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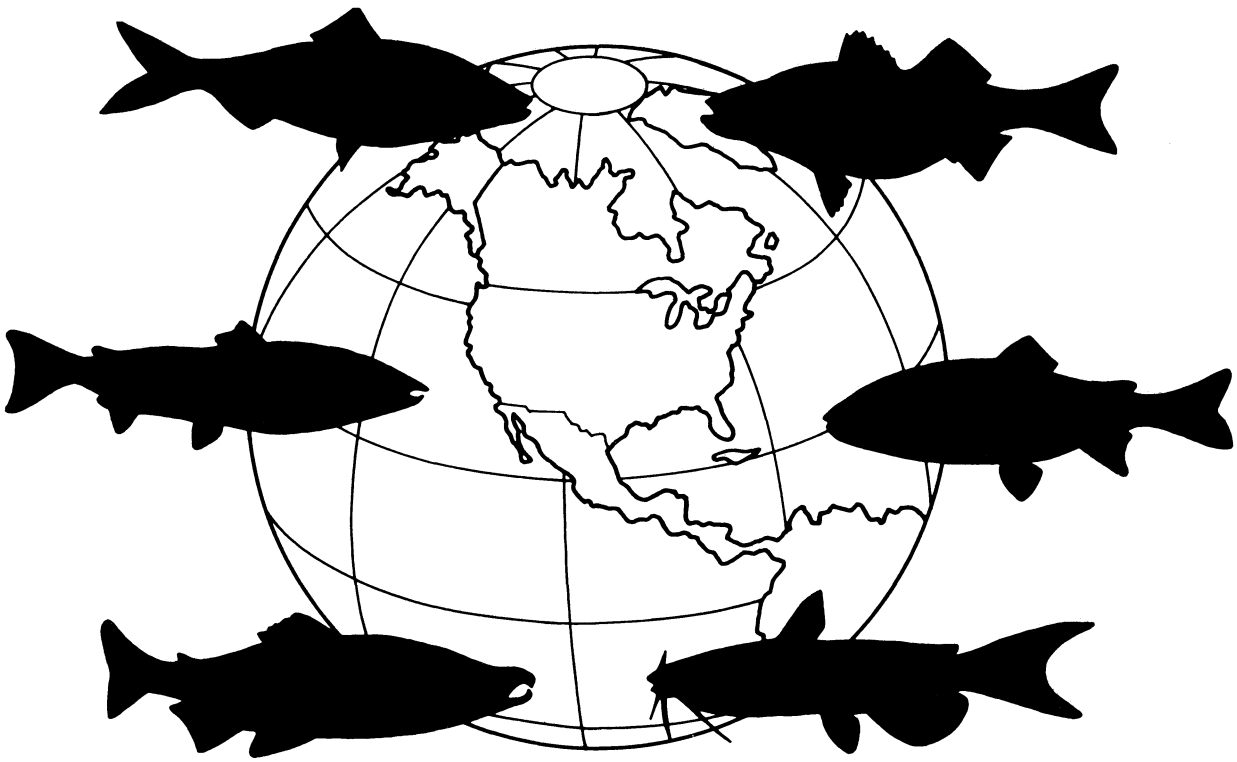

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Introduction

Enteric redmouth disease (ERM) is a systemic bacterial infection of fishes, but is known principally for its occurrence in rainbow trout, *Oncorhynchus mykiss*. It was first reported in the 1950's in Idaho rainbow trout and described by Rucker (1966). In recognition of that first description, the causal organism was named *Yersinia ruckeri* by Ewing et al. (1978).

