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ENTERIC REDMOUTH DISEASE OF SALMONIDS

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INTRODUCTION

Enteric redmouth disease (ERM) is a systemic bacterial infection of fishes, but is principally known for its occurrence in rainbow trout, *Salmo gairdneri*. The disease was first reported in the 1950's by Rucker (1966), and in recognition of that fact, the causal organism was named *Yersinia ruckeri* by Ewing et al. (1978). Although Wagner and Perkins (1952) described a bacterial disease that produced a red-mouth condition in rainbow trout in California and Colorado, it was caused by a different bacterium; they isolated *Pseudomonas hydrophila* rather than *Y. ruckeri*.

IDENTIFICATION

In early acute epizootics, affected trout are lethargic and anoretic, and have subcutaneous hemorrhages in and around the mouth, oral cavity, and isthmus, and at the bases of and between fin rays. Gill filaments may be hemorrhagic. Petechial hemorrhages 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 or whitish fluid. As the epizootic progresses, unilateral or bilateral exophthalmus occurs, commonly accompanied by hemorrhages around the ocular cavity and iris. Affected eyes commonly rupture. If fish survive, they darken and seek shelter, or withdraw from other fish (Rucker 1966; Busch 1983). 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 heavily vascularized tissue. Bacterial colonization is followed by dilation of small blood vessels, petechial hemorrhage, erythrocyte congestion, and edema of the highly vascularized tissues of the kidneys, liver, spleen, heart, and gills. Focal necrosis occurs in the liver, and marked accumulations of mononuclear cells occur in periportal areas. Hemorrhages occur 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 that may eventually result in heavy losses. Large-scale and acute epizootics can occur if chronically infected fish are stressed by hauling, or exposed to low, marginal, or inadequate concentrations of dissolved oxygen or to other unfavorable environmental conditions.

DIAGNOSIS AND DETECTION

Tentative diagnosis of ERM is based on findings of clinical signs of the disease, but confirmation is based on isolation and identification of the causal bacterium. *Yersinia ruckeri* is a gram-negative motile rod that is cytochrome oxidase negative, metabolizes glucose with the production of acid but no gas, grows on triple sugar iron agar with an alkaline slant and acid butt, gives a positive reaction with ornithine and lysine decarboxylase, and--most important--agglutinates with rabbit anti-ERM serum. Although three serotypes of the bacterium have been described, almost all outbreaks are caused by the serotype that was first isolated and described, and that is referred to as Type 1. Presumptive diagnosis can be made by applying a direct fluorescent antibody test on smears of infected kidney tissue. Still another presumptive diagnosis was described by Busch (1973), who used a modified passive hemagglutination test to screen sera from populations of salmonids. Sera that showed agglutinins against the ERM bacterium were considered as having come from fish that had been exposed to ERM.

CAUSE OF THE DISEASE

For several years after the original isolation of *Y. ruckeri* at Hagerman, Idaho, all isolates appeared to be serologically homogeneous (Ross et al. 1966; Busch 1973). Since then, two other serotypes have been described. O'Leary (1977) described a distinct or second serotype--Type 2--from Pacific salmon (*Oncorhynchus* sp.)--that differed antigenically from the Type 1 in its ability to ferment sorbitol. And Bullock et al. (1978) described a third serotype, from Australia, that failed to react with antisera from either Types 1 or 2.

Researchers at the National Fish Health Research Laboratory, using electrophoresis, distinguished 22 enzymes from 51 isolates of *Y. ruckeri*. Their work indicated only a low genetic variation among all isolates, and they found no correlation between enzyme patterns and the three serotypes (B. Schill, personal communication). Additionally, a comparison of *Y. ruckeri* isolates from different geographic locations revealed that most were Type 1 (McCarthy and Johnson 1982; Bullock et al. 1978). Bullock et al. (1981) also demonstrated that Type 1 was far more virulent than either Type 2 or Type 3.

