However, it is more important to determine the primary agent or factor which has preceded fungus germination. If examinations of sick eels found floundering near the surface or floating along the walls of their ponds reveal that some exhibit no fungus infection while many of them do exhibit signs of red fin disease, then the Aeromonas bacteria is probably the primary invader. On the other hand, if all the eels exhibit heavy fungus parasitization and there are hardly any signs of red fin disease, it is difficult to establish the primary disease agent. In this case, it is necessary to administer microbiological as well as histological tests on diseased fish in addition to considering environmental conditions which were present before the outbreak.

Disease Control Measures

Fungus is harmful, even as a secondary invader, because it damages the epidermis extensively and

causes local necrosis in skin and muscle tissues. Therefore, fungus pathogens need to be exterminated. In order to exterminate fungi, spray the pond with malachite green. The effective concentration is 0.2 ppm, but it should be more concentrated (for example, 0.5 - 1 ppm) for a pond containing a large amount of organic matter, such as a pond stocked with cultivated adult eels. When applying malachite green to a pond the use of a water agitator or a similar device is necessary because it is very dangerous to have the chemical unevenly distributed in the pond. The use of formalin spray at a concentration of approximately 15 ppm is also a recommended treatment for fungus. Spraying of these drugs may increase the mortalities among emaciated eels since those susceptible to cotton-covered disease have a low systemic resistence to begin with. Treatments for revitalizing the fish, such as raising the water temperature or the salinity of the pond (as in the case of gill nephritis) are also effective.

When red fin disease is the primary invader, we recommend the application of sulfa drugs, antibiotics, etc. (according to the manufacturer's directions) during a feeding period if the red fin disease and the feeding period coincide. As a prophylactic measure to control outbreaks of cotton-covered disease, reduce the amount of feed in early spring when the temperature changes drastically; and to control outbreaks of red fin disease, add a Furan agent to the feed for a while after the feeding period starts. An indirect but effective control measure is to refrain from adding seed eels after the summer season.

Figure VII - 1. The head of an eel infected with cotton-covered disease.

Figure VII - 2. Fungus hyphae.

Figure VII - 3. Photo 3 shows multiple germination of heavy-walled spores.

Figure VII - 4. Four examples of water fungus invasion of cotton-covered disease. The eel at the top exhibits signs of red fin disease and the water fungus has lightly invaded only the frontal part of the lower jaw.

Figure VII - 5. In the middle of May when water temperatures rise, the water fungi die and slough off. Ulcers can be seen on the mouth and on the side of the belly of the eel at the top and on the head of the eel in the middle. These are marks left after the water fungi, along with the necrosis and eroded skin, slough off. In the bottom example, water fungi attached to the pectoral fin and to the area above the fin are sloughing off.

(Text by Egusa)

(Photographs by Egusa)

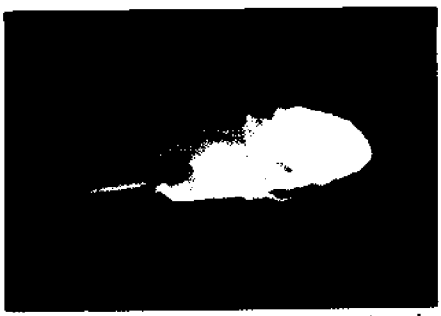

Photo 1

Photo 2

Photo 3

Photo 4

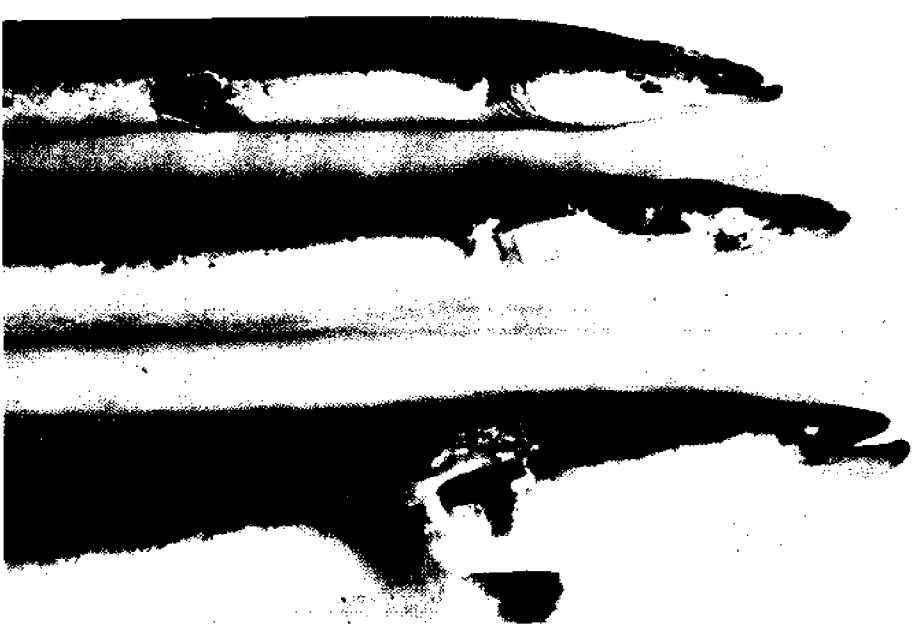

Photo 5

VIII. COTTON-COVERED DISEASE (2)

(Whitebait eels)

During the time that whitebait eels are being kept in ponds before the feeding season starts, or soon after the feed season begins, many of the whitebait eels may suddenly become covered with fungi on the body surface. It is common to see that whitebait eels, when kept in a low water temperature pond, become constantly infected with fungi in small numbers, and they become emaciated and die one after another. In the case of the eels sporadically infected with fungi, the lips are most often infected with the belly or the tail being the next parts affected. It is possible that mechanical abrasions precede fungus invasion, but usually some other microbial infections seem to precede the fungus. This is the usual situation, but the case described above at the beginning is different.

In the case of explosive outbreaks of cotton-covered disease in whitebait eels, fungi parasitize any place on the eel posterior to the middle of the body. Random examination of the diseased eels which are floating near the surface will reveal that while some fish do not exhibit fungus parasitization, they suffer from whitening and bleeding of the body. The bacterium, Aeromonas, will be isolated from almost all of the diseased eels including these fish. Because of this fact, Aeromonas infections are considered to be the primary cause of cotton-covered disease attacks. However, we have encountered an unusual case of an explosive outbreak of cotton-covered disease in whitebait eels in which almost all of the diseased fish were infected by fungi only on the breast (around the liver). In this particular case, the bacterium, Aeromonas, did not take part and the primary cause was undetermined.

Etiology

The invading fungus which causes cotton-covered disease in whitebait eels is the same fungus which causes cotton-covered disease in adult and seed eels. The pathogen is a Phycomycete fungus, either Saprolegnia parasitica or Saprolegnia sp., in which sexual reproduction is not observed. Unlike the case of

large-sized eels, hyphae invading under the skin of whitebait eels easily infect important organs (major blood vessels, heart, liver, spinal cord, brain, etc.). Once infected, whitebait eels will not recover naturally, and they will die sooner or later without exception. Thus, these fungi are very harmful to whitebait eels.

Diagnosis

In the initial stage, areas infected by fungus exhibit slightly raised white spots on the body surface. These white spots expand gradually, and begin to look as though bits of fluff were attached to the skin. No other disease exhibits this sort of abnormal sign, and it is easily recognized.

Disease Control Measures

Sprinkling the pond with malachite green is effective for exterminating the fungus. Since whitebait eels are sensitive to the toxin of this drug, it is important to keep the concentration of the drug at 0.2 ppm. When a bacterial Aeromonas infection is confirmed after the feeding season starts, administer a drug susceptibility test to the bacterium and then orally administer a chemotherapeutic drug which has been indicated as appropriate by the results of the test.

Figure VIII - 1. Whitebait eels suffering from fungus infections on the lips.

Figure VIII - 2. Whitebait eels suffering from fungus infections on the belly.

Figure VIII - 3. Whitebait eels taken at random from a pond in which an explosive outbreak of cotton-covered disease has occurred. The fish with severe fungal infections have been removed. There are those with practically no recognizable external abnormalities as well as those with whitening of a part of the body but with no sign of fungus growth. The body is usually slightly bent at the place at which whitening has occurred, and water fungi will invade that place. The Aeromonas bacterium was isolated from every one of these eels.

Figure VIII - 4. Whitebait eels from the unusual case, described in the text, in which the fungus attacked only the breast region of nearly all of the diseased fish in a pond.

