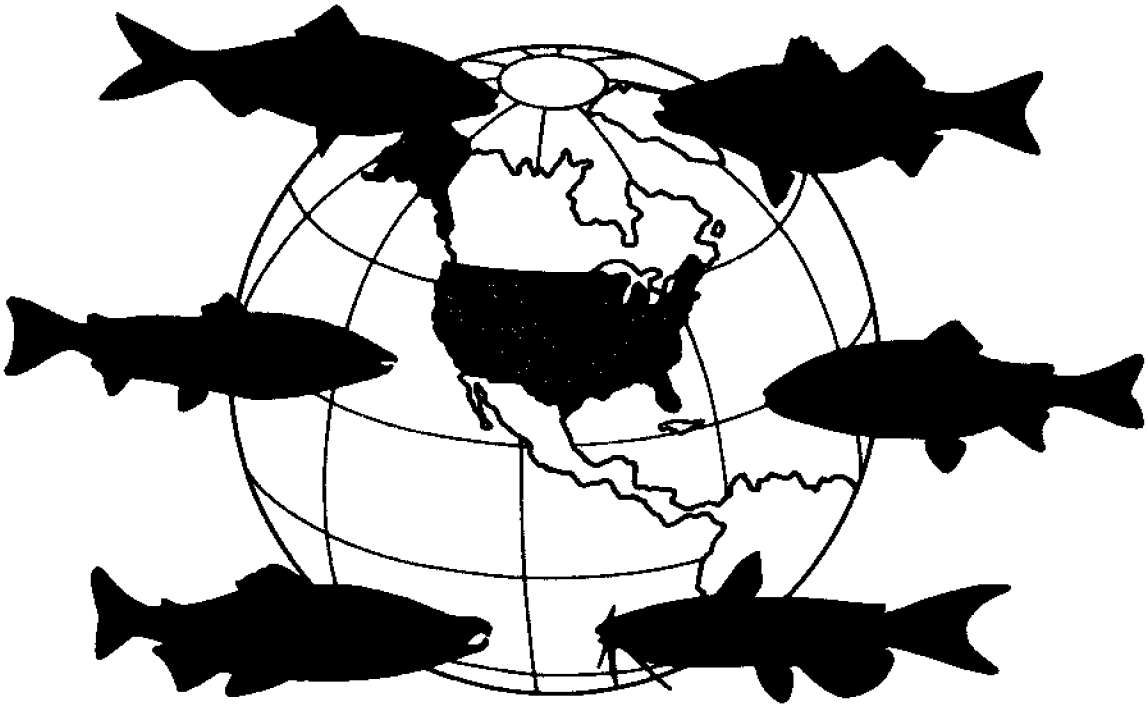
Fish Disease Leaflet 80

Ceratomyxa shasta, a Myxosporean Parasite of Salmonids



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Introduction

Ceratomyxsis is a disease of salmonid fishes caused by the myxosporean *Ceratomyxa shasta*. The parasite has a tropism for the intestinal tissue of the fish and causes high mortalities in susceptible strains of salmonids. The disease was first observed in 1948 in fall-spawning rainbow trout (*Salmo gairdneri*) from Crystal Lake Hatchery, Shasta County, California (Wales and Wolf 1955). The etiological agent was established as a new species by Noble (1950), who described *C. shasta* as the first species of *Ceratomyxa* to parasitize freshwater fish and the only member that is histozoic. Other species of *Ceratomyxa* that occur in marine fishes parasitize the lumen of the gall bladder and urinary bladder.

Ceratomyxa shasta is an important parasite in the Pacific Northwest because it not only causes losses in hatchery-reared and wild juvenile salmonids but also contributes significantly to prespawning mortality in adult salmon. Although the parasite has not been detected outside the Pacific Northwest, its distribution in that region has expanded. It is not known whether this increase reflects a true spread of the disease or only improved detection methods.

Diagnosis and Identification

External and Internal Signs

Clinical signs of ceratomyxosis vary among salmonid species. Infected juvenile rainbow trout (and steelhead) become anorexic, lethargic, and darken. Ascites may distend the abdomen, the vent may be swollen and hemorrhaged, and exopthalmia is common (Schafer 1968). Infected juvenile chinook salmon (*Oncorhynchus tshawytscha*) first become emaciated and later sometimes develop large fluid-filled blebs and kidney pustules (Conrad and Decew 1966).

