

Vibriosis in Fish: A Review

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INTRODUCTION

Vibriosis is a bacterial disease of fish and shellfish caused by *Vibrio anguillarum*. The mode of transmission and infection is uncertain.

EPIZOOTIOLOGY

The disease was first described in eels but is now known to occur worldwide in a variety of marine fish and shellfish.^{1,2,3} Vibriosis occurs primarily in fish in shallow brackish water, particularly in late summer as water temperature exceeds 50°F.⁴ This situation is of greatest importance to commercial culturists raising salmon in pens.

An outbreak of vibriosis in rainbow trout was reported at an Arizona trout farm where a salinized portion of the Colorado River was the water source for the farm.⁵ *Vibrio anguillarum* was also isolated as the etiologic agent of an infection in tropical fish in a freshwater aquarium.⁶

Vibrio anguillarum is an alimentary microflora of apparently healthy wild and cultured fish. Outbreaks have been associated with feeding marine fish offal.^{5,7}

Two strains of *Vibrio* pathogenic to fish have been described. *Vibrio anguillarum* strain 775, which was originally isolated from a diseased coho salmon at the National Marine Fisheries Service in Manchester, Washington, is designated as Type I.⁸ In 1976 a fish pathogen

characterized as *Vibrio* sp. strain 1669 but not agglutinated by rabbit anti-*Vibrio anguillarum* type I serum and differing in test media reactions was designated as Type II.^{9,10,11} *Vibrio ordalii* sp. nov. is the name proposed for the bacterium previously designated as *V. anguillarum* type II (1669).¹²

The incubation period varies with temperature, strain virulence, and the degree of stress under which the fish are living. Mortalities in an outbreak of cultured fish may be higher than 50%, especially among young fish.¹³

CLINICAL PATHOLOGY

First signs of imminent mortality are anorexia, darkening of the skin and sudden death. In young fish particularly, sudden death may be the only sign. Periorbital edema ("popeye") and/or ascites ("dropsy") may also be observed.⁴

In an outbreak, older fish may develop an acute or chronic phase. The gills are usually pale due to anemia. Acutely affected fish have ulcerated skin lesions which may become deep and necrotic.⁷ These lesions further demonstrate marked muscle necrosis accompanied by interfibrillar hemorrhage and congestion without a leukocytic response.¹⁴ Internally, the spleen is enlarged and there is liquefactive necrosis. There is also liquefaction of the kidney. Intestines may have focal areas of increased numbers of lymphocytes in the submucosa, and there may be petechial hemorrhage of the visceral and parietal peritoneum.⁶ In the liver there may be marked cytoplasmic degeneration of hepatocytes, and the sinusoids are distended with blood.

In chronically infected fish, skin lesions may become granulomatous. Fibrinous adhesions may be apparent among the abdominal viscera. Severe hemolytic anemia induced by

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the lytic toxin of *Vibrio* results in heavy deposition of hemosiderin in the melanomacrophage centers of the remaining splenic and renal hematopoietic tissue.

The lesions of vibriosis have been attributed to endo- or exotoxins.^{15,16} Yet a study by Horbell et al., in which coho salmon were challenged with "exotoxin" or endotoxin, did not result in mortalities.⁸ Histologic changes were not described in their study.

DIAGNOSIS

Vibrio anguillarum is a Gram negative, straight or slightly curved rod, motile by a single polar flagellum, cytochrome oxidase-positive, ferments glucose in O/F medium and is inhibited by vibriostatic agent O/129 and novobiocin.¹⁷ It is viable in distilled water, but growth is enhanced in 1-2% sodium chloride.¹⁸

A presumptive diagnosis of vibriosis is frequently made based on the history, symptoms, and external and internal lesions. Vibriosis may be confused with furunculosis when only these criteria are considered.¹⁹ The etiologic agent of furunculosis is not inhibited by the addition of O/129 to the growth medium.¹⁹

TREATMENT

Three methods of treatment have been suggested: 1. terramycin in the feed at 3.0 grams per 100 pounds of fish for 10 days or until mortalities have returned to normal rates for five days, 2. sulfamerazine in the feed at 5 grams per 100 pounds of fish for 10 days, 3. a combination of 3 grams of sulfamethazine and 2 grams of furoxone in the feed per 100 pounds of fish for 10 days in cases not responding well to the above regimens.²⁰ While antibiotic therapy is used in clinical outbreaks, this is of value only to those fish still feeding.