(Text by Egusa)

(Photographs by Egusa)

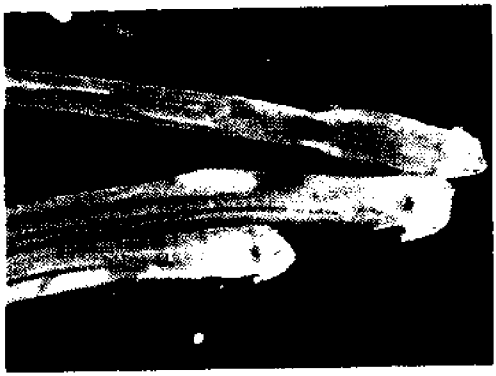

Photo 1

Photo 2

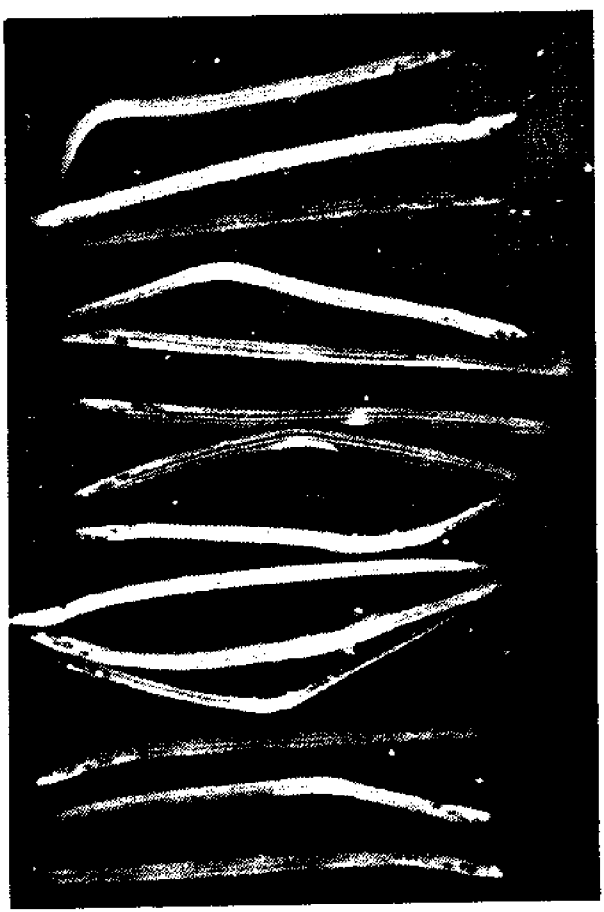

Photo 3

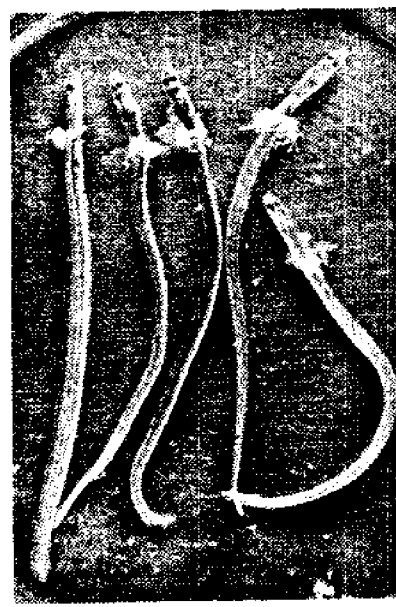

Photo 4

IX. TRICHODINA INFECTION

Trichodina infection is the result of parasitization by the protozoan Trichodina. It is the most well-known external parasitic disease of freshwater fishes, and it occurs on both warm and cold water species. There are many species in the genus Trichodina.

Parasitization is found among eels of all sizes, but it is more common among small eels in the whitebait phase and causes them great damage. The number of outbreaks of Trichodina infection in Shizuoka Prefecture makes it the most prevalent disease of whitebait eels in that Prefecture.

Trichodinids are found principally on the gills, the body surface, and the fins. They do not enter the body. Trichodinids attacking the gills cause gill tissue destruction and abnormal mucous secretion followed by difficulty in breathing which can be of serious consequence to the host fish.

Diseased fish do not usually exhibit severe abnormalities externally, but excessive secretion of mucous on the gills and the body are observed and the epidermis is hypertrophied and reddened by congestion in heavy infestations. In a serious condition, the tips of the gill filaments are missing and appear as if they had been worn off. Disease signs include poor appetite, reduced activity and emaciation. Naturally, diseased fish are very sensitive to lack of oxygen and to rough handling, and these can easily result in death.

Outbreaks of Trichodina infection are seen all year round, but they are most frequent in the early spring. Also, mixed infections with other parasites are often seen. The European eel, in contrast to the Japanese eel, is particularly susceptible to trichodinids.

Etiology

The pathogens of Trichodina infection are members of the peritrich genus Trichodina which is a ciliate protozoan. The pathogens are considered to be several in number, but have not yet been identified.

The pathogen is a most highly differentiated protozoan with a very complex structure. It looks like a perfect circle from underneath which is the abdominal side, and when seen edge-on looks like a hemisphere indented at the base. It is ciliated around the body. It moves about on the surfaces of the body and the gills of host fish. It is about 0.07 mm in diameter and 0.03 mm in height. Reproduction is mainly by binary fission, but sexual reproduction is also possible. Transmission from one eel to another is by direct methods such as contact.

Diagnosis

Diagnosis is done by microscopic examinations (using approximately 100x magnification) of smears of mucous from the body surface and from the gills of affected fish in order to confirm the existence of parasites. Generally, the parasites are actively moving around on the tissue surface using rotary motions of their cilia.

Disease Control Measures

Extermination of these parasites is relatively easy. The use of formalin for approximately 12 hours will generally accomplish complete extermination.

Figure IX - 1. A trichodinid (side view) inhabiting a gill filament is shaped like a hemisphere which is indented at the base and is ciliated around the body. By moving the cilia the parasite actively moves from location to location on the gills or body surface.

Figure IX - 2. A trichodinid viewed from the abdominal side. The orbicular rim is covered with cilia. The disc inside is an organ used to attach the parasite to the host fish. The disc has hooks arranged in a complex circular structure which is used as an identifying mark of the various species in the genus.

Figure IX - 3. Side view of Trichodina.

(Text and photographs: the Shizuoka Prefecture Fisheries Institute, Lake Hamana Station)

Photo 1

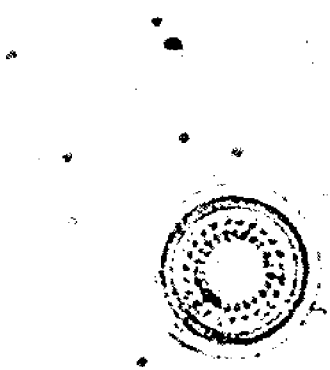

Photo 2

Photo 3

X. WHITE SPOT DISEASE

("Ich")

White spot disease is a parasitic infection caused by Ichthyophthirius, a ciliate protozoan. Infections on the host fish's skin look like white spots, thus the name white spot disease. This parasite is known to inhabit almost every species of freshwater fish, and every case of parasitization is called white spot disease.

The occurrence of white spot disease was virtually unknown in Japanese eels until the infection broke out in a high percentage of European whitebait eels which were imported to Japan for the first time in the spring of 1969. This incident drew new attention to the existence of white spot disease, and the proposition that white spot disease could have been brought into Japan with the European eels was presented. However, Japanese eels are not totally immune to this disease, and outbreaks followed by mass mortalities among slow growing larval eels have been reported. Other imported eels, such as American eels, A. rostrata, and Southeast Asian-bred eels, A. bicolor, are known to be very susceptible to white spot disease.

This disease is not usually fatal, but the problem this infection causes to the eel culture business is a reduction in the growth of eels due to the infection and its treatments (such as various medicated dips) which in turn bring about poor feeding.

Etiology

The pathogen of white spot disease is the ciliate protozoan Ichthyophthirius multifiliis. It is unicellular, and its form is originally egg-shaped; but as it moves, it becomes elongated or gourd shaped. The mature organism reaches 500 - 800μ in size and has a large transparent (often golden-looking) nucleus (usually horseshoe-shaped). A small nucleus, which appears to be attached to the large nucleus, also exists.

This ciliate reproduces by megasporangium formation. In order words, the mature trophozoite drops off the host into the water and attaches itself to the bottom of the pond or a floating object and becomes encysted. The multiple cellular divisions that follow produce between 200 and 1,000 larval ciliates. The larval ciliate is 30 - 45μ, egg-shaped, with a small protuberance, and covered with cilia just like the adult. Larval ciliates break out of the megasporangia and swim into the water. When they contact a fish, they penetrate beneath the epidermis and begin the parasitic stage of their life cycle where they remain until maturity.

