

1977

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Recommended Citation

Curry, Carol (1977) "Diseases of Salt Water Fish," *Iowa State University Veterinarian*: Vol. 39 : Iss. 1 , Article 7.
Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol39/iss1/7

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Diseases of Salt Water Fish

by
Carol Curry*

In a previous article (*ISU Veterinarian* Vol. 38, No. 2, 1976), the environmental problems associated with keeping saltwater fish were reported. This article will deal with the diagnosis and treatment of common coral fish diseases.

To diagnose a disease, the veterinarian must first know about the normal animal. Fish anatomy resembles that of domestic animals, with a few additions, i.e., gills, swim bladder, and head kidney (see diagram A). Most fish diseases are diagnosed by a post mortem examination of one or several individuals, although history and clinical signs should also be considered. The movement and behavior of a diseased fish should be observed. Is it swimming fast or slow? Does it make darting, erratic movements or lay at an angle on the bottom? Are the respirations rapid or does it gulp air at the top of the tank? Is an escape reflex present? All of these things could aid in making a diagnosis.

The external features may also reflect a clinical problem. One common sign of diseased fish is a pale color, particularly noticeable in the brightly colored marine fish. Raised scales, a disoriented skin pattern, abrasions or ulcers are also pathological. Spots are a common sequella of the fish parasites. A magnifying glass will aid in the examination, which can be done out of water. Use caution, however; some marine fish are poisonous to man.

Smears and scrapings can be taken from live or dead specimens. The procedure is the same as with other domestic animals. The skin and gills are often examined for parasites. The most common bacteria

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isolated from fish are *Aeromonas*, *Pseudomonas*, and *Vibrio*. The primary external fungal pathogen is *Saprolegnia*.

Euthanasia can be accomplished in several ways. With small fish, a single cut in the nape of the neck (see Diagram B) is the best technique because it does very little damage to the fish's body. Large fish can be electrified or killed by a blow to the head. A fish anesthetic, MS-222 (tricaine methanesulfonate), is also available.

A thorough post mortem examination should be performed. Four incisions will expose all the important areas for inspection (see Diagram C). Samples of internal organs, smears, and culture swabs are taken according to standard procedure. Several books listed in the reference section contain pictures and descriptions of the pathology and parasites involved in fish diseases.

The following are some of the more common fish diseases seen in marine aquarium fish. The clinical signs, pathogenesis, diagnosis, and treatment involved for each disease will be discussed.

A. *Velvet*

1. Etiology: *Oodinium pillularis*, a light and temperature sensitive dinoflagellate. A temperature of 23-25°C. and a bright light are necessary for maximum growth.

2. Clinical signs: subtle in the early stages when the primary sign is loss of color. The fish becomes anorexic, thin, and depressed. Gradually, the skin becomes peppered with fine white spots which merge to create a yellow, velvety appearance to the skin. The fish rubs on rocks and coral, eventually causing lacerations and tags of skin along the sides and back. Respiration becomes rapid. The disease is

most often seen in West Indian Ocean fish, especially clown fish and puffers. In aquariums, the problem often occurs after new arrivals are introduced to the tank.

3. Pathogenesis: the parasite borrows into the epidermis. This causes a local inflammatory reaction which irritates the skin and gills and results in itching plus labored respiration. The parasite drops off the fish and falls to the bottom where it forms spores which divided into dinospores. These dinospores must find a host within 24 hours. The entire cycle takes about 10 days.

4. Diagnosis: examine skin or gill scrapings for the parasite.

5. Treatments:

a. Copper sulfate is highly effective. All invertebrates, not only the parasites, are sensitive to copper and must be removed from the treatment tank. Add 1.5 cc of a stock solution (1 gm/liter) to one liter of water for 3-10 days. This can be increased to a concentration of 20 mg/gal before the fish will show signs of toxicosis, such as ulceration of the body and necrotic liver. The CuSO_4 is replenished daily because bicarbonates in the water inactivate 30% of the copper every 24 hours. Brine shrimp will concentrate the copper and therefore should not be fed to fish during the treatment period to avoid accidental poisoning.

b. 3% salt bath for 5-15 minutes in a clean, dark tank. The contaminated tank should be fully illuminated to accelerate the life cycle of the remaining parasites. Set the temperature at 30°C. for 24 hours and then allow it to cool for 48 hours.

c. Methylene blue—1% stock solution. Use 3 cc. stock solution/ten liters of water for 10 days. This will turn the water dark blue but the color will gradually fade as the dye is reduced.

d. Acriflavine (trypaflavine)—1 gm/100 liters water used as a bath for 20-30 minutes.

e. Aureomycin (chlortetracycline)—13 mg/liter of water used as a bath for 20-30 minutes.