Clinical Signs

In early acute epizootics, typically affected rainbow trout are lethargic and anorexic and have subcutaneous hemorrhages in and around the mouth, oral cavity, and isthmus, and at the base of fins. Gill filaments may be hemorrhagic. Petechial hemorrhages may occur on the surface of the liver, pancreas, pyloric caeca, and swim bladder, and in the lateral musculature. The spleen is enlarged and friable, gonads are hemorrhagic, and the lower intestine is inflamed and filled with a thick, yellowish fluid. Exophthalmus occurs, commonly accompanied by hemorrhages around the ocular cavity and iris. The affected eyes commonly rupture. If fish survive, they darken and seek shelter, or withdraw from other fish (Busch 1983; Rucker 1966). In atypical infections that sometimes occur, no hemorrhages develop on the mouth and gill cover; fish merely become dark and swim near the surface (Frerichs et al. 1985). Histological examination of tissues from infected trout shows an acute bacteremia and attendant inflammatory response in virtually all tissues. Bacteria are especially conspicuous in vascular tissue and in areas of petechial hemorrhage (Rucker 1966). Bacterial colonization occurs in the capillaries of well vascularized tissue and is followed by dilation of small blood vessels; petechial hemorrhages; erythrocyte congestion; and edema of the kidneys, liver, spleen, heart, and gills. Focal necrosis may occur in the liver, and marked accumulations of mononuclear cells in periportal areas. Hemorrhages develop in outer portions of the digestive tract, and the lining or mucosa becomes edematous and necrotic and sometimes sloughs into the lumen (Busch 1983).

Enteric redmouth disease commonly causes sustained, low-level mortality, eventually resulting in high losses. Large-scale, acute epizootics sometimes occur if chronically infected fish are stressed.

Etiology

Serotypic differences of *Y. ruckeri* were originally associated with the ability of isolates to ferment sorbitol. For several years after the original isolation of *Y. ruckeri* from salmonids in the Hagerman Valley, Idaho, all isolates were serologically similar and did not ferment sorbitol (Ross et al. 1966; Busch 1983). These were called Type I. Even today, most *Y. ruckeri* that do not ferment sorbitol form a single and distinct serotype (Pyle et al. 1985, 1987).

O'Leary (1977) described another serotype of *Y. ruckeri*, isolated from Pacific salmon (*Oncorhynchus* spp.), that fermented sorbitol. This was called Type II. Later research has shown that collections of *Y. ruckeri* that ferment sorbitol can be differentiated into as many as five distinct serotypes (Pyle et al. 1985, 1987; Stevenson and Airdrie 1984). Only Types I and II are now known to cause ERM epizootics.

Diagnosis and Detection

Presumptive diagnosis of ERM can be based on clinical signs and on the inoculation of infected tissue on the differential medium of Shotts and Waltman (1983). The direct or indirect fluorescent antibody test, and monoclonal-based, enzyme-linked immunosorbent assay (Austin et al. 1986), may be used for serotypes I and II; however, as previously mentioned, three additional serotypes have been described and if these serological tests are negative, ERM cannot be ruled out. Antisera for these additional serotypes are not generally available. Serological identification of Type I and Type II is based on agglutination or fluorescent antibody tests. Confirmatory diagnosis of ERM requires isolation and identification of the causative agent. Identification is based on the isolation of a gram-negative, motile, rod-shaped bacterium with distinctive properties: it is cytochrome oxidase negative; produces acid but usually no gas in glucose; produces an alkaline slant and acid butt in triple sugar iron agar; and reacts positively with ornithine and lysine decarboxylase. The isolate should also be negative with esculin and salicin to separate it from certain isolates of *Serratia liquifaciens* that do not ferment sucrose.

Source and Reservoir of Infection

Although *Y. ruckeri* was first isolated in Idaho by R. Rucker in the 1950's, it has now been established that the bacterium was also present in West Virginia and Australia in the 1950's (Bullock et al. 1977); consequently, the true source of the organism is unknown. Busch and Lingg (1975) showed that 25% of the rainbow trout surviving an experimental ERM challenge became asymptomatic carriers in which the bacterium was local-

ized in the lower intestine. Such trout serve as reservoirs of infection.

Mode of Transmission

Natural infections spread from fish to fish by direct contact with infected fish or carriers. Rucker (1966) transmitted the disease by exposing healthy rainbow trout to waterborne bacteria shed by infected trout. Bullock et al. (1976) similarly infected Atlantic salmon (*Salmo salar*). Vertical transmission has not been demonstrated (Dulin et al. 1976), and probably does not occur. Stressors have been shown to play a significant role in triggering ERM outbreaks (Hunter et al. 1980).