Diagnosis

The lesions of white spot disease are located on parts of the body which make direct contact with the water, such as the skin, the gills, inner walls of the mouth, etc.

Generally, the pathogen is capable of moving freely on the outer surface or under the epidermis of the parts of the body which it invades. When an outer surface is involved, the damage the pathogen causes is slight. When it is under the epidermis of the skin or the inner walls of the mouth, the tissue becomes hypertrophied and a form of necrosis or hemorrhages are observed on the dermis beneath the infected area. However, when the gills are infected, neither hypertrophy of the gill epithelium nor any kind of necrosis or hemorrhages are observed.

Disease Control Measures

Various methods of controlling white spot have been tried for a long time, but a satisfactory method has not yet been developed.

Extermination measures generally used at present can be divided into two categories; 1) the use of drugs and 2) techniques which make use of the ecology of the pathogen. The first category is effective only for extermination of motile pathogens of the body surfaces of the hosts, and it includes methods which are applicable to grow-out ponds.

(1) Apply formalin to the pond at a concentration of 30 ppm. Repeat this treatment every 20 to 30 days.

(2) Apply methylene blue at a concentration of 3 ppm. Repeat this treatment three or four times at intervals of 3 or 4 days.

(3) Administer brine dips using a concentration of 5 to 7 ppm.

These three methods are not very effective during the low temperature seasons. In addition to these methods, a medicated bath of chloramine-T is said to be effective.

The method which makes use of the ecology of the pathogen consists of raising the water temperature to 26 to 30°C for 4 to 10 days to make the pathogen leave the host fish. This method is relatively safe, and it is also quite effective. In order to get rid of the released parasites, it is necessary to clean the walls and the bottom of the pond and replace the water.

Figure X - 1. Appearance of diseased eels (European eel fry, 6 to 7 cm in body length). The numerous white spots on the body are the places where white spot disease pathogens have invaded. From 2 to 10 individuals of the ciliate are often concentrated at one place under the skin of the host fish. (Due to this fact, the mode of reproduction was once considered to be binary division. This theory is no longer considered to be true.)

After the middle of June when water temperatures exceed 25°C, the pathogen automatically drops off the host eel and disappears.

Figure X - 2. The pathogen has invaded under the gill epithelium.

Figure X - 3. A microscopic photograph of a white spot pathogen removed from the skin. The organism propels itself by moving the cilia which cover its body. When it bumps into an object, it indents at the point of contact as seen in the photo. The object inside the cell wall which looks like a slightly bent nail is the large nucleus. The body is filled with innumerable granules.

(Text by Oka)

(Photographs: Photo 1 & 3 by Egusa, 2 by Oka)

Photo 1

Photo 2

Photo 3

XI. TRICHOPHRYA INFECTION

(Suctoria Parasitization)

Trichophrya infection occurs rarely in European eels, and it is not known to occur in Japanese eels. The infection is caused by very heavy infestations of the gills by a species of suctorian protozoan and is most prevalent in summer. Only eels smaller than middle-sized are subject to this infection, while large members of the species are apparently immune to the infection. The pathogen does not cause direct damage to the gills; but when the pathogens infest the entire surface of the gills, they cause breathing difficulty, poor appetite, and reduced activity. In addition, high water temperatures and reduced availability of oxygen in the water make the eels suffer from extreme breathing difficulty and cause them to behave frantically.

Etiology

The pathogen of Trichophrya infection is a species of Suctoria, Trichophrya sp. Eight species of Trichophrya have been reported in the world, but the species which parasitizes eels in Japan has not been identified. The pathogen is shaped like an eggplant, approximately 30-90 x 20-80 μ, and has 2 to 35 haustella (suction pipes) at the front like a tassel. Occasionally, it can also have a few haustella at the back end. The pathogen adheres to the gill epithelium with the side of its body. It moves about somewhat, like an amoeba. The suctorian sucks in microbes which are carried to it by water currents; these microbes are its main food. Some scientists believe that the suctorian also consumes the red blood corpuscles of the fish. Reproduction is performed by creating spores in the body of the suctorian and then ejecting the spores from the body.

Diagnosis

Remove a section of the gills, and mount a thin slice of gill tissue on a microscope slide using a cover glass. Perform a microscopic examination to confirm the existence of parasites which have a tassel of suction pipes.

Disease Control Measures

Due to the fact that there have not been many outbreaks of *Trichophrya* infection, we do not know the proper disease control measures. Use of formalin at a concentration of 30 ppm in the pond may be effective to a certain degree.

Figures XI - 1 & 2. Trichophrya attached to the gill, as seen by microscopic photos of sections of the gill.

Figures XI - 3. A specimen of gill tissue parasitized by many Trichophrya. The parasites have adhered to the surface of the gill epithelium. The large black spot in the center of the body is the large nucleus. The arrows point to trichodinids. Both species are often found together.

Figures XI - 4. Specimen of Trichophrya sp.. It is easy to recognize this species by the many suction pipes on the front of the body.

(Text by Egusa)

(Photographs: 1&2 by the Fisheries Institute of Shizuoka Prefecture, Lake Hamana Station; 3&4 by Egusa)

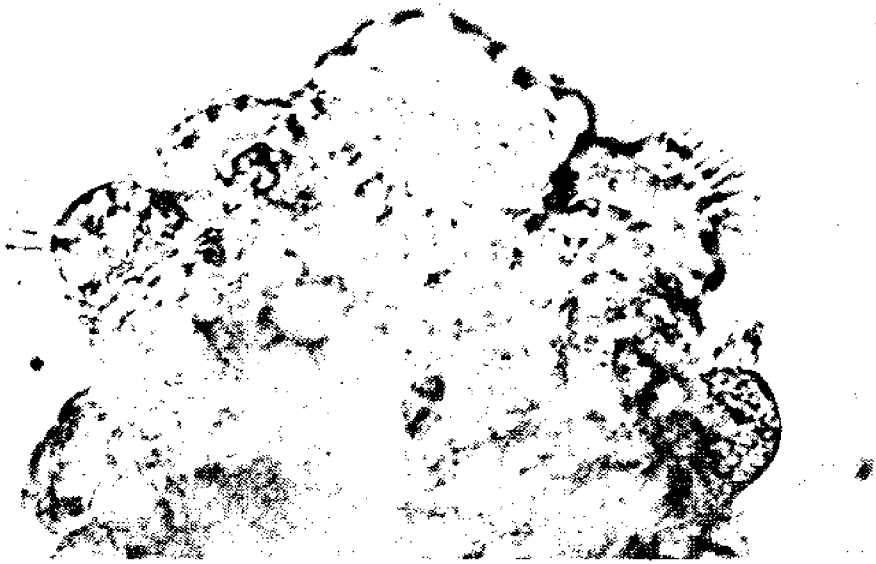

Photo 1

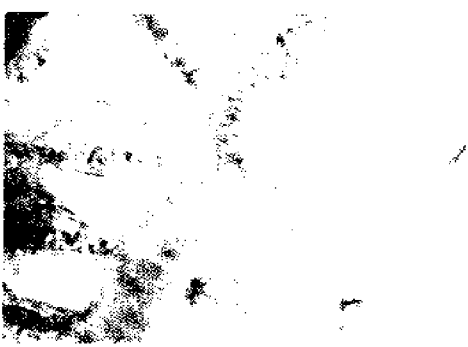

Photo 2

Photo 3

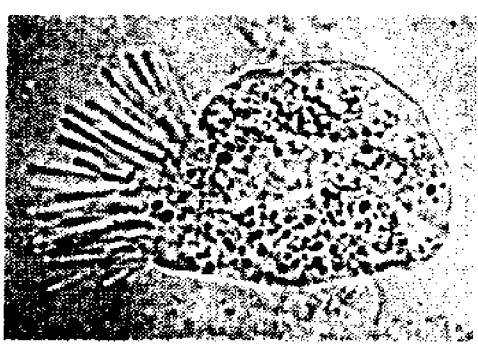

Photo 4

XII. MYXIDIUM DERMATITIS

(White Spot Disease)

Myxidium dermatitis is a parasitic disease whose causative agent is a myxosporidan protozoan of the genus Myxidium.