Although the above reports suggested two serotypes within *Y. ruckeri*--differing in serology and pathogenicity--recent data from the National Fish Health Research Laboratory did not support these differences. Rather, the types were similar in biochemical reactions, total protein profiles, isoenzyme analyses, and experimental mortalities in salmonids. Although there are certain antigenic differences among strains, these differences have not been correlated with the reported serotypes.

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. 1978); consequently the original source of the organism is unknown. Busch and Lingg (1975) showed that 25% of the rainbow trout surviving an artificial ERM challenge became asymptomatic carriers and that the bacterium localized in the lower intestine. Such trout serve as reservoirs of infection.

MODE OF TRANSMISSION

Natural infections spread from fish to fish by direct contact, or by exposure to carriers. Rucker (1966) transmitted the disease by exposing healthy trout to waterborne bacteria from 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 transmission (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. Spread of ERM from Idaho was initially 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 Italy (Giorgetti et al. in press), West Germany (Fuhrmann et al. 1983), Great Britain (Roberts in press), and France (Lesel et al. 1983). The host range has also expanded to include Atlantic salmon and Pacific salmon, and nonsalmonids such as emerald shiners, *Notropis atherinoides* (Mitchum 1981). Furthermore, Stevenson and Daly (1982) reported the isolation of *Y. ruckeri* from the intestine of a muskrat (*Ondatra zibethicus*). The increase in host and geographic ranges probably represents, in part, an increase in surveillance.

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 hyperosmotic infiltration. Continued developments in the delivery system showed that either simple immersion or spray application of bacterin resulted in practical protection. Bacterins now available consist of formalin-killed *Y. ruckeri* and accompanying growth products. Delivery can be by immersion or by spray. The kinetics of the immune response to ERM bacterin were reviewed by Anderson et al. (1979). Although two principal serotypes of *Y. ruckeri* have been reported, commercial bacterins contain only Type 1, and to date there have been no reports of failure of the bacterin that could be attributed to the presence of Type 2 in the fish. Additionally, 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).

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. There is no evidence for vertical transmission (from parent to egg) of ERM (Dulin et al. 1976).

Treatment

When outbreaks of ERM have occurred, a combination of sulfamerazine at 20 g/100 kg of fish per day for 5 days, followed by oxytetracycline or chloramphenicol at 5.0 g/100 kg of fish per day for 3 days, has been used successfully to treat outbreaks of ERM (Rucker 1966). McDaniel (1971) stated that a combination of sulfamerazine at 6.6 g/100 kg of fish per day and furazolidone at 4.4 g/100 kg of fish per day, fed for 5 days, gave excellent control of ERM outbreaks. The potentiated sulfonamide Ro5-0037 (a combination of sulfadimethoxine and ormetoprim) has controlled

outbreaks when it was fed at the daily rate of 5 g/100 kg of fish for 5 days (Bullock et al. 1983). However, none of the above drugs have been registered for use in the United States for treatment of ERM in fish intended for human consumption.

ANNOTATED BIBLIOGRAPHY

Amend, D. F., and R. W. Eshenour. 1980. Development and use of commercial fish vaccines. Salmonid 3(6):8-12.

A comprehensive review of factors affecting development of commercial bacterins for control of vibriosis and ERM.

Amend, D. F., and D. C. Fender. 1977. Uptake of bovine serum albumin by rainbow trout from hyperosmotic solutions: a model for vaccinating fish. Science 192(4241):793-794.

Authors demonstrated that fish could concentrate a high molecular weight protein in their tissues following exposure to a hyperosmotic sodium chloride solution.

Amend, D. F., K. A. Johnson, T. R. Croy, and D. H. McCarthy. 1983. Some factors affecting the potency of *Yersinia ruckeri* bacterins. J. Fish Dis. 6(4):337-344.

Potency of *Yersinia ruckeri* bacterin was not affected by pH or incubation time, nor by formalin or chloroform inactivation. However, potency was increased by cell lysis at pH 10.

Anderson, D. P., and J. R. Nelson. 1974. Comparison of protection in rainbow trout (*Salmo gairdneri*) inoculated with and fed Hagerman redmouth bacterins. J. Fish. Res. Board Can. 31(2):214-216.