B. Coral Fish Disease

1. Etiology: *Oodinium ocellatum*. This species of *Oodinium* has more of an affinity for gill tissue than the *Oodinium* which

causes velvet disease (affinity for the skin).

2. Clinical signs: small, round, white nodules on the gills which progress to hemorrhagic inflammation and necrosis. Respirations become rapid and the fish eventually comes to the surface to gulp for air. The skin may appear cloudy during the later stages.

3. Pathogenesis: same cycle as velvet only this parasite penetrates the subepithelial tissue of the gills. Inflammation is caused by secondary bacteria.

4. Diagnosis: examine wet mount smear of skin or gills (preferred) for the parasite.

5. Treatments: must be done during the early stage of the disease, or permanent gill damage will result. If the skin is affected, it is probably too late.

a. Copper sulfate—stock solution is 1.5 gm/liter of water. Add 2 ml/liter of water in the treatment tank. Follow same precautions as for treatment of velvet.

b. Methylene blue—same as for velvet.

c. Acriflavine (trypaflavine)—same as for velvet.

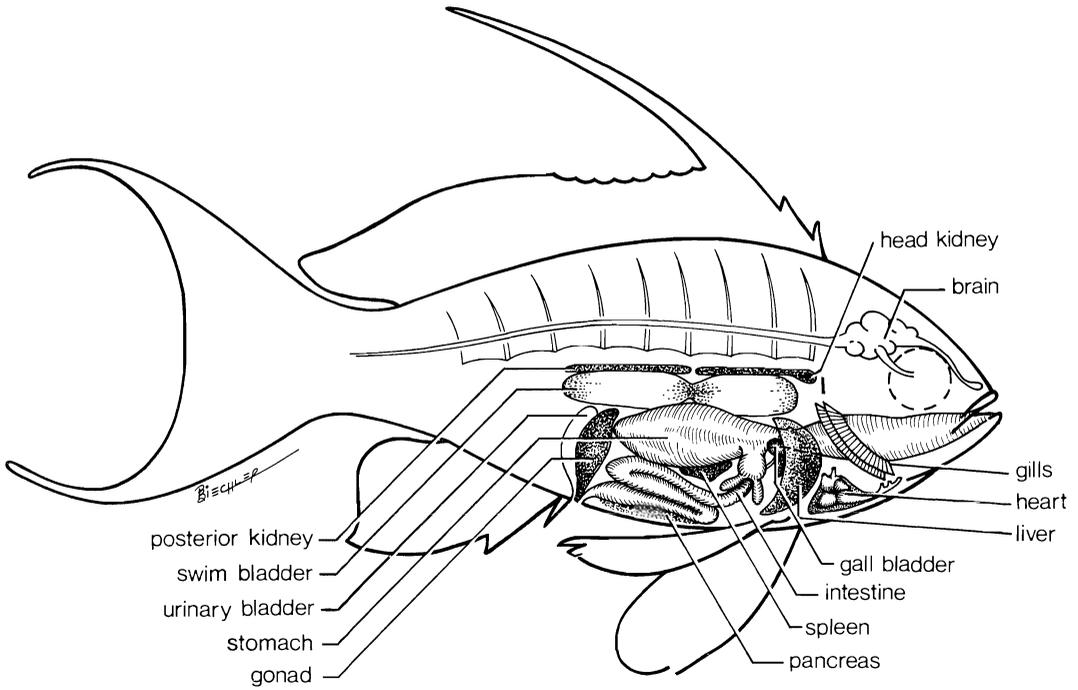
d. Aureomycin—same as for velvet.