In early summer when the eels come to the feeding stations, some may be covered in a salt and pepper fashion with white spots as big as sesame seeds. If white-spotted eels are carefully observed every morning, you will notice that the white spots get bigger and also increase in number. Eventually the entire body except the head and the tail will become covered with the spots and faint internal hemorrhages will be observed as well. Subsequently, the white spots fade and disappear. The entire process takes about a month. The number of eels which exhibit these pathological changes is usually small, but a case in which every eel in a grow-out pond was infected by this form of dermatitis has been reported. This infection occurs most often from the middle of May to early August. The disease affects both Japanese and European eels.

Even though the Myxidium infection is most visible on the skin, it is known to infect both the gills and kidneys. Microscopic examinations at low magnification should reveal small lumpy sporocysts (which will be described later) on the gill epithelium. Kidney infections can be detected only by histological examination.

Etiology

Myxidium dermatitis is the result of parasitization by species of the genus Myxidium, of the Order Myxosporozoa, of the Class Sporoza of the Phylum Protozoa. Different species are said to attack different parts of the eel (i.e. kidney, skin, dorsal fins, or anal fin), but there are many morphological similarities in form and parasitic behavior among them. Even though it has not been previously reported, eels which suffer from Myxidium infection of the skin also suffer from infections in the gills and the kidneys without exception. Therefore, in spite of the common belief that different species of Myxidium attack different parts of the body, there is a possibility that they will all be classified as the same species.

Diagnosis

The morphology of dermal parasitization can be divided into a sporocyst formation type and pervasive and infiltration type. In either case, parasitization occurs in the tissue of the dermis (skin).

In the sporocyst type, soft and slightly raised, irregularly shaped, white or yellowish-white spots (about 2 mm in diameter) are observed on the skin. In the pervasive and infiltrate type, as in the former type, soft, slightly raised, but indistinct and slightly larger pale white spots (about 5 mm in diameter) can be observed.

Microscopic examinations of the milky white fluid obtained by breaking open either type of the spots should easily reveal spores.

When the spores reach maturity, the sporocysts automatically collapse, and the spores flow out into the dermal tissue. They then move up into the epidermis tissue and then onto the body surface. It takes about one month from the appearance of visible spots to the release of the spores. Very similar processes take place in the case of the pervasive and infiltration type.

Parasitization of the kidneys can be confirmed only by examining tissue speciments. The parasitization is of a muscle-invading type.

Disease Control Measures

As described above, Myxidium parasitization occurs in the gills, the kidneys, and the skin, but the infection of the gills and the kidneys cannot be detected from the outside.

The actual damages caused by Myxidium are not generally recognized as seriously pathological. Since the parasitization of the kidneys and the gills is slight and does not endanger the life of the eel, it can be neglected.

However, dermal parasitization, particularly when severe, ruins the appearance of the eels and lowers their market value. Even then, if the diseased eels are left alone in their grow-out ponds, the Myxidium spores will drop off the host eels about a month after the start of the infection. The marks of the infection will heal and become almost inconspicuous. In other words, one only needs to continue feeding as usual and the fish will heal by themselves.

Figure XII - 1. Eels with a Myxidium infection of the skin. White spots cover the entire body except for the head and tail regions. The infection is the sporocyst (morphological) type, and each white spot is a sporocyst in which countless spores are reproduced.

The eels pictured here are in the beginning stage of the infection. The white spots will increase in number, and at the peak of the infection they merge into one another.

Figure XII - 2. Myxidium spores. The Myxidium spore is oval-shaped with rather sharp points at both ends. It has two polar capsules inside, one on each end. It is covered with two layers of husks which have vertical striations. The spore is about 12μ long, and about 6μ wide; the polar capsule is about 3μ in diameter.

(Text by Oka)

(Photographs by Oka)

Photo 1

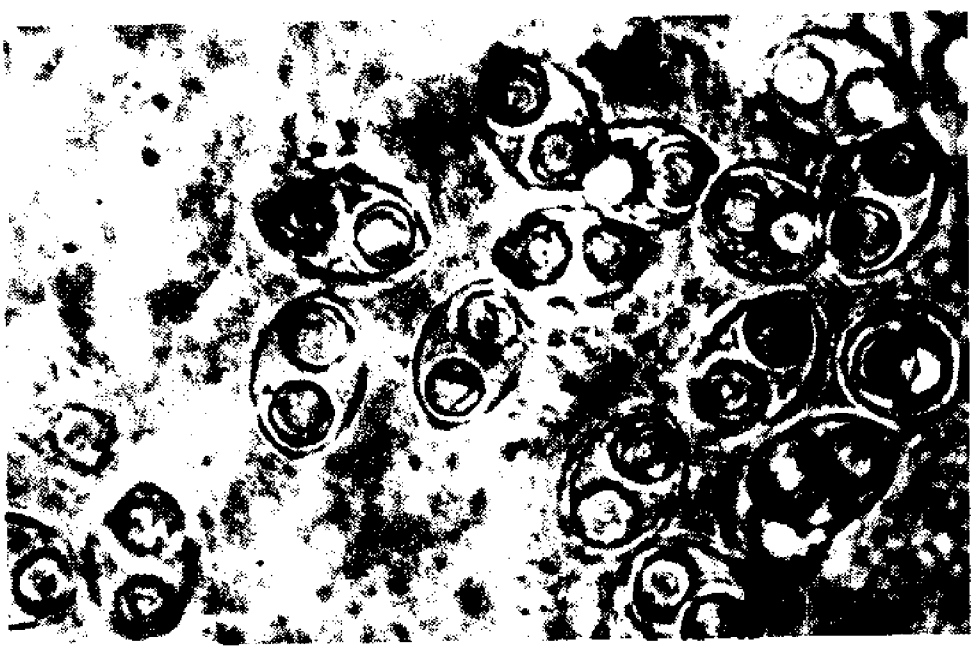

Photo 2

XIII. "BEKO" DISEASE

"Beko" disease has been known for a long time. It results from a microsporidan invasion of the body muscles. It was reported that "Beko" disease caused great damage in eel grow-out ponds in Yoshida, Shizuoka Prefecture during the years of 1936 and 1938. Damage from this disease has declined in recent years. However, there are signs that the occurrence of this disease may rise as the importation of seed eels from foreign countries has increased, and eel culture in warm water has become more widespread in the past two to three years. Generally, the rate of outbreaks and the damage caused by this disease are not high. Seed eels weighing from 1 to 20 grams are most susceptible to the infection.

This disease can be recognized by conspicuous bulges on the body of the host, with hard, convex bumps and soft, weak concave depressions of the surface. Dissection of the soft and weak pockets reveals a thick milky-white fluid formed by disintegration and liquefaction of the muscle tissue. When the disease first attacks smaller eels, a part of the muscles becomes white-spotted in appearance; when the disease is in an advanced stage, the entire body will be covered with milky yellow spots. Diseased fish live for a fairly long time, eat well, and seldom suffer a sudden death. However, once diseased, the fish will not recover by themselves, and eventually emaciation caused by infection of all the body muscles will result in death.

The time of prevalence for the disease is not limited to any particular season. It commonly begins in spring when the water temperatures rise, and it continues through the fall. Also, it generally tends to break out earlier in warm areas and in grow-out ponds where warm water eel culture is practiced.

Etiology

"Beko" disease is caused by Plistophora anguillarum, a species of the Microsporidia group of parasitic protozoa.

The pathogen forms cysts of various sizes in the muscle fibers. The cysts are either round or oval, are 28 to 195u by 26 to 117u in size and contain many sporo-blastocysts which in turn contain spores. The spores are egg-shaped with one slightly concave side, and are 6 to 10u by 3 to 5u in size.

Diagnosis

It is easy to make a diagnosis from external examinations because diseased eels exhibit irregular bumps on the body surface. A further microscopic examination (using a magnification of 100 to 250x) of smears taken from muscles or from the milky-white liquefied muscles found in pockets on the body should confirm the existence of sporozoans.

Disease Control Measures

There is no appropriate treatment for "Beko" disease at the present time. It is important to remove and dispose of diseased eels as soon as they are found during harvesting or selection in order to prevent further spreading of the disease. Furthermore, it is desirable to disinfect the ponds because it is possible that some sporozoans will survive in the water and mud to infect eels newly released into the ponds. "Kalk" (bleaching powder) is recommended as the disinfectant agent.

Figure XIII - 1. Diseased European eels. The body surfaces are bumpy and have turned white.

Figure XIII - 2. Diseased eels (13 to 15 cm in body length). The white parts of the bodies are parasitized by microsporidans. Bulges are not yet visible.

Figure XIII - 3. The bumpy body surface of a diseased eel. The protruding parts are hard and the caved-in sections are soft and weak. Dissection of the pockets reveals a thick, milky white fluid formed by destruction and liquefaction of the muscular system.