Rainbow trout fed 1 mg *Yersinia ruckeri* bacterin per fish for 2 weeks had no detectable circulating agglutinating antibody, whereas agglutinating antibody was present for 3 months in trout receiving a single 1.0-mg subcutaneous injection. Trout fed bacterin lost protection against an artificial challenge of *Y. ruckeri* within 6 weeks after immunization, but injected trout were protected through 3 months.

Anderson, D. P., B. S. Roberson, and O. W. Dixon. 1979. Induction of antibody producing cells in rainbow trout, *Salmo gairdneri* Richardson, by flush exposure. J. Fish Biol. 15(3):317-322.

Rainbow trout immunized with as little as 5 µg of *Yersinia ruckeri* O-antigen by flush exposure showed splenic antibody producing cells.

Anderson, D. P., and A. J. Ross. 1972. Comparative study of Hagerman redmouth disease oral bacterins. Prog. Fish-Cult. 34(2):226-228.

Effectiveness of four bacterins for providing protection against ERM was compared. A chloroform-killed whole-cell preparation gave the best protection; whole cells killed with 0.5 or 3.0% phenol, or a sonicated and formolized preparation, provided much less protection.

Antipa, R., R. Gould, and D. F. Amend. 1980. *Vibrio anguillarum* vaccination of sockeye salmon *Oncorhynchus nerka* (Walbaum) by direct and hyperosmotic immersion. J. Fish Dis. 3(2):161-165.

Immunization of salmon against *Yersinia ruckeri* by hyperosmotic infiltration provided only slightly better protection than immunization by direct immersion.

Bosse, M. P., and G. Post. 1983. Tribissen and tiamulin for control of enteric redmouth disease. J. Fish Dis. 6(1):27-32.

Fifteen drugs were tested for control of ERM. A 14-day treatment with tiamulin given at 5.0 mg/kg of trout per day, or Tribissen given at 1.0 mg/kg of trout per day, provided the most effective control of experimental infection.

Bullock, G. L., G. Maestrone, C. Starliper, and B. Schill. 1983. Potentiated sulfonamide therapy of enteric redmouth disease. Can. J. Fish. Aquat. Sci. 40(1):101-102.

The potentiated sulfonamide Ro5-0037, fed for 5 days at 50 mg/kg per day, effectively controlled natural and artificial ERM infection.

Bullock, G. L., E. B. Shotts, Jr., and C. Starliper. 1981. Biochemical, serological, and virulence studies with *Yersinia ruckeri*. Pages 53-54 *in* Proceedings of the Joint Meeting of the Fifth Annual Fish Health Section/American Fisheries Society Sixth Annual Eastern Fish Health Workshop, Mississippi State University, Mississippi, 21-23 July 1981. (Abstract)

Biochemical, serological, and virulence studies on *Yersinia* isolates showed that Type 1 (sorbitol negative) predominated in disease outbreaks and was virulent, whereas Type 2 (sorbitol positive) was isolated from asymptomatic trout and was less virulent than Type 1.

Bullock, G. L., H. M. Stuckey, and R. L. Herman. 1976. Comparative susceptibility of Atlantic salmon (*Salmo salar*) to the enteric redmouth bacterium and *Aeromonas salmonicida*. J. Wildl. Dis. 12(3):376-379.

Yersinia ruckeri is as pathogenic as *Aeromonas salmonicida* to Atlantic salmon. A 30-min exposure of salmon to waterborne cells of *Y. ruckeri* killed half of the test salmon within 14 days.

Bullock, G. L., H. M. Stuckey, and E. B. Shotts, Jr. 1977. Early records of North American and Australian outbreaks of enteric redmouth disease. Fish Health News 6(2):96-97.

Examination of cultures obtained in the early 1950's from diseased trout in West Virginia and Australia showed that they were *Yersinia ruckeri*. This finding indicated that ERM may not have originated in Idaho as had been previously believed.