C. Ick—White Spot Disease

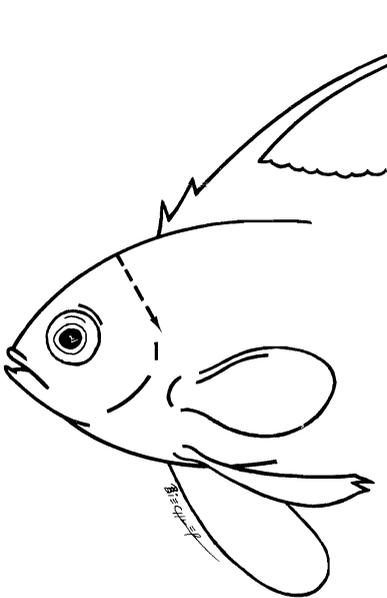
1. Etiology: *Ichthyophthirius marinus*, a ciliate. This parasite will spread quickly in a tank following stress, such as overcrowding, or a sudden change in temperature. It is more severe in marine than in freshwater fish.

2. Clinical signs: large white spots, about 1 mm in diameter, are seen on the body, usually starting on the fins and spreading to other areas. Large areas can merge and slough. It is very irritating to the skin, causing the fish to swim erratically and rub on the rocks and coral. This irritation also increases the mucus secretion of the gills so respiration is impaired.

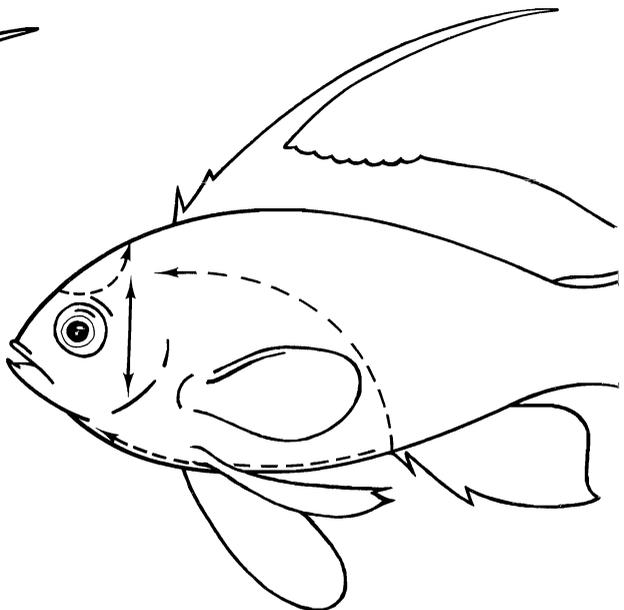
3. Pathogenesis: the parasite drops off fish when it matures. One adult can divide into thousands. *Ichthyophthirius* is hardier than *Oodinium* (velvet); it can survive without a host for a longer period of time and will withstand much lower temperatures. The free living stage bores into the epidermis and slides along the dermis developing small tunnels. The white spots are due to an epidermal proliferation



Anatomy—Diag. A



Euthanasia Incision—Diag. B



Post Mortem Incisions—Diag. C

caused by the burrowing. Secondary bacterial or fungal infection may also be established. The life cycle lasts approximately 24 days. *Ichthyophthirius* is often isolated from normal, healthy fish, indicating the possibility of immunity and a carrier state. Stress of any kind may cause an outbreak.

4. Diagnosis: direct wet smear taken from skin or gills; clinical signs are usually indicative of the need for treatment.

5. Treatments:

a. increase the temperature 10 °F. and turn out the lights.

b. Acriflavine (trypaflavine)—20-30 minute bath at the same dosage as for velvet; increase temperature to 30°C. to accelerate the parasite life cycle. Treat for 3 weeks; the drug is effective only against those parasites not attached to a host.

c. Malachite green—.15 mg/liter for ten days. Stop filters when using this dye.

d. Methylene blue—1% stock solution. Use 4 cc/gallon of water on the first day. Repeat the dose every other day for a total of six treatments.

e. If the fish are removed from the contaminated tank, the remaining parasites will be cleared in three days and the water can be reused. Quinine or meparine are used to treat this disease in freshwater tanks but the high pH of salt water will inactivate these drugs.

D. *Ichthyophonus*—*Ichthyosporidium*

1. Etiology: *Ichthyophonus hoferi*. It is a fungus which may be transmitted via food, brine shrimp, or copepods.

2. Clinical signs: vary according to which organs are attacked. The belly may become bloated (liver) or gaunt with abnormal swimming movements (swim bladder). The skin may become ulcerated with the scales protruding, creating a sandpaper effect.