Figure XIII - 4. Sporozoans (200x magnification) extracted from a lesion. The spore is egg-shaped, with one side slightly caved in. Living spores have the saturated front and the caudal vacuole with 3 or 4 horizontal lines across the inside. There is no polar capsule. In the upper right corner of the picture sporoblastocysts containing several spores are seen.

(Text by the Fisheries Institute of Shizuoka Prefecture, Lake Hamana Station)

(Photographs: 1&4 by same as above, 2&3 by Egusa)

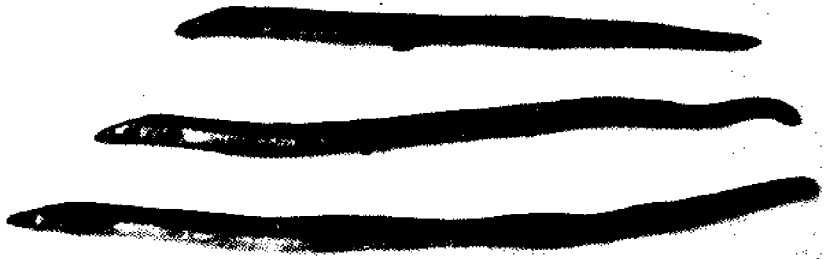

Photo 1

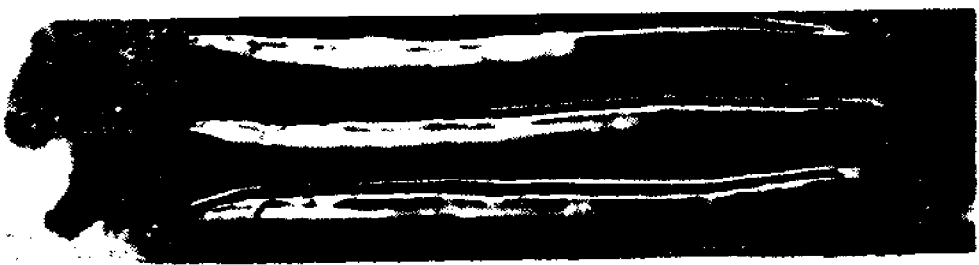

Photo 2

Photo 3

Photo 4

XIV. CONNECTED HOLE DISEASE

(Hole making disease)

Connected hole disease is a strange disease that once broke out explosively among adult eels in some of the eel growing ponds in Yaizu City which used rivers as their water source. The following year saw sporadic outbreaks of the disease, but no occurrence was seen after the third year. This infection has not been reported in any other localities. Therefore, it may not be of great concern, but we will describe major aspects of the disease here.

The body surfaces of eels suffering from connected hole disease exhibit many various-sized ulcers which are bright red on the bottom. The eels look so grotesque that they are not marketable. The ulcers penetrate deep into the muscles and form connective holes which contain pink fluid. Several ulcers are often connected by these holes.

Connected hole disease infected mostly adult eels and very seldom did it infect seed eels. Its outbreaks are most frequent in summer. Eels infected in the summer became thin; but after the fall, their ulcers healed naturally and eel moralities seemed to be low. However, there were many eels which suffered simultaneously from red fin disease caused by the bacterium, Aeromonas, or from Paracolo disease caused by Edwardsiella bacteria, and most of these eels died. There were also quite a few diseased eels which exhibited the sign (irregular bumps on the body) of "Beko" disease.

Etiology

The causes of connected hole disease are unknown. Microsporidans which looked like Plistophora, Aeromonas and Edwardsiella bacteria, and actinomycetes (fungi) were often discovered in the foci of infection, but none of these were consistently found in most of the foci. We reached the tentative conclusion that a microsporidan was the primary agent; and with other microbes as secondary agents, it caused the peculiar pathological changes described here.

Diagnosis

One glance at its outwardly apparent disease signs is enough to diagnose connected hole disease. By slicing the body near the surface lesions, one can easily observe either pink or red connected holes leading deep into the body.

Figure XIV - 1. Eels afflicted with connected hole disease. The eel in the center exhibits reddening of the dorsal fin from a complication with red fin disease. The eel at the bottom, on the other hand, suffers both from connected hole disease and Paracolo disease.

Figure XIV - 2. Cross sections of tails sliced from eels infected with connected hole disease. The lesions have penetrated into the deep layers of the muscles and are connected to some of the ulcers on the body surface.

Figure XIV - 3. Eels acquired in late fall which are on the way to a natural recovery. The redness of the ulcers has faded into a flesh tint and the surfaces of the ulcers are covered with hypoderma. Bumps as seen in "Beko" disease are often observed. Generally, eels that are infected by connected hole disease in summer become very thin, as in this photograph.

(Text by Egusa)

(Photographs by Egusa)

Photo 1

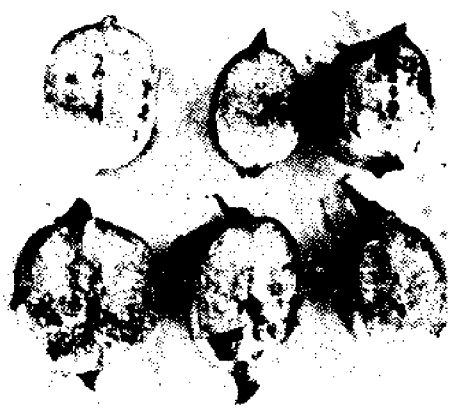

Photo 2

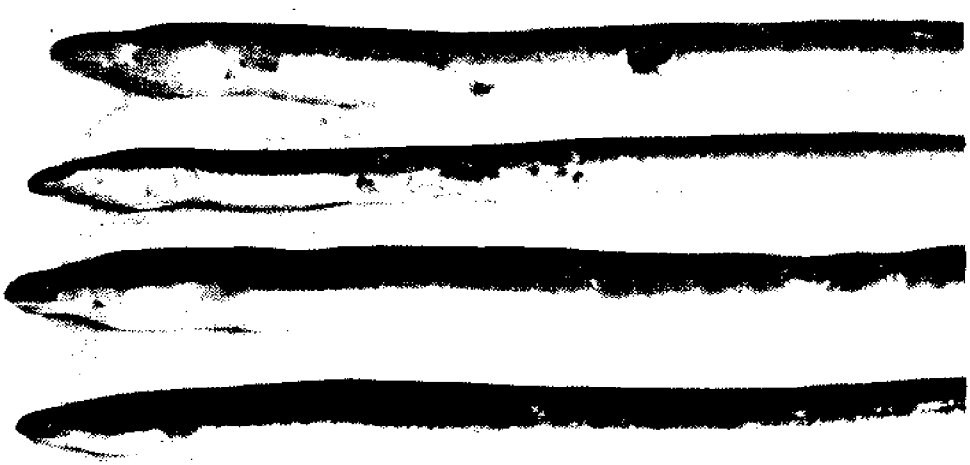

Photo 3

XV. DACTYLOGYRUS INFECTION

Dactylogyrus infection is the result of parasitization by the trematode, Dactylogyrus and has been known for a long time. In the case of Japanese eels, this parasite attacks cultivated adult eels rather than seed eels, and damage has not been extensive. European eels, on the other hand, are highly susceptible to the parasite, and suffer grave consequences if infected. This parasitization is considered to be directly related to the abnormal mass mortalities of European eels in summer.

Dactylogyrus usually attacks the gills, and diseased eels do not exhibit severe external pathological changes. However, heavy infestations cause excess secretion of mucous on the gills and damage to gill tissue. Affected fish show poor appetite, refusal to eat, reduced activity, and emaciation.

The disease is most prevalent in summer when water temperatures rise, and mixed infections involving other species of parasites are often observed.

Etiology

The parasitic pathogen of Dactylogyrus infection is either the trematode, Dactylogyrus, or a very similar platyhelminth, Neodactylogyrus.

The pathogen is about 0.4 to 0.8 mm in length, and has four spikes on the front of its head plus two pairs of eyes. There are no morphological differences between males and females. The parasite is oviparous, and the eggs fall to the pond bottom after they are released. The newly hatched larvae swim about and find hosts. In summer, several days are required for the eggs to hatch. The larvae attain maturity in about a week, and they then begin to lay eggs.

Diagnosis

Diagnosis consists of microscopic examinations (using 100x magnification) to confirm the existence of the parasite from smears taken from mucous found on the body surface and on the gills. The smears should

reveal parasites which are actively expanding and contracting the body while keeping the posterior disk securely attached to the tissue of the fish. Morphologically, Dactylogyrus resembels Gyrodactylus very closely. One of the key ways in which they can be distinguished is that the former has two pairs of eyes.