Bullock, G. L., H. M. Stuckey, and E. B. Shotts, Jr. 1978. Enteric redmouth bacterium: comparison of isolates from different geographic areas. J. Fish Dis. 1(4):351-356.

A comparison of characteristics of 18 *Yersinia ruckeri* cultures from North America and Australia showed the strains to be biochemically and morphologically homogeneous.

Busch, R. A. 1973. The serological surveillance of salmonid populations for presumptive evidence of specific disease association. Ph.D. Thesis, University of Idaho, Moscow. 196 pp.

A modified passive hemagglutination test was adapted to screen salmonid serum samples for the presence of ERM bacterium agglutinins. The test was 8 times more sensitive than tube agglutination procedures. It can be used to screen large populations for the presence of ERM agglutinins, which offers presumptive evidence of exposure to ERM.

Busch, R. A. 1978*a*. Enteric redmouth disease (Hagerman strain). Mar. Fish. Rev. 40(3):42-51.

A review of ERM that includes a description of the etiological agent, pathogenesis, the carrier state, and a serological test for presumptive screening of fish for *Yersinia ruckeri*.

Busch, R. A. 1978*b*. Protective vaccines for mass immunization of trout. Salmonid 1(6):10, 14, 22.

A review of the state of the art for fish vaccines. The history and mechanism of immunity, as well as currently available commercial vaccines, are discussed.

Busch, R. A. 1983. Enteric redmouth disease (*Yersinia ruckeri*). Pages 201-222 *in* D. P. Anderson, M. Dorson, and Ph. Dubourget, eds. Antigens of fish pathogens. Collection Fondation Marcel Merieux, Lyon, France.

An up-to-date, comprehensive review of ERM: history, etiology, epizootiology, pathogenesis, diagnosis, prevention, and treatment.

Busch, R. A., and A. J. Lingg. 1975. Establishment of an asymptomatic carrier state infection of enteric redmouth disease in rainbow trout (*Salmo gairdneri*). J. Fish. Res. Board Can. 32(12):2429-2433.

Forty-five days after an artificial challenge with *Yersinia ruckeri*, 25% of the surviving rainbow trout were asymptomatic carriers in which the bacterium had localized in the lower intestine. Inclusion of sampling from the lower intestine of suspect carriers of *Y. ruckeri* was suggested as part of the routine inspection or certification procedures.

Cook, T. M., and P. Gemski. 1982. Studies of plasmids in the fish pathogen, *Yersinia ruckeri*. Page 97 *in* Proceedings of the 13th International Congress of Microbiologists, Boston, Massachusetts, 8-13 August 1982.

An examination of 17 *Yersinia ruckeri* cultures for plasmids showed that all virulent strains contained a 70 Mdal plasmid, whereas avirulent strains did not.

Croy, T. R., and D. F. Amend. 1977. Immunization of sockeye salmon (*Oncorhynchus nerka*) against vibriosis using hyperosmotic infiltration. Aquaculture 12(4):317-325.

Salmon were successfully immunized by hyperosmotic infiltration in which *Vibrio* bacterin was used with Hanks balanced salt solution and sodium chloride.

Dulin, M. P., T. Huddleston, R. E. Larson, and G. W. Klontz. 1976. Enteric redmouth disease. Univ. Idaho, Moscow, For. Wildl. Range Exp. Stn., Contrib. No. 16. 15 pp.

History, etiology, epizootiology, pathology, immunology, diagnosis, and control of ERM are discussed, as well as the current status of the disease in Idaho.

Ewing, W. H., A. J. Ross, D. J. Brenner, and G. R. Fanning. 1978. *Yersinia ruckeri* sp. nov. the redmouth (RM) bacterium. Int. J. Syst. Bacteriol. 28(1):37-44.

On the basis of biochemical reactions and guanine-cytosine ratio, the etiologic agent of ERM was described as a new species of the genus *Yersinia*.

Fuhrmann, H., K. H. Böhm, and H. J. Schlotfeldt. 1983. An outbreak of enteric redmouth disease in West Germany. J. Fish Dis. 6(3):309-311.

