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COMPARATIVE SUSCEPTIBILITY OF ATLANTIC SALMON (*Salmo salar*) TO THE ENTERIC REDMOUTH BACTERIUM AND *Aeromonas salmonicida*

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Abstract: The bacterium causing enteric redmouth (ERM) and *Aeromonas salmonicida* were found to be equally pathogenic for fingerling Atlantic salmon (*Salmo salar*). Injection of 5×10^5 cells of ERM or *A. salmonicida* killed all salmon within 96 h. After a 30 min exposure to water-borne cells of the two test bacteria about one-half of the test salmon died within 14 days. Both ERM and *A. salmonicida* were transmitted horizontally. Results indicate that efforts should be made to prevent introduction of ERM into watersheds where Atlantic salmon occur.

INTRODUCTION

Redmouth disease was first observed in rainbow trout (*Salmo gairdneri*) in Idaho by R. Rucker¹ in the 1950's. The causative bacterium was characterized by Ross *et al.*² as a member of the Enterobacteriaceae, but was not assigned to a genus or species. The term "enteric redmouth" (ERM) was recently introduced¹ to designate the disease and this name is used in the present report.

Until recently, ERM occurred principally in rainbow trout in the intermountain areas of the Western United States; however, the Eastern Fish Disease Laboratory now has cultures from outbreaks in Ohio, Arkansas, Virginia, and Tennessee; Wobeser³ described an epizootic in Saskatchewan, Canada.

Inasmuch as ERM has extended its range into areas where Atlantic salmon (*Salmo salar*) are cultured, we determined its pathogenicity for this species.

MATERIALS AND METHODS

Two cultures each of the ERM bacterium and *A. salmonicida* were used: the ERM cultures were originally isolated from chinook salmon (*Oncorhynchus tshawytscha*) and rainbow trout and the *A. salmonicida* isolates were from brown

trout (*Salmo trutta*) and brook trout (*Salvelinus fontinalis*).

Cultures were taken from laboratory stocks held at -80°C , grown on tryptic soy agar for 48 h at $20-30^\circ\text{C}$, and suspended in sterile saline to an optical density of 1.0 at 525 nm. These suspensions contained between 7.0×10^8 and 1.3×10^9 viable bacteria per ml as determined by spread plate counts.

Two groups, each containing 10 Atlantic salmon with an average weight of 2.15 g, received a 0.05 ml intraperitoneal injection containing 6.5×10^7 or 6.5×10^8 bacteria. Two methods of water transmission were attempted. In the first, 10 salmon were placed in each of two 1500-ml beakers of water containing 10^7 ERM or *A. salmonicida* cells for 30 min and then transferred to running water. In the second method, we attempted horizontal transmission by holding 25 salmon in each of two aquaria receiving effluent from rainbow trout infected with ERM or *A. salmonicida*. All trials were run in duplicate.

Controls were handled in the same manner, but received saline or effluent from saline-injected rainbow trout. All fish were kept in 12.5°C spring water of known composition.⁷ Fish were observed for 21 days and all dead salmon were

examined bacteriologically. Bacteria isolated were identified by slide agglutination with specific antisera.

Tissues from moribund salmon that had been exposed to ERM were fixed in Bouin's solution for 24 h, transferred to 65% alcohol, dehydrated, stained with hematoxylin-eosin or Giemsa, and examined histologically. Fish with furunculosis were not similarly examined because the histopathological changes caused by *A. salmonicida* in salmonids have been well described.^{3,4}

RESULTS

Atlantic salmon were easily infected by injection of test organisms: all salmon injected with 5×10^7 cells of ERM or *A. salmonicida* died within 72 h, and all fish injected with 5×10^5 cells of either organism died within 96 h (Table 1).

Mortality among salmon exposed to water-borne organisms, although high, was less than that among injected fish and slower to develop. About one-half of the salmon exposed for 30 min to water-borne cells of two strains of ERM and one strain of *A. salmonicida* died within 14 days, almost all of the fish exposed to the second strain of *A. salmonicida* died during the same period (Table 1).

Both ERM and *A. salmonicida* were transmitted horizontally. First deaths occurred within 48 h among rainbow trout injected with either bacterium and within 10 days among salmon receiving effluent from injected trout. After 21 days, mortality was 54% among salmon receiving effluent from trout injected with ERM and 60% among those receiving effluent from trout injected with *A. salmonicida* (Table 1).

The mortality pattern was similar for the two pathogens (Fig. 1). None of the control fish died from ERM or furunculosis.

There were no consistent gross pathologic changes among salmon dying from ERM or *A. salmonicida*. About one-half of the salmon dying from ERM showed hemorrhages in the mouth and gill covers, and at the bases of fins. Internally, many salmon infected with either bacterium had a flaccid inflamed intestine containing yellow mucus. The histopathologic changes in salmon with ERM were seen primarily in the kidneys. The anterior kidneys showed severe necrosis of hematopoietic elements, with scattered pyknotic cells and karyorrhectic remnants. Necrosis also was noted in the mucosal epithelium of the intestine, pyloric caeca, and liver cells. The retina showed edema and necrosis, and gill epithelial cells were

TABLE 1. Comparative virulence of the ERM bacterium and *Aeromonas salmonicida* for fingerling Atlantic salmon (*Salmo salar*). (Numerator = number of fish that died, denominator = number of fish in aquarium).