Disease Control Measures

Immersion of the fish in a medicated bath containing trichloroethyl alcohol at a concentration of 0.3 to 0.5 ppm for over 48 hours is effective for exterminating the pathogen, and this method is widely used. It is recommended that European eels be given such a medicated bath regularly once every 15 days to prevent them from contracting Dactylogyrus disease since they are very susceptible to it.

Figure XV - 1. Dactylogyrus parasitizing the gill. It is secured firmly to the gill tissue by the posterior disk and engages in movements of expansion and contraction. A recently laid egg (←) is seen at the tip of the gill.

Figure XV - 2. Dactylogyrus has a long oval shape with two pairs of eyes in the head. There are 14 valves on the rim around the posterior disk; at the center of the disk, there is a pair of sickle-shaped hooks by which Dactylogyrus firmly attaches itself to the host fish.

(Text by the Lake Hamana Station, Fisheries Institute of Shizuoka Prefecture)

(Photographs: 1 by the same above, 2 by Egusa)

Photo 1

Photo 2

XVI. GYRODACTYLUS INFECTION

Gyrodactylus infection is caused by the parasitic trematode, Gyrodactylus. Although it has been known for a long time, there have been few occurrences which have caused severe damage.

This trematode parasitizes the gills and the body surfaces. Diseased eels do not exhibit severe external pathological changes. Heavy infestations cause damage to gill tissue and excess secretion of mucous on the gills and the body surface. Affected fish show poor appetite, refusal to eat, reduced activity, and emaciation.

The disease is most prevalent in summer when water temperatures rise, and mixed infections involving other parasites are often observed.

Etiology

Gyrodactylus infection is caused by the platyhelminth trematode, Gyrodactylus. The trematode is long and oval in shape, with two cone-shaped spikes at the front end where the opening of the adhesive gland is located. The back part of the body is shaped like a disk with two sickle-shaped hooks in the center, and a small hook at each of 16 valves around the disk. The parasite is 0.2 to 0.8 mm in length, and it is bisexual and viviparous. An embryo is formed inside the body in the first and second generations of this worm, and this is why it is sometimes called the third generation worm. The parasite dies within several hours after it drops off its host.

The method of transmission is by contact between eels. The parasite first adheres to the host using mucous from the adhesive gland on its head, and it later firmly attaches itself in tissue of the host eel by using both the hooks located on the small valves and those in the center of the posterior disk.

Diagnosis

Diagnosis is done by microscopic examination (using 100x magnification) of smears of mucous from

the body surface and from the gills of diseased fish in order to confirm the existence of the parasite. The examination should reveal the parasite actively engaged in expansion and contraction movements while keeping its rear disk secured into the tissue of the host fish.

Disease Control Measures

Administration of a medicated bath containing trichloroethyl alcohol at a concentration of 0.3 to 0.5 ppm for longer than 48 hours is effective for extermination of the pathogens.

Figure XVI - 1. The tremotode, Gyrodactylus, infesting the gills. With its rear disk firmly secured into the gill tissue, the parasite is engaged in contraction and expansion movements.

Figure XVI - 2. Gyrodactylus is long and flat, and it has a disk at the rear end. There are 16 small petal-like hooks around the disk and two large hooks inside. It attaches itself firmly to the host using the rear disk. The head is located at the front extremity and consists of two cone-shaped protuburances, between which the opening of a mucous gland is situated. In the center of the body is an embryo.

(Text and Photographs by the Fisheries Institute of Shizuoka Prefecture, Lake Hamana Station)

Photo 1

Photo 2

XVII. ANGUILLICOLA DISEASE

(Air Bladder Nematode Parasitization)

Anguillicola disease is caused by an extremely heavy infestation of the air bladder by the nematode, Anguillicola. This abnormality is seen mainly in European eels. It has been known for a long time that this worm attacks Japanese eels in their natural habitats as well as in grow-out ponds. However, this parasitic disease has not attracted much attention due to the fact that although heavy infestations may cause inflammation of the air baldder they seldom disrupt the eels' normal activities. Compared to Japanese eels, the European species is much more susceptible to this worm, and they suffer from a higher rate of infection and larger numbers of worms per eel. If heavily infested from the fall through the summer of the first year of whitebait cultivation, young eels may not only cease eating and become emaciated, but they may even die.

The type of nematode parasitization which has most serious consequences for the host occurs when worms grow up and completely fill the air bladder making it enormously overblown whereupon it will press against other internal organs, blood vessels, etc. Compared to these effects, bad inflammation or bleeding of the air bladder and the air ducts caused by usual heavy infestations are not as serious. When enlargement of the air bladder is extreme, the lower abdomen swells and the belly sometimes exhibits an irregular contour. Reddening of the belly skin due to congestion is occasionally seen. In addition, the anal zone stretches and turns extremely red. In an extreme case (especially when the nematodes have reproduced in the eel) the rear end of the air bladder bursts open and worms spill out into the lower abdominal cavity. They may then be pushed out of the eel through the anus or the opening of the urethra.

Etiology

The pathogen of Anguillicola disease is a species of nematode, Anguillicola grobiceps. Both sexes parasitize eels and feed on eel blood. The female is

larger than the male and may grow to be 7 cm long. The male does not grow longer than a little over 4 cm. The smallest visible worms are about 2 mm long. Usually several worms (up to 10) are found in a host eel. In extreme cases, this number may be greater than 20 worms per eel.

Reproduction occurs during the entire year. A large number of larvae are born and are released into the air bladder of the host eel after the uterus and body of the parent worm break open. The digestive tract of the mother worm is also torn when it gives birth, and the chocolate-colored contents of the tract flow out. A small number of the larvae penetrate into the gill tissue in which they mature, but the rest of them move into the water by way of the air duct and the digestive tract. Their first intermediate hosts are probably some sort of aquatic animals that will be eaten by eels, which will in turn become infected. This method of transmission has not, however, been firmly established.

Diagnosis

Only one species of air bladder parasitizing nematode is known in Japan. If many nematodes which look like those shown in the pictures on the following page are found when dissecting the air bladder, the illness should be diagnosed as Anguillicola disease.

Disease Control Measures

No effective treatment for this disease exists at present. Since the complete life cycle of Anguillicola is still unknown, no appropriate prophylactic measures are available. Since eel infection by the larval phase is considered crucial, the pond water should be replaced and the pond bottom should be disinfected by appropriate cleaning agent such as "Kalk" (bleach powder) when using a pond which has contained eels infected with Anguillicola parasites. When purchasing seed eels, it is necessary to inspect them to see whether or not they are carrying Anguillicola parasites.

Figure XVII - 1. The nematode, Anguillicola grobiceps, inside the air bladder.

Figure XVII - 2. A dissected air bladder after the nematodes have reproduced in it. The chocolate-colored liquid has flowed out of the nematode digestive tracts. This substance consists mainly of digested red corpuscles of eel blood. A large number of larvae are present in this chocolate-colored liquid.

Figure XVII - 3. There were 18 to 28 worms in the air bladders of these European eels. Some of the worms were fully grown. The eels' air bladders were enormously swollen. Swelling of the belly and reddening are seen. The anal zone is distended and has turned red. A nematode can be seen coming out of the anus of the center eel.

(Text and Photographs by Egusa)

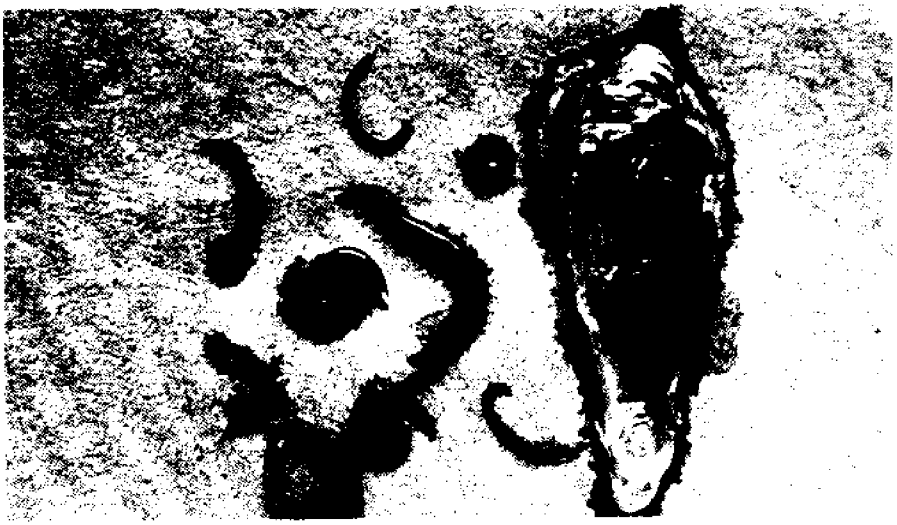

Photo 1

Photo 2

Photo 3

XVIII. ANCHOR WORM INFECTION

Anchor worm infection has been known longer than any other eel disease. Its occurrence in eel grow-out ponds and its damage to eels were reported as early as the Taisho era (1910 to 1925).