First record of ERM in West Germany, which occurred in 1981.

Giorgetti, G., G. Ceschia, and G. Boud. In press. First isolation of *Yersinia ruckeri* in farmed rainbow trout in Italy. *In* A. E. Ellis, ed. Proceedings of the European Association of Fish Pathologists, Plymouth, England, 20-23 September 1983.

A description of the first occurrence of enteric redmouth disease in Italy.

Gould, R. W., P. J. O'Leary, R. L. Garrison, J. S. Rohovec, and J. L. Fryer. 1978. Spray vaccination: a method for the immunization of fish. Fish Pathol. 13(1):63-68.

Describes the application of a spray technique used to successfully vaccinate coho salmon and rainbow trout against vibriosis. Fish were sprayed 5-10 s at 7 kg/cm^2 pressure with a formalin killed bacterin.

Green, M., and B. Austin. 1983. The identification of *Yersinia ruckeri* and its relationship to other representatives of the Enterobacteriaceae. Aquaculture 34(3,4):185-192.

After extensive characterization of *Yersinia ruckeri*, the authors concluded that the ERM bacterium more closely resembled *Salmonella arizoniae* than *Yersinia*.

Hansen, C. B., and A. J. Lingg. 1976. Inert particle agglutination tests for detection of antibody to enteric redmouth bacterium. J. Fish. Res. Board Can. 33(12):2857-2860.

Charcoal particles and latex spheres sensitized with antigen extracts of *Yersinia ruckeri* were used to detect rainbow trout antibody against *Y. ruckeri*. The inert particle test was found to be sensitive and practical for detecting the antibody.

Harrell, L. W. 1979. Immunization of fishes in world mariculture: a review. Proc. World Maricult. Soc. 10:534-544.

An overview of vaccine development for vibriosis, furunculosis, ERM, and bacterial kidney disease.

Hester, F. E. 1973. Fish health: a nationwide survey of problems and needs. Prog. Fish-Cult. 35(1):11-18.

Enteric redmouth was identified as one of the important bacterial diseases of salmonids that often results in heavy losses.

Hunter, V. A., M. D. Knittel, and J. L. Fryer. 1980. Stress-induced transmission of *Yersinia ruckeri* infection from carriers to recipient steelhead trout (*Salmo gairdneri* Richardson). J. Fish Dis. 3(6):467-472.

Rainbow trout that were carriers of *Yersinia ruckeri* transmitted the bacterium to recipient trout when stressed by water heated to 22 °C. No transmission occurred when carrier trout were held at 15 °C. Trout that had been immunized against ERM and then challenged with *Y. ruckeri* carried the organism in the intestine for 3 days but did not transmit the disease.

Johnson, K. A., and D. F. Amend. 1983*a*. Comparison of efficacy of several delivery methods using *Yersinia ruckeri* bacterin on rainbow trout, *Salmo gairdneri* Richardson. J. Fish Dis. 6(4):331-336.

A comparison of injection, immersion, shower, and spray applications of commercial *Yersinia ruckeri* bacterins showed that injection provided the best protection, followed by immersion, shower, and spray.

Johnson, K. A., and D. F. Amend. 1983*b*. Efficacy of *Vibrio anguillarum* and *Yersinia ruckeri* bacterins applied by oral and anal intubation of salmonids. J. Fish Dis. 6(5):473-476.

Oral and anal applications of *Yersinia* bacterins in rainbow trout showed that anal application provided the best protection. It was suggested that oral bacterin would be more effective if it could be protected from degradation in the upper intestine.

Johnson, K. A., J. K. Flynn, and D. F. Amend. 1982*a*. Onset of immunity in salmonid fry vaccinated by direct immersion in *Vibrio anguillarum* and *Yersinia ruckeri*. J. Fish Dis. 5(3):197-205.

Immunity in salmonids is a function of size, not age. The minimum size at which maximum immunity occurred was between 1.0 and 2.5 g.