Incubation Period

Experimental evidence suggests that incubation time is 5 to 10 days at 13–15° C. In natural outbreaks, the incubation period is undoubtedly affected by environmental factors such as temperature, pH, and dissolved oxygen.

Host and Geographic Range

Since ERM was first reported, knowledge of the host and geographic ranges has increased. The spread of ERM from Idaho was originally associated with the transportation of carriers, and within 20 years the disease had been spread to virtually all trout-producing regions of the United States and Canada. The disease has now been reported in most European countries where trout are cultured. To date there are no reports of ERM outbreaks in Japan, Australia, or New Zealand. The host range has also expanded to include Atlantic salmon and Pacific salmon, and nonsalmonids such as emerald shiners, *Notropis atherinoides* (Mitchum 1981); fathead minnows, *Pimephales promelas* (Michel et al. 1986); goldfish, *Carassius auratus* (McArdle and Dooley-Martyn 1985); and farmed whitefish, *Coregonus* spp. (Rintamäki et al. 1986). Additionally, ERM infections have occurred in several farmed marine species such as turbot, *Scophthalmus maximus*; seabass, *Dicentrarchus labrax*; and seabream, *Sparus auratus* (Vigneulle 1984).

Methods of Control

Prevention

Enteric redmouth disease is the first fish disease for which a practical, commercially available bacterin was developed. The first successful experimental bacterin, reported by Klontz (1963), was intended for oral delivery. It was improved by later investigators (Ross and Klontz 1965; Anderson and Ross 1972). Anderson and Nelson (1974) then showed that injection of a bacterin was superior to oral administration. However, injection is not practical for immunizing large numbers of small fish.

Croy and Amend (1978) showed that fish could be immunized by immersion in a hyperosmotic solution of sodium chloride, followed by immersion in the bacterin. The first commercial ERM bacterin, licensed in 1976, was delivered by this procedure. Continued developments in the delivery system showed that either simple immersion or spray application of bacterin resulted in protection. Cipriano and Ruppenthal (1987) found that brook trout (*Salvelinus fontinalis*) immunized with *Y. ruckeri* of serotypes I or II were protected against experimental challenge regardless of the serotype used for immunization. The use of commercial ERM bacterin has resulted in decreased losses from the disease, decreased need for antibacterial therapy, and—surprisingly—an increased growth rate in immunized fish (Amend and Eshenour 1980; Tebbitt et al. 1981).

A considerable body of information is now available on the immune response of trout to *Y. ruckeri* and ERM vaccines. Blazer and Wolke (1984) found that rainbow trout fed a commercial diet showed a reduced immune response to *Y. ruckeri* antigens in comparison with trout fed a complete test diet. They postulated that ascorbic acid or vitamin E enhanced the immune response. Exposure of trout to endrin adversely affected the immune response (Bennett and Wolke 1987). Although humoral *Y. ruckeri* antibodies were not correlated with protection (Cossarini-Duner 1986; Cipriano and Ruppenthal 1987), these antibodies were correlated with an increased rate of phagocytic ingestion of *Y. ruckeri* (Griffin 1983). When salmonids are immunized by a bath procedure, the gill pavement cells seem to play a major role in antigen uptake (Zapata et al. 1987); low (1:1000) vaccine dilution and an exposure time as long as 6 h provided the best protection (Tatner and Horne 1985). Recently Horne and Robertson (1987) developed a model that enables the value of ERM vaccination to be determined under field conditions.

Tests conducted in vitro by Ross and Smith (1972) showed that 25 ppm iodine of the iodophors Betadine or Wescodyne destroyed cells after only a 15-s exposure. The authors suggested that a 5-min exposure of salmonid eggs to 25 ppm iodine would be effective in killing ERM cells.

Treatment

Several antibacterials, including oxytetracycline, erythromycin, quinolones (Ceschia et al. 1987), and the

potentiated sulfonamide Romet (Bullock et al. 1983), have been reported to be effective in controlling ERM. Although the antibacterial tiamulin reportedly controlled ERM (Bosse and Post), it failed to control experimental *Y. ruckeri* infection in rainbow trout (Bullock and Herman 1988). To date no antibacterial is registered with the Food and Drug Administration for control of ERM in cultured food fish.

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