Anchor worms emerge and reproduce in eel ponds from April through November with summer being the time of peak activity. Only a few of the adult anchor worms survive the winter conditions.

Parasitization by anchor worms alone seldom causes mass mortalities of eels. However, heavy infestations make eels vulnerable to other infections or reduce their reproductive capacity, and these may result in chronic eel mortalities.

The anchor worm inhabits only the mouth cavity. Heavy infestations prevent the fish from closing its mouth, and force the mouth to remain open. Another characteristic of this mouth parasite is that the front end of the worm penetrates deeply into the muscular tissue of the lower jaw and often causes hemorrhages inside the lower jaw.

Parasitization by anchor worms reduces the appetite for a long time and causes the eel to lose weight. In addition, the infection coincides with the period when the eels are growing the most. Therefore, the economic losses caused by this infection are significant.

Anchor worms tend to attack market-sized or nearly market-sized eels. Seed eels are very rarely affected.

Etiology

The parasitic pathogen of anchor worm infection is the copepod, Lernaea cyprinacea. In early spring, the female copepods, which have been hibernating, mature and lay their eggs. After the first egg laying, generations are repeated four or five times, and some females from the fourth and fifth generations become the next winter generation in late autumn. Only the female copepods are parasitic. They have two egg pouches located at the posterior end of the body.

The larvae swim freely in the water after hatching and mature by a process of repeated ecdysis (molting). As they grow, they become dependent on fish, and they eventually attach to the body surface of a fish. At this time, the female and male copepods mate. After mating the males die and the females penetrate the tissues of the host fish. Approximately three weeks at a temperature of 20°C, 10 days at 27°C, are required to proceed from hatching to the stage of parasitic attachment and infestation. The number of eggs per reproduction averages 360, and the female breeds more than 10 times during her lifetime.

Diagnosis

Open or cut open the mouth in order to confirm an invasion of the copepod.

Disease Control Measures

Early detection and early treatment are most important for controlling and exterminating the copepod. The most widely used method is to spray trichloroethyl alcohol in the pond. However, trichloroethyl alcohol is effective only for larvae; it has no effect on adult copepods. Thus, in order to use this method, the times of spraying must be synchronized with the appropriate stages in the life cycle of the copepods.

Generally, trichloroethyl alcohol is used in the amount of 200 to 500 grams per 1,000 meters by 1 meter depth of pond water volume (which corresponds to a concentration of 0.2 to 0.5 ppm). It should be applied 3 or 4 times at intervals of one week when larvae are present. The most effective measure is to spray the drug 2 or 3 times during March and April when the first generation of eggs begin to hatch.

Figure XVIII - 1. A heavy infestation of anchor worms which are 5 to 9 mm in size is visible to the naked eye. The copepod is shaped like a thin stick which has, as its name indicates, an anchor-like front end which penetrates deeply into the tissue of the host fish.

Figure XVIII - 2. A heavy infestation of anchor worms makes the eel unable to close its mouth. The eel cannot eat and becomes emaciated. Secondary infections often occur in areas infected by anchor worms.

(Text and photographs by Ohue)

Photo 1

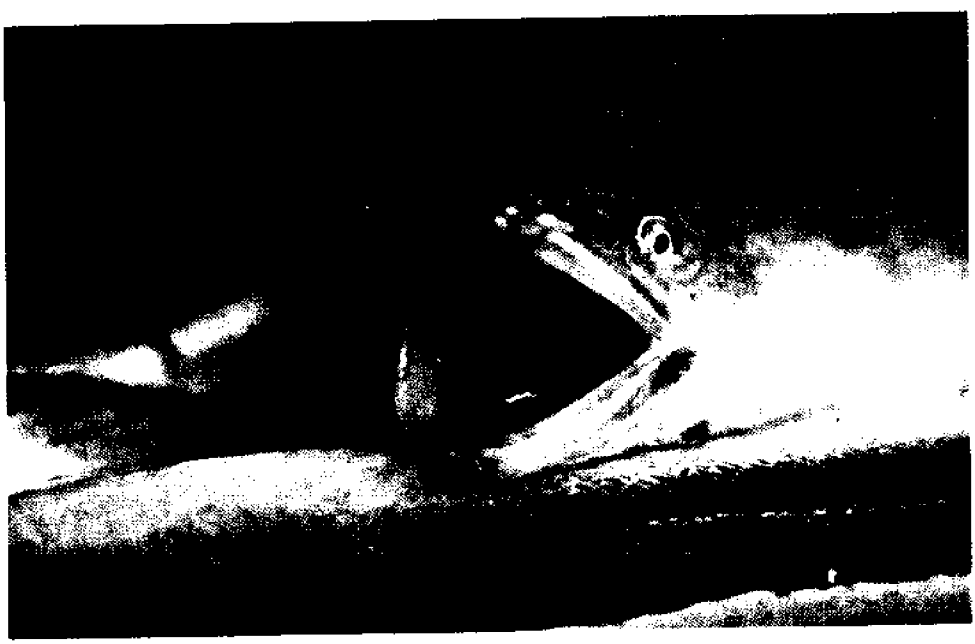

Photo 2

XIX. GILL NEPHRITIS

(Gill - Kidney Inflammation)

Gill nephritis occurred repeatedly in the Yoshida, Yaizu, and Ohigawa areas of Shizuoka Prefecture during the winters of 1969-70 and 1970-71, and caused extensive mortalities. Because the characteristic pathological changes affecting the eels were on the gills and in the kidneys, this disease was named "gill nephritis".

The etiology of this disease has not yet been determined, but the name "gill nephritis", without being precisely defined, has come to be widely used. One of the characteristics of this disease is that it has no apparent visible disease sign. Since this makes detection difficult, any disease of undetermined origin tends to be diagnosed as gill nephritis. In addition, the best known characteristic of gill nephritis (pathological changes in the gills, e.g. multiplication of the epithelial cells, deformation, hypertrophy and adhesion of the gill epithelia, and adhesions of the gill filaments which eventually develop into club-like masses) are not limited to gill nephritis. These symptoms can also be observed in cotton-capped disease, and they almost always exist in abdominal dropsy and myxobacterial gill diseases. Also, gill nephritis often emerges when small amounts of dissolved heavy metals or other harmful substances are present in the water. Abnormalities of the kidneys, such as pathological changes of epithelial cells of kidney tubules which makes the cells appear like glassy drops, can also be seen in other infections. Another characteristic of gill nephritis which can also be observed in other diseases is an extreme reduction of the volume of chlorine ions in the blood plasma. Whether gill nephritis should be established as a separate entity or not is not clearly determined at this time, and further study of the disease will be necessary to answer this question.

The definition of gill nephritis at this time is as follows. It is a disease which occurs after eels abstain from eating for a period of time in the winter. Characterization of this disease includes multiplication of the epithelial cells of the gills, deformation, hypertrophy and adhesion of the gill epithelia, which finally result in the gill filaments become club-shaped.

Disease signs which are found in the kidneys include the emergence and enlargement of drops of a protein-like substance in the kidney glomerulus, detachment of the epithelial cells and the emergency of a protein-like substance in the Bowman's capsules, and collapse and bleeding in the glomerulus. The pathological changes in the kidney tubules are dropsy, turbidity and hypertrophy of the epithelial cells, and the epithelial cells exhibit a marked glassy, drop-like appearance in advanced cases. These glassy drop-like changes are quite prominent in seriously diseased fish. No pronounced abnormality exits in the viscera, the skin, or the fins. None of the identified bacteria, fungi, or parasites are the pathogens of gill nephritis. Infectiousness of the disease has not been determined.

Etiology

The etiology of gill nephritis is not known at the present time, though environmental variables and metabolic disorders are suspected to be closely related to the causes of the disease.

There is a myxobacterium known to cause similar pathological changes in the gills, but this infection has been established as "myxobacterial gill disease", a separate entity. Recently, a virus was discovered in infections of gill nephritis and in other similar infections, and this has brought up the possibility that gill nephritis is a viral infection.