Johnson, K. A., J. K. Flynn, and D. F. Amend. 1982*b*. Duration of immunity in salmonids vaccinated by direct immersion with *Yersinia ruckeri* and *Vibrio anguillarum* bacterins. J. Fish Dis. 5(3):207-213.

Duration of immunity in salmonids immunized with *Yersinia ruckeri* and *Vibrio anguillarum* bacterins increased with size of fish.

Klontz, G. W. 1963. Oral immunization of rainbow trout against redmouth. Page 121 *in* Proceedings of the Northwest Fish Cultural Conference, Olympia, Washington, 5-6 December 1963.

First report of oral immunization of rainbow trout against *Yersinia ruckeri*.

Lesel, R., M. Lesel, F. Gavini, and A. Vuillaume. 1983. Outbreak of enteric redmouth disease in rainbow trout, *Salmo gairdneri* Richardson, in France. J. Fish Dis. 6(4):385-387.

First report of ERM in France. It may be more common in France than previously thought.

McCarthy, D. H., and K. A. Johnson. 1982. A serotypic survey and cross-protection test of North American field isolates of *Yersinia ruckeri*. J. Fish Dis. 5(4):323-328.

Examination of 23 *Yersinia ruckeri* isolates from North America showed that strains predominantly conformed to the original description of the Hagerman strain.

McDaniel, D. W. 1971. Hagerman redmouth ... a new look at an old fish problem. Am. Fishes U.S. Trout News 15(5):14-28.

Includes a comprehensive description of ERM and problems associated with the disease: historical aspects, similarity of ERM to other bacterial diseases, effect of low-level mortality on production, clinical signs, host and geographic range, and control measures.

McDaniel, D. W., editor. 1979. Suggested procedures for the detection and identification of certain infectious diseases of fishes. American Fisheries Society, Fish Health Section. U.S. Fish and Wildlife Service, Washington, D. C. 96 pp.

Standard procedures for diagnosis and detection of the major bacterial, parasite, and viral diseases of fishes. Methods are periodically revised by the Fish Health Section to reflect newly developed techniques.

Mitchum, D. L. 1981. Concurrent infections: ERM and furunculosis found in emerald shiners. Fish Health Sect./Am. Fish. Soc. Newsl. 9(4):2.

Aeromonas salmonicida and *Yersinia ruckeri* were found in emerald shiners imported into Wyoming from Wisconsin. Of the first 24 shiners examined, 5 were infected with *A. salmonicida* and all were infected with *Y. ruckeri*.

Newman, S. G., and J. J. Majnarich. 1982. Direct immersion vaccination of juvenile rainbow trout, *Salmo gairdneri* Richardson, and juvenile coho salmon, *Oncorhynchus kisutch* (Walbaum), with a *Yersinia ruckeri* bacterin. J. Fish Dis. 5(4):339-341.

Coho salmon and rainbow trout were immunized for 2 min with a 1:100, 1:500, or 1:1000 dilution of a commercial *Yersinia ruckeri* bacterin; after 18 to 80 days they were injected with virulent *Y. ruckeri*. Coho salmon immunized with the 1:100 dilution showed 83.7% survival when challenged 18 days after immunization and 70% after 88 days.

Salmon immunized with the 1:1000 dilution showed 73-78% survival at both 15 and 45 day challenge periods. Eighty-eight percent of the rainbow trout immunized with the 1:100 dilution survived when challenged 70 days post immunization, and 96.7% survived a 14 day challenge following immunization with the 1:500 bacterin dilution.

O'Leary, P. J. 1977. Enteric redmouth bacterium of salmonids: a biochemical and serological comparison of selected isolates. M.S. Thesis. Oregon State Univ., Corvallis. 93 pp.

First description of a second serotype of *Yersinia ruckeri*.

O'Leary, P. J., J. S. Rohovec, and J. L. Fryer. 1979. A further characterization of *Yersinia ruckeri* (enteric redmouth bacterium). Fish Pathol. 14(2):71-78.

Biochemical tests of 17 *Yersinia ruckeri* cultures were carried out at 9, 18, 22, 27, and 37 °C. Motility varied with temperature, as did some biochemical tests. Results from the study supported the present classification of the ERM bacterium.