Internally, the intestinal tract of juvenile rainbow trout becomes swollen and hemorrhaged and the intestinal contents mucoid, and caseous material lines the intestine and pyloric caeca (Conrad and Decew 1966). The entire digestive tract, the liver, gall bladder, spleen, gonads, kidney, heart, gills, and skeletal muscle may become diseased, hemorrhaged, and necrotic (Wales and Wolf 1955). Infected adult chinook salmon may have nodular lesions in the intestine that perforate, causing death. These nodules may be accompanied by gross lesions in the liver, kidney, spleen, and muscle. Infected adult coho salmon (*O. kisutch*) show grossly thickened intestinal walls and pyloric caeca, and large abscessed lesions in the body musculature (Wood 1979).

Histopathology

In juvenile rainbow trout the first sign of infection appears in the posterior intestine. The progress of the infection is temperature dependent, the first sign of infection appearing between days 12 and 18 postexposure in fish held at 12°C, and at 7 days in fish held at 18°C (Yamamoto and Sanders 1979). Trophozoites are first seen in the mucosa; their appearance is followed by a strong inflammatory response in the lamina propria. As the infection progresses, the parasite multiplies in all layers of the intestine and causes severe inflammation and desquamation of the mucosal epithelium. Trophozoites penetrate the intestinal tract, spread into the surrounding adipose and pancreatic tissues, and enter the bloodstream, which carries them to other tissues and organs. In late stages of infection, the parasite is in most tissues and organs adjacent to the intestine, including the liver, kidney, pyloric caeca,

and spleen. Diagnosis of the disease is sometimes delayed because the spore stage of *C. shasta* is not evident until the terminal stages of the infection.

Identification

Diagnosis requires that spores be found and identified by their size, shape, and location. The American Fisheries Society *Fish Health Blue Book* (Amos 1985) recommends the following procedures: (1) examination of wet mounts from the lower intestinal wall, ascites, gall bladder, and lesions by phase contrast or bright light microscopy at ×400; (2) examination of air-dried smears stained by the Ziehl-Neelsen method but without heating; or (3) fixation of smears in Schaudin's fixative and staining with Heidenhain's iron hematoxylin (for permanent preparations). For examination of live fish, an intestinal lavage technique can be used (Coley et al. 1983). Spores of *C. shasta* are about 14 to 23 μm long and 6 to 8 μm wide at the suture line (Fig. 1). The ends of the spores are rounded and reflected posteriorly; the suture line is distinct (Noble 1950). In smears stained by the Ziehl-Neelsen method, the polar capsules stain red against a bluish sporoplasm and background. Trophozoites, which are rounded but variable in shape, mature to form a sporoblast that usually contains two spores (Fig. 2). Because of the variability in size and shape of the trophozoites and their similarity to this stage in other myxosporeans, observation of trophozoites by light microscopy is not sufficient for diagnosis. Consequently, serological techniques have been developed in which monoclonal antibodies are used. The antibodies produced react specifically with the prespore stages of the parasite and do not cross-react with trophozoite or spore stages of other myxosporeans. Use of the monoclonal antibodies and fluorescein or enzyme conjugated secondary antibodies enables the reliable detection of early infections (J. L. Bartholomew, J. S. Rohovec, and J. L. Fryer, in preparation).

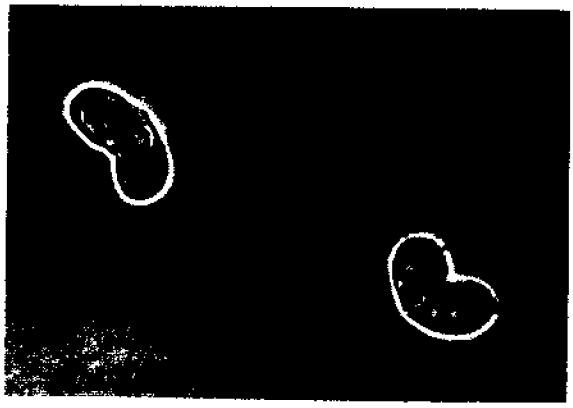

Fig. 1. Phase contrast photomicrograph showing morphology of *Ceratomyxa shasta* spores.

