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ENTERIC REDMOUTH DISEASE

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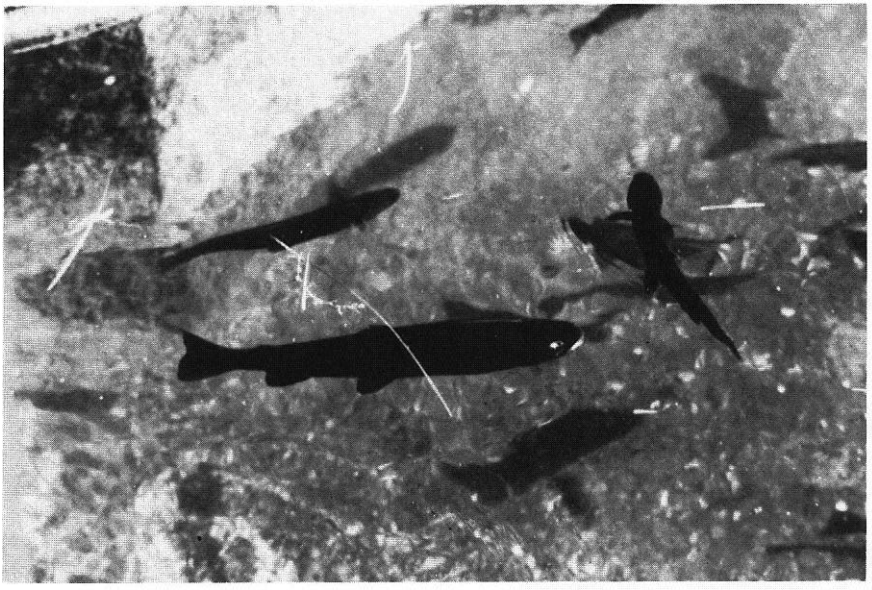
Enteric redmouth (ERM) is an acute to chronic systemic bacterial disease of salmonids. It is especially severe among intensively cultured rainbow trout reared in water warmer than 13°C (Dulin et al. 1976). The disease is caused by a motile, Gram-negative, rod-shaped bacterium identified as Yersinia *ruckeri* (O'Leary et al. 1979; Ewing et al. 1978). This pathogen is readily transmitted from fish to fish by contact and through the water (Rocker 1966). Where ERM has become established, it usually causes sustained low-level losses. Occasionally, severe epizootics occur with mortalities exceeding 50% if corrective measures are not taken (Ross et al. 1966; Bullock et al. 1976). Surviving fish frequently become asymptomatic carriers which can spread the disease if these fish are transferred to new locales (Busch and Liigg 1975; Hunter et al. 1980). Enteric redmouth disease can be successfully controlled by a combination of environmental improvement and antibactetial drugs (Rocker 1966; McDaniel 1971). Vaccines have been developed that can reduce the impact of the disease (Busch et al. 1978).

SIGNS OF INFECTION

Acute cases of ERM seldom go undetected. The clinical signs, however, are similar to those of other common bacterial infections. *Aeromonas hydrophila* and *A.* salmonicida infections are indistinguishable from ERM if the diagnosis is based solely upon clinical signs (Dtin et al. 1976). Enteric redmouth causes different signs at different stages of the disease and a wide variety of signs can be observed among the victims of a single epizootic (Wobeser 1973; Rocker 1966).



Rainbow trout infected with enteric redmouth bacterial disease are frequently dark in color and exhibit lesions associated with mucous membranes of the mouth and eyes. (U.S. Fish and Wildl. Serv.

EXTERNAL SIGNS

1. Dark, lethargic fish that do not feed and appear to isolate themselves from the main population are evident in the early stages of an ERM outbreak. This condition will also be common among carriers after losses have subsided. Affected fish often have missing eyes or exophthalmia (popeye) and are blind and sluggish with little avoidance reaction.

2. During the acute stage of the disease, small bright hemorrhages occur along the gum line of the mouth and on the tongue, which, together with general inflammation, give the "redmouth" appearance for which the disease is named.

3. The normally white ventral body surface (belly) may be speckled with small hemorrhages that may also be evident at the base of the fins.