Diagnosis

The only visible disease sign is reddening of the skin and the gills. Dissection does not reveal any conspicuous abnormalities. However, close examination of the gills reveals that the gill filaments do not form the regular, radiating pattern of the filaments of healthy fish. Many of the gill filaments of diseased fish are crooked and appear swollen. Low-power microscopic examinations of dissections from the gill lamellae will reveal irregularly spaced, hypertrophied, crooked or adhered gill epithelia.

In a healthy fish, these would be lined up regularly like the teeth of a comb. Advanced pathological cases exhibit an overall adhesion of the gill epithelia which results in deformation of the gill filaments into club-like shapes. Judgement about the presence and seriousness of pathological changes of the glomerulus and the tubules in the kidneys must depend on tissue examinations. Also, it is necessary to confirm that the infection being diagnosed is not caused by any of the identified parasites, fungi, or bacteria.

Disease Control Measures

Neither prophylaxis nor treatment has been established since the etiology of gill nephritis is still unknown. Experiments have proven that salt in a concentration of 0.5 to 0.8% in the pond water is effective for stopping eel mortalities and for curing the disease, but why this brine treatment is effective is still a puzzle.

Figure XIX - 1. Gills of fish infected by gill nephritis. The gills appear dark red because of congestion and the gill filaments are crooked and swollen. The orderly, radiating pattern of the gill filaments of a healthy fish is no longer observed. The white spots seen on the gill of the top eel in this picture are cysts caused by sporozoans and are unrelated to gill nephritis.

Figure XIX - 2&3. Low power microscopic pictures of a section of a branchial leaf. The tips of the gill epithelia are hypertrophied and adhesions have occurred in several places (Fig. 2). The gill filaments have been completely deformed into a club-like shape, as is shown in Figure 3.

Figure XIX - 4,5&6. Tissue specimens which show a process of multiplication of epithelial cells and of deformation of the adhered gill filaments into a club-like shape.

Figure XIX - 7&8. Tissue specimens showing the advancement of the pathological changes, which look like glassy drops, in the kidney tubules.

(Text by Egusa)

(Photographs by Egusa)

Photo 1

Photo 2

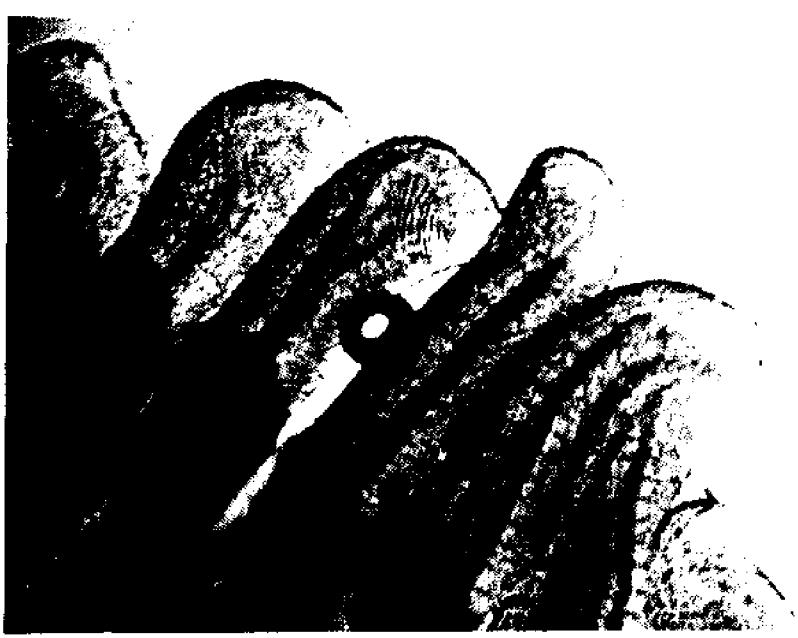

Photo 3

Photo 4

Photo 5

Photo 6

Photo 7

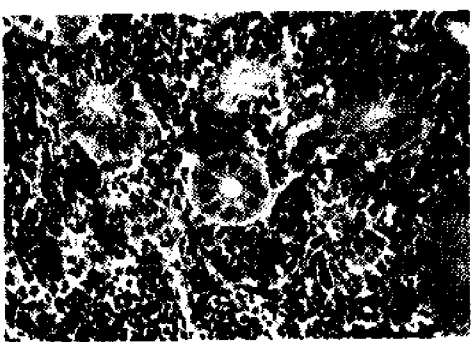

Photo 8

XX. ABDOMINAL DROPSY DISEASE

Abdominal dropsy disease is an infection that produces a large amount of serous fluid which accumulates in the abdominal cavity and causes the belly to swell. Initially, a colorless aqueous liquid accumulates; but gradually the fluid increases in its viscosity, and it solidifies immediately after the belly is opened in a laparotomy. Further, the aqueous humor gradually becomes blue-green and solidifies into a jelly in the abdominal cavity. Affected fish stop eating and swim slowly near the surface of the pond. Generally, mortalities are low and the affected fish recover naturally some time after they stop eating.

Occasionally, an Aeromonas infection occurs simultaneously with abdominal dropsy disease, and the resultant rate of mortality is quite high.

Abdominal dropsy disease affects all sizes of eels from whitebait to adult eels. Outbreaks are seen mainly in the spring and the fall.

Etiology

The cause of abdominal dropsy disease is not known at this time. Possible causes for the accumulation of abdominal dropsy are malfunctions of the kidneys and circulatory disorders, but the external agents which bring about these abnormalities have not been identified. Because many eels in the same pond may contract abdominal dropsy disease, it is suspected to be infectious. However, the infectiousness has not been established. Poisoning by some toxic substance is another possible cause.

Diagnosis

It is easy to diagnose abdominal dropsy disease by dissections since no other disease manifests similar symptoms. Every affected eel exhibits a swollen belly which is soft and from which an aqueous fluid flows

when it is cut open. This fluid immediately solidifies into jelly in some cases. Eels suffering from advanced cases of abdominal dropsy disease exhibit bluish abdominal walls externally, and internally the abdominal cavity is filled with a bluish-green jelly. Affected eels which have developed a concurrent bacterial Aeromonas infection will exhibit hemorrhages on the body surface and on the walls of the abdominal cavity, and they will have reddened intestines.

Disease Control Measures

Appropriate prophylaxis and treatment are not available because the causes of the disease are undetermined. Red fin disease, which often accompanies abdominal dropsy, should be treated if it is observed.

Figure XX - 1. Two examples of eels affected by abdominal dropsy disease. Abdominal dropsy has not accumulated too much in the eel at the top. Red spots caused by an Aeromonas infection are observed on the belly skin.

Figure XX - 2. The abdominal cavity is filled with bluish-green jelly which is solidified abdominal dropsy. This eel has also developed a complication of red fin disease.

Figure XX - 3. A whitebait eel affected by abdominal dropsy disease. Whitebait eels affected by abdominal dropsy disease exhibit the warped body posture pictured here.

(Text by Egusa)

(Photographs by Egusa)

Photo 1

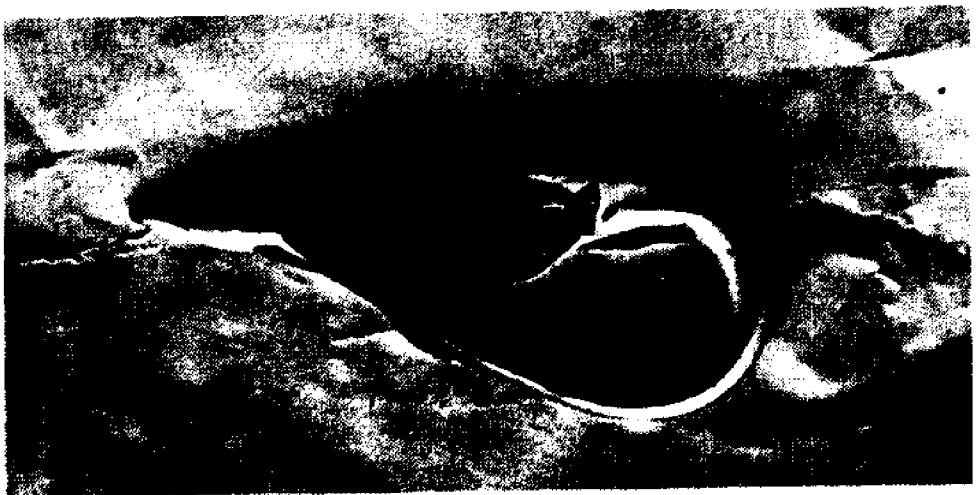

Photo 2

Photo 3