3. Pathogenesis: primarily attacks the liver and heart with some affinity for the kidney, spleen, and muscles. Infectious spores enter the intestine and are absorbed by the blood stream. Cysts are formed which send out hyphae; these encyst elsewhere in the viscera. It is a contagious, insidious, chronic disease.

4. Diagnosis: look for fungal cysts in scrapings from the heart and liver. Stain with Giemsa's stain.

5. Treatment: successful if diagnosed at an early stage. It may, however, take months to a year before the fish shows any clinical signs or dies. By then, the other fish in the tank may already be beyond the point where treatment is effective.

a. Phenoxethol: soak dried food in a 1% solution. Also soak fish in a phenoxethol bath for 1-2 hours using 10 cc of a 1% solution/liter of water.

b. Antibiotics: intramuscular injections of penicillin or chloramphenicol in the muscles of the side and belly.

E. *Lymphocystis*

1. Etiology: pox virus (DNA); inclusion bodies in the cytoplasm.

2. Clinical signs: mineralized masses occur on the skin which look like patches of warts or raspberry-like growths. It usually isn't fatal but the lesions may become secondarily infected or affect the swimming action of the fish.

3. Pathogenesis: first nodules are seen about two months post-infection; growth is very slow. The fibroblasts become greatly enlarged.

4. Diagnosis: based on characteristic clinical signs and inclusion bodies in cytoplasm of hypertrophied fibroblasts.

5. Treatments: none are very effective.

a. Remove the fish which are showing clinical signs. The remaining fish cannot be considered safe for at least two months.

b. Empty the tank and disinfect it with HCl.

c. Remove the lesions surgically and bathe the site with iodine to prevent secondary infection.

F. *Fin Rot*

1. Etiology: *Vibrio ichthyodermis* or *Pseudomonas ichthyodermis*; classification is currently being questioned.

2. Clinical signs: frayed fins and tail are the primary signs. They may become stumps in advanced cases. Minute hemorrhages and subsequent ulceration of the skin occur in these more serious infections.

3. Diagnosis: clinical signs plus isolation and identification of the bacterial agent.

4. Treatments:

a. Antibiotics: pen-strep or chloramphenicol, parenterally; oxytetracycline in

the food.

b. Touch the affected areas with iodine solution. Let the fish recover from the shock of being handled, then put them in a clean tank containing chlortetracycline (250 mg/5 gal of water) for 3-4 days.

G. *Exophthalmus*—*Popeye*

1. Etiology: unsure of exact mechanism; exophthalmus is a common sign of many diseases.

a. Malnutrition
b. Excessive aeration
c. Dirty water
d. Bright lighting, especially important for fish which live at depths below 30 feet (approximate level of light penetration in the ocean) or fish which are used to murky water.

e. Bacteria

f. EPS (exophthalmus producing substance)—may be a substance produced in the hypophysis or a lack of its antagonist. It stimulates an increase of fluid in the orbit.

2. Clinical signs: more common in marine than freshwater fish. The eye swells and protrudes from the side of the head. If this continues, it will eventually pop out of its orbit. Secondary bacterial infections are common. One or both eyes may be affected.

3. Pathogenesis: one theory concerns excessive aeration. If the water temperature becomes high, oxygen is driven off rapidly. The oxygen partial pressure in the fish's blood is in equilibrium with this pressure in the water. When external oxygen pressure in the water drops, the equilibrium is upset; the gases can't diffuse out of the blood fast enough. Hemoglobin will help equalize the oxygen pressure but nitrogen has a low solubility in blood. Bubbles of nitrogen are trapped in the blood stream and accumulate behind the eyes so the eye swells. This is approximately the same mechanism as

caisson's disease or the bends in man.

4. Diagnosis: usually based on clinical signs.

5. Treatments: vary according to the cause.

a. clean the tank
b. balance the diet
c. decrease the light

d. swab with 2% silver nitrate, then follow this with 2% potassium dichromate. A three day bath with 1 gm potassium dichromate/5 gal plus one-half ounce rock salt is the last resort.

These are only a few of the diseases which coral fish may contract. Most of these treatments are fairly simple and inexpensive. They also offer low risk to the fish and may be attempted without jeopardizing the economic investment of the owner; it may, in fact, be the only course of action which can save a tankful of expensive fish.

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