INTERNAL Signs

1. Flaccid stomachs filled with clear fluid are common in fish with ERM. *This* is an *important* sign! Enteric redmouth should be suspected whenever rainbow trout with hemorrhages in the mouth are found to also have flaccid, fluid-filled stomachs.

2. Enteric redmouth victims often have enlarged, dark spleens, hemorrhagic specks on the air bladder and pyloric cecae, and reddening of the posterior intestinal tract.

DIAGNOSIS

The diagnosis of enteric redmouth disease depends upon the detection isolation and identification of the causative bacterium, Y *mckeri*. Bacteriological samples from the lower intestine and posterior kidney should be collected from dark, sluggish fish and from any fish with hemorrhages in the mouth and flaccid, fluid-filled stomachs. A quick presumptive diagnosis can be made by conducting fluorescent antibody tests on smears prepared from these samples (Bullock and Snieszko 1979: Johnson et al. 1974). One method for detecting Y ruckeri in asymptomatic carriers is to apply fluorescent antibody tests on smears prepared from scrapings of the posterior intestinal tract (Busch and Liigg 1975; Hunter et al. 1980). Confirmation of the presence of Y *ruckeri,* however, requires the bacteriological isolation of Gram-negative, motile rods which have the following biochemical characteristics: cytochrome oxidase negative; acid, but no gas from glucose; positive for ornithine and lysine decarboxylase but not arginine dihydrolase; and, a positive agglutination with rabbit anti-K *ruckeri* serum (Bullock et al. 1978).

EPIZOOTIOLOGY

GEOGRAPHIC AND HOST RANGES

In the USA, ERM has occurred in at least 18 of the lower 48 states and in Alaska (Bullock et al. 1978). In Canada, the disease or its agent has been detected in British Columbia, Saskatchewan, Ontario and Nova Scotia (Bullock et al. 1978). The disease is most prevalent in rainbow trout. It has also been reported from cutthroat trout and from coho, chinook, and Atlantic salmon (Bullock and Snieszko 1979). The principal factor involved in the spread of the disease has been the shipment of infected carriers (Dulin et al. 1976; Busch and Lingg 1975). The causative bacterium, Y.*ruckeri,* occurs naturally in some areas and ERM can occur when susceptible fish are subjected to stress (Rucker 1966; Hunter et al. 1980). There are no reports of natural outbreaks of ERM among free-ranging wild fish.

SOURCES AND RESERVOIRS OF Infection

Asymptomatic carriers are the primary source and reservoir of Y. *ruckeri* (Rocker 1966; Busch and Lingg 1975). After a population of fish has recovered from an ERM outbreak, carriers in the group serve as a major source of reinfection. An infection/recovery/re-infection cycle of 36-40 d was reported by Busch and Liigg (1975) in rainbow trout held at 14.5°C. Their work showed that more than 25% of the survivors carried localized infections in their lower intestine. Invertebrates, such as crayfish, that inhabit the water supplies of hatcheries where ERM persists have been found to harbor Y.*ruckeri* but the role of invertebrates in the etiology of this disease is unknown (Dulin et al. 1976).

MODES OF TRANSMISSION

Fish to fish transmission has been frequently reported from both hatchery and laboratory fish populations (Dulin et al. 1976; Rucker 1966; Hunter et al.

1980). Direct fish-to-fish contact is not necessary. If water from infected fish passes through rearing units containing susceptible fish, these fish readily become infected (Ross et al. 1966). No evidence of the transmission of Y. ruckeri from parent to progeny with eggs has been reported.

SUSCEPTIBILITY AND RESISTANCE FACTORS

Rainbow trout are severely affected by Y. ruckeri infections and enteric redmouth disease causes serious losses in raceway-cultured fish, especially in commercial hatcheries in southern Idaho (DuIin et al. 1976). Water temperature, crowding, excess ammonia, low oxygen, obesity, and handling stresses are important factors that influence the timing and severity of epizootics (Rocker 1966). Since these factors can be controlled by fish culturists, the disease can be managed by maintaining good environmental conditions (Dulin et al. 1976; McDaniel 197l).