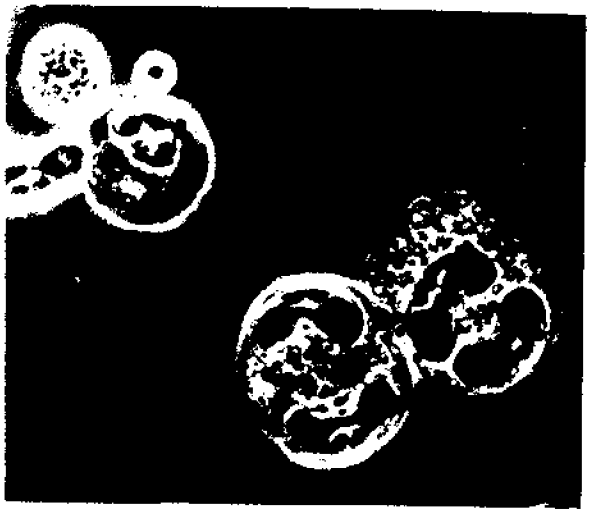

Fig. 2. Phase contrast photomicrograph of trophozoite and disporoblast stages of *Ceratomyxa shasta*.

Ecology

Effects of Temperature

Infection by *C. shasta* was once believed to occur only when water temperatures exceeded 10° C, thus accounting for the seasonal occurrence of the disease; however, later reports indicated that fish can become infected in water at temperatures as low as 4 to 6°C (Ratliff 1983; Ching and Munday 1984a). Although fish are infected at these lower temperatures, the progress of the disease is temperature dependent and most infections are detected later, after the water warms. Udey et al. (1975) reported that rainbow trout exposed to the infective stage of *C. shasta* and held at water temperatures of 6.7 to 23.3°C had little or no ability to overcome the infection, and that the mean time from exposure to death was directly correlated to temperature (e.g., about 155 days at 6.7°C and 14 days at 23.3°C). In rainbow trout the disease process was suppressed at 3.9°C; however, when the infected fish held at this temperature were transferred to water at 17.8°C, many died. Coho

salmon appeared better able to combat the infection at low water temperatures, but the mean time to death remained temperature dependent.

Effects of Salinity

Only limited information is available about the effects of salt water on the progress of a *C. shasta* infection. Johnson (1975) reported that infections were prevented at salt concentrations greater than 15 ppt. Although this would protect juvenile salmonids from infection in estuarine areas, the fate of fish that were infected in fresh water and then migrated into salt water was not determined. Acute ceratomyxosis has been reported in juvenile chum salmon captured off the coast of British Columbia, Canada (Margolis and Evelyn 1975), demonstrating that the disease is not attenuated by salt water. Ching and Munday (1984b), who exposed chinook salmon to the infective stage of *C. shasta*, found that the disease caused 100% mortality when the fish were held in either fresh water or salt water. Similarly designed experiments with steelhead indicated that migration to salt water may reduce the progress of the disease, but the extent of attenuation may be masked in fish overwhelmed by a high number of infectious units (Hoffmaster 1985).

Host Range and Susceptibility

It is accepted that only salmonids are susceptible to *C. shasta* infection (Table 1), but this susceptibility may vary within a species. Experiments to test resistance of different strains of the same species to *C. shasta* indicated that juvenile salmonids originating from waters containing the infective stage of the parasite were more resistant than strains from areas free of the infective stage (Johnson 1975; Zinn et al. 1977; Buchanan et al. 1983; Hoffmaster 1985). The susceptibility to infection by *C. shasta* in progeny produced from crosses between resistant and susceptible coho salmon is intermediate between that of the parental stocks (Hemmingsen et al. 1986). The management implications of these studies are that relocation of salmonids from areas where *C. shasta* is not endemic into areas endemic for the parasite is not likely to be successful, and that these introductions may adversely affect the survival of resident resistant strains if interbreeding occurs.

Although juvenile salmonids from waters endemic for *C. shasta* are resistant to infection, ceratomyxosis has been determined to be an important cause of prespawning mortality in the adults. Coley et al. (1983) reported that 94% of adult spring chinook salmon at Rapid River Hatchery, Idaho, were infected with *C. shasta*. Similar incidences of infection in adults have been reported by other researchers (Sanders et al. 1970; Yasutake et al. 1986; Chapman 1986). Although infection by *C. shasta* occurs during the freshwater phase of the fish's life cycle, it is not known whether these fish were infected before they entered salt water or after they reentered fresh water.