PREVENTION AND CONTROL

Disease resistance depends on the interaction of host, pathogen, and environment.²¹ Variable protective results have been obtained when fish were immunized and later challenged.^{22,23,24,25} The heritability of vibriosis resistance in river strains of salmon parr was estimated by Gjderen and Aulestad for sire and dam components.²⁶ It has been suggested that selection of resistance to one disease may at the same time result in fish more susceptible to other diseases, as well as decreased growth rates.²⁷

Another line of host defense is provided by the proteins transferrin and lactoferrin, which bind iron, rendering it unavailable to pathogens.^{28,29,30} Crosa reported evidence showing that *V. anguillarum* type I possesses a virulence plasmid that specifies a very efficient iron-sequestering system, thereby enabling bacteria to survive in conditions of limited iron availability.³¹

However, Winter et al. published evidence suggesting transferrin genotypes exist in coho salmon and steelhead trout, but did not demonstrate any difference in resistance to vibriosis.¹⁷ Plasmid-mediated virulence has been discussed elsewhere.^{11,32}

The concept of vaccines for fish has been present for several years. As early as 1942, trout have been successfully immunized against certain infectious diseases.³³ Interest in vaccines waned in the 1940's and 1950's due to availability of effective chemotherapeutics and antibiotics. However, due to antibacterial resistance, these antimicrobial agents have become less effective in many fish production facilities. In addition, the fisheries industry has grown and has become an attractive market to vaccine manufacturers.

Vaccines for fish have been administered by several routes, including parenteral injection, incorporation of vaccines into the fish diet, hyperosmotic infiltration, direct immersion of fish into vaccine suspensions, and spraying or showering fish with vaccine preparations.^{25,34,35,36,37,38,39,40,41,42}

Oral vaccines in feed are relatively easy to administer and less stressful to fish. However, proper dosage can not be assured. The parenteral route provides accurate dosage and assures that each fish is vaccinated. However, this method may be too costly or impractical for large numbers of fish.³⁵ Amend and Fender showed that bovine serum protein is taken up by rainbow trout through the lateral line thus the basis of the immersion method for vaccinating fish.⁴³ Whereas antigenic competition in animals has been observed with simultaneous administration of two or more antigens,⁴⁴ no antigenic competition between the two *Vibrio* strains has been demonstrated.^{41,45} Immersion bivalent vaccines have been shown to be protective against challenge with both strains of *Vibrio*, but monovalent vaccines only protect against homologous strains.

Results of adaptive transfer of immunity suggest that humoral antibody is the primary

defense against *V. anguillarum*. While intraperitoneal injections of bacterins produce higher humoral antibody levels than oral vaccinations, protection against vibriosis does not necessarily differ.³⁹ Also, spray vaccination may provide suitable results at lower cost and be performed in less time than parenteral injections.⁴² *Vibrio* sp. phenotypically similar to *Vibrio anguillarum* type II (1669) has been isolated from shell fish, but was not pathogenic to salmonid fish when tested under laboratory conditions.⁴⁷ Perhaps this isolate could be used in a live vaccine if there is cross protection against pathogenic *Vibrio anguillarum*.

CONCLUSION

Vibriosis is an important disease to commercial salmon culturists. While the pathogenesis is not certain, fish mortalities can be reduced by proper immunization, antibiotic therapy, and managing fish with minimal exposure to stressful conditions.

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In 1936 Dr. C. H. Stange was Dean of the Iowa State Veterinary College. On a trip to Europe in 1930 he was greatly impressed with the art he saw on the veterinary buildings, so it became his desire to see art connected with the ISU Veterinary school.

Christian Petersen came to ISU in 1934 to finish some sculptures in the Dairy Court annex. Veterinary professors stopped in at his studio frequently to chat, and constantly prodded him to make a replica of the "Gentle Doctor" statue in the Veterinary court yard. Christian eventually made the statuette which has become a symbol of the Veterinary profession.

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