Mode of transmission	Observation period	<i>Aeromonas salmonicida</i> strain		Enteric redmouth strain	
		1	2	1	2
Intraperitoneal injection of 5×10^7 cells	72 h	10/10	10/10	10/10	10/10
		10/10	10/10	10/10	10/10
Intraperitoneal injection of 5×10^5 cells	96 h	10/10	10/10	10/10	10/10
		10/10	10/10	10/10	10/10
Exposure for 30 min to 10^7 cells/ml spring water	14 days	4/10	9/10	5/10	5/10
		6/10	8/10	5/10	3/10
Exposure to contaminated effluent	21 days		17/25	16/25	
			13/25	11/25	

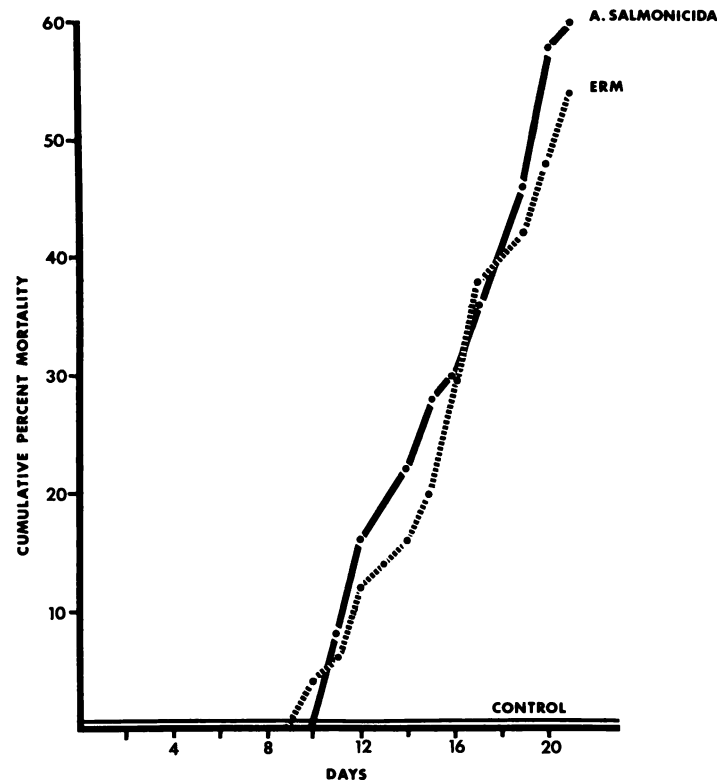


FIGURE 1. Cumulative mortality among fingerling Atlantic salmon (*Salmo salar*) receiving effluent from rainbow trout (*Salmo gairdneri*) infected with the ERM bacterium or *Aeromonas salmonicida*.

swollen and sloughed. Hemorrhagic areas were noted around the ureters and swim-bladder. High concentrations of ERM bacteria were found in most internal tissues, especially in vascular tissue of the liver, kidneys, and gills. Bacteria also were found in meninges, spinal cord and choroid. None of the lesions noted in infected specimens were found in control salmon.

DISCUSSION

These studies clearly show that the ERM bacterium is pathogenic for Atlantic salmon and as virulent as *A. salmonicida*. Results of experiments on water

transmission of ERM to Atlantic salmon are similar to those obtained by Rucker⁶ in which rainbow trout receiving effluent from trout infected with ERM showed a 5-day incubation period to first mortality and a 52% loss in 19 days. In Atlantic salmon the incubation period was 10 days and the loss was 54% within 21 days. The differences in incubation period may have been due to differences in water temperature: (15 C in Rucker's⁶ experiments and 12.5 C in the present study).

The histopathologic changes in ERM infected salmon were similar to those reported by Rucker⁶ and Wobeser⁸ for rainbow trout. In both salmonids, the disease was characterized by an acute

bacteremia; bacteria were particularly conspicuous in the highly vascular kidneys, liver, gills, and spleen. The kidneys were most severely affected in both salmon and trout; their hematopoietic tissue was destroyed.

Since Atlantic salmon are susceptible to ERM, and the disease has spread to the Northeast, ERM is a potential problem in fish husbandry. Mortality may not be limited to the hatchery; survivors of an epizootic may become carriers² and succumb to the disease from the stress of migration to and from saltwater.

Since the spread of ERM is often associated with shipments of live fish,^{6,8} additional spread of the disease can be limited by restricting movement of salmonids from areas where ERM is present. Alternatively, fish could be screened for the presence of carriers. Busch² has developed a passive hemagglutination test for agglutinins against the ERM bacterium, that makes possible the identification of individual carriers.

Every effort should be made to prevent the introduction of ERM into watersheds where Atlantic salmon occur.

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