Geographic Distribution

Ceratomyxa shasta has been identified in salmonids from marine and freshwater environments in northern California, Oregon, Washington, Idaho, and British Columbia. However, waters where infected fish have been found do not necessarily contain the infective stage of the parasite

Table 1. *Host range of* Ceratomyxa shasta.

Common name	Scientific name	Reference
Rainbow trout	*Salmo gairdneri*	Noble 1950
Chinook salmon	*Oncorhynchus tshawytscha*	Conrad and Decew 1966
Coho salmon	*Oncorhynchus kisutch*	Conrad and Decew 1966
Steelhead	*Salmo gairdneri*	Conrad and Decew 1966
Brook trout	*Salvelinus fontinalis*	Schafer 1968
Brown trout	*Salmo trutta*	Schafer 1968
Atlantic salmon	*Salmo salar*	Sanders et al. 1970
Cutthroat trout	*Salmo clarki*	Sanders et al. 1970
Sockeye salmon	*Oncorhynchus nerka*	Sanders et al. 1970
Chum salmon	*Oncorhynchus keta*	Margolis and Evelyn 1975
Pink salmon	*Oncorhynchus gorbuscha*	Bell and Traxler 1985

(Johnson et al. 1979). This is exemplified in the Columbia River Basin, where infected adult coho and chinook salmon and steelhead migrate and distribute spores throughout the drainage, but the infective stage of *C. shasta* has not been demonstrated in many tributaries to which these fish have access. This suggests that the presence of spores alone is insufficient to cause transmission and disease.

The presence of the infective stage of *C. shasta* is demonstrated by using sentinel populations of susceptible salmonids and examining them for development of the disease and appearance of spores. The distribution of the infective stage (Fig. 3) has been documented by Johnson et al. (1979), Ching and Munday (1984a), and Hoffmaster et al. (1988).

The confinement of this parasite to salmonids of the Pacific Northwest is unique. The distribution of many other fish pathogens has been expanded as a result of shipments of eggs or fish. This geographic isolation is compatible with the hypothesis that an as yet unknown factor is required for the completion of the life cycle of this parasite.

Transmission and Life Cycle

The life history of *C. shasta*, like that of most other myxosporeans, is unknown. Natural transmission occurs when susceptible salmonids are exposed to water or sediments containing the infective stage (Schafer 1968; Fryer and Sanders 1970; Johnson 1975) and exposure periods as short as 30 min are sufficient for infection to occur. Neither attempts to transmit ceratomyxosis from fish to fish nor the feeding of infected tissues containing spores and trophozoites have resulted in transmission of the disease (Wales and Wolf 1955; Schafer 1968; Wood 1968; Johnson 1975). But infections developed when susceptible fish were exposed to bottom sediments collected from a site endemic for the parasite (Fryer and Sanders 1970).

Laboratory transmission of ceratomyxosis has been established by intraperitoneal injection and anal intubation of ascites from infected fish (Schafer 1968; Fryer and Sanders 1970; Johnson et al. 1979; Bower 1985). The natural route of infection has not been established, but Schafer (1968) suggested that the establishment of infection in rainbow trout was not dependent on ingestion of the spore. Differential filtration of waters endemic for *C. shasta* shows that the infective stage is larger than 14 μm.

The inability to transmit ceratomyxosis between susceptible fish has led to speculation that an intermediate host may be involved in the life cycle. As yet there is no conclusive evidence to support this hypothesis.

Control

Because *C. shasta* infections are not transmitted directly between fish, outbreaks of the disease in hatchery fish occur only as a consequence of introducing the infective stage through the water supply. Because no chemotherapeutic agent yet tested has been useful in controlling ceratomyxosis, the most effective means of disease prevention in a hatchery situation is avoidance of water supplies containing the infective stage. In hatcheries where alternative water supplies are unavailable, water treatment methods to eliminate the infective stage have been developed. Bedell (1971) found that ultraviolet irradiation or chlorination of water supplies reduced the number of *C. shasta* infections but did not eliminate them. Sanders et al. (1972) and Bower and Margolis (1985) determined that sand filtration, in combination with either ultraviolet irradiation or chlorination, was effective in reducing the incidence of disease. Tipping (1986) reported that ozone was effective in controlling ceratomyxosis in hatchery fish. However, the most successful approach for control of ceratomyxosis in both hatchery and wild populations is the introduction of resistant salmonids (Buchanan et al. 1983).

Acknowledgments

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