Roberts, M. S. In press. Enteric redmouth disease--a cause for concern in the United Kingdom. *In* A. E. Ellis, ed. Proceedings of the European Association of Fish Pathologists, Plymouth, England, 20-23 September 1983.

Ross, A. J., and G. W. Klontz. 1965. Oral immunization of rainbow trout (*Salmo gairdneri*) against an etiologic agent of redmouth disease. J. Fish. Res. Board Can. 22(3):713-719.

Rainbow trout received food containing phenol-killed ERM cells daily for 2 weeks and then weekly for 8 weeks; when challenged with injected live ERM cells, 90% survived. Among infected, nonimmunized trout, only 20% survived the same challenge.

Ross, A. J., R. R. Rucker, and W. H. Ewing. 1966. Description of a bacterium associated with redmouth disease of rainbow trout (*Salmo gairdneri*). Can. J. Microbiol. 12:763-770.

Using morphological, biochemical, and some serological characteristics of ERM isolates, the authors placed the ERM bacterium in the Enterobacteriaceae but could not assign a genus or species. Enteric redmouth was transmitted by exposing healthy trout to infected trout.

Ross, A. J., and C. A. Smith. 1972. Effect of two iodophors on bacterial and fungal fish pathogens. J. Fish. Res. Board Can. 29(9):1359-1361.

In vitro tests were conducted on the efficacy of Betadine and Wescodyne in killing cells of the major gram-negative fish pathogens, the kidney disease bacterium, and two fungal fish pathogens. Cultures exposed for 5 min to 25 ppm iodine either were killed or the number of viable cells was greatly reduced.

Rucker, R. R. 1966. Redmouth disease of rainbow trout (*Salmo gairdneri*). Bull. Off. Int. Epizoot. 65(1-2):825-830.

Includes a description of the epizootiology, clinical signs, etiology, and control of ERM, and a discussion of the influence of temperature, crowding, excess ammonia, low oxygen, and nutrition on severity of disease outbreaks.

Stevenson, R. M. W., and J. G. Daly. 1982. Biochemical and serological characteristics of Ontario isolates of *Yersinia ruckeri*. Can. J. Fish. Aquat. Sci. 39(6):870-876.

Examination of *Yersinia ruckeri* isolates from hatchery and wild fishes in Ontario, and from the intestine of a muskrat, indicated that variations in biochemical and serological properties of strains are greater than previously believed.

Tebbitt, G. L., J. D. Erickson, and R. B. Vande Water. 1981. Development and use of *Yersinia ruckeri* bacterins to control enteric redmouth disease. Pages 395-401 *in* D. P. Anderson and W. Hennessen, eds. Proceedings of the International Symposium on Fish Biologics: Serodiagnostics and Vaccines, held at the National Fish Health Research Laboratory, Kearneysville, West Virginia, 26-30 April 1980.

Authors summarize development of *Yersinia ruckeri* bacterin and delivery system. Results of a 2-year evaluation of a commercial bacterin showed that immunized trout had fewer outbreaks of ERM and also showed a better conversion than nonimmunized control trout.

Wagner, E. D., and C. L. Perkins. 1952. *Pseudomonas hydrophila*, the cause of "red mouth" disease in rainbow trout. Prog. Fish-Cult. 14(3):127-128.

The original description of redmouth caused by *Aeromonas hydrophila* (originally called *Pseudomonas hydrophila*). The symptoms noted in affected fish and characteristics of the bacterium are described. Outbreaks were associated with the presence of *Gyrodactylus* on trout.

Wobeser, G. 1973. An outbreak of redmouth disease in rainbow trout (*Salmo gairdneri*) in Saskatchewan. J. Fish. Res. Board Can. 30(4):571-575.

The first description of ERM in Canada. The outbreak occurred in rainbow trout that had been imported from Idaho and were reared in cages in a lake. Descriptions are given of the bacterium, of the gross pathology and histopathology of infected trout, and of results of transmission experiments.