Several workers report that fish size affects the susceptibility of fish (Rucker 1966; Dulii et al. 1976). While the disease is seldom a problem in rainbowtrout less than 7.5 cm in length, size alone may not be the determining factor. In many hatcheries, fish must be periodically weighed, moved to new rearing units, or sorted to harvest market-sized fish. These activities often take place when the fish population has reached or exceeded the optimal carrying capacity for the unit. Under these circumstances, stress from handling can trigger outbreaks of ERM, especially if water temperatures exceed 13°C (Dulin et al. 1976; Busch and Lingg 1975). At these temperatures rainbow trout grow rapidly, oxygen demands are high, and economics losses due to ERM will often be high among large fish (Dulin et al. 1976).

INCUBATION PERIOD

At 15°C, enteric redmouth disease kills 7.5-10.0 cm rainbow trout in 5-19 d (Dulin et al. 1976: Ross et al. 1966). In 125°C water, Atlantic salmon 6.0 cm in length, began to die 9 dafter exposure (Bullock et al. 1976). Cumulative losses of 52% occurred in three separate sets of experiments in which the fish were not medicated. After mortality ceases, Y. *ruckeri* can be detected in the survivors for another 60 d, but after 80 d the bacteria may be no longer detectable (Hunter et al. 1980).

SEASONAL Incidence

No reports suggest that the occurrence of enteric redmouth is seasonal. If water temperatures are constant, stress-mediated epizootics may occur at any time (Hunter et al. 1980).

METHODS OF CONTROL

PREVENTION

The transfer of carriers to hatcheries previously free of ERM has been well documented as the primary way this disease has been spread (Dulin et al. 1976;

Busch and Liigg 1975). Thorough inspections of hatchery fish populations prior to the shipment of fish to other hatcheries can help to prevent the inadvertant introduction of ERM. Persistent monitoring of mortalities for cause of death coupled with annual fish health inspections are invaluable in developing a reliable history of the absence of ERM disease at a facility. These measures are necessary because the detection of Y ruckeri in apparently healthy carrier fish can be difficult, especially when the fish are being reared in good environmental conditions. This fact, however, demonstrates the efectiveness of good fish cultural conditions in reducing the overall impact of ERM in hatcheries where it has been troublesome in the past. Other important preventive steps include the use of chlorine to disinfect water supply springs, ditches, pipelines and head-boxes to remove possible disease-carrying fish, invertebrates and bacteria (McDaniel 1971).

Immunization of cultured trout against ERM can also be beneficial (Busch et al. 1978). Commercial vaccines are now available which improve the ability of fish to ward off the disease. While immunization does not orovide total protection against ERM, it apparently contributes sufficiently to thewell-being of the fish to be worthwhile. A word of caution is in order, however. Care should be taken to starve the fish for 24-72 h prior to handling and prophylactic treatments should be given to rid the fish of sub-clinical cases of bacterial gill disease or external parasites. If precautionary measures are neglected, stresses associated with the immunization process can elicit outbreaks of other diseases (Busch et al. 1978).

THERAPY

Sulfamerazine and oxytetracycline (Terramycin) have been used extensively to control ERM (Dulin et al. 1976). Regardless of the effectiveness of antibacterial drugs, they alone cannot be relied upon for the control of this disease. Adverse environmental factors and excessive handling stresses must be eliminated or the disease may recur shortly after drugs are withdrawn. Neither sulfamerazine, oxytetracycline, nor any other drugs used in the past against ERM are currently approved by the U.S. Food and Drug Administration for use in food fish to control ERM. Research studies needed for the registration of compounds effective against ERM should be given a high priority.

KEY STEPS TO REMOVE THE DISEASE and/or AGENT FROM FISH POPULATIONS

IMMEDIATE

No procedures have been developed that will completely free fish populations of Y. *ruckeri* bacteria once they have become infected.

LONG TERM

Although prevention is the key to minimizing the affect of ERM, several steps must be followed after the disease has been detected. A clean water supply that is free of fish and invertebrates is essential. Rearing facilities should be

depopulated and disinfected. This can be accomplished all at once, or in carefully-orchestrated phases while fish production continues (McDaniel 1971). Strict control over all fish brought into the hatchery or over the associated geographic area will help to protect ERM-free hatcheries and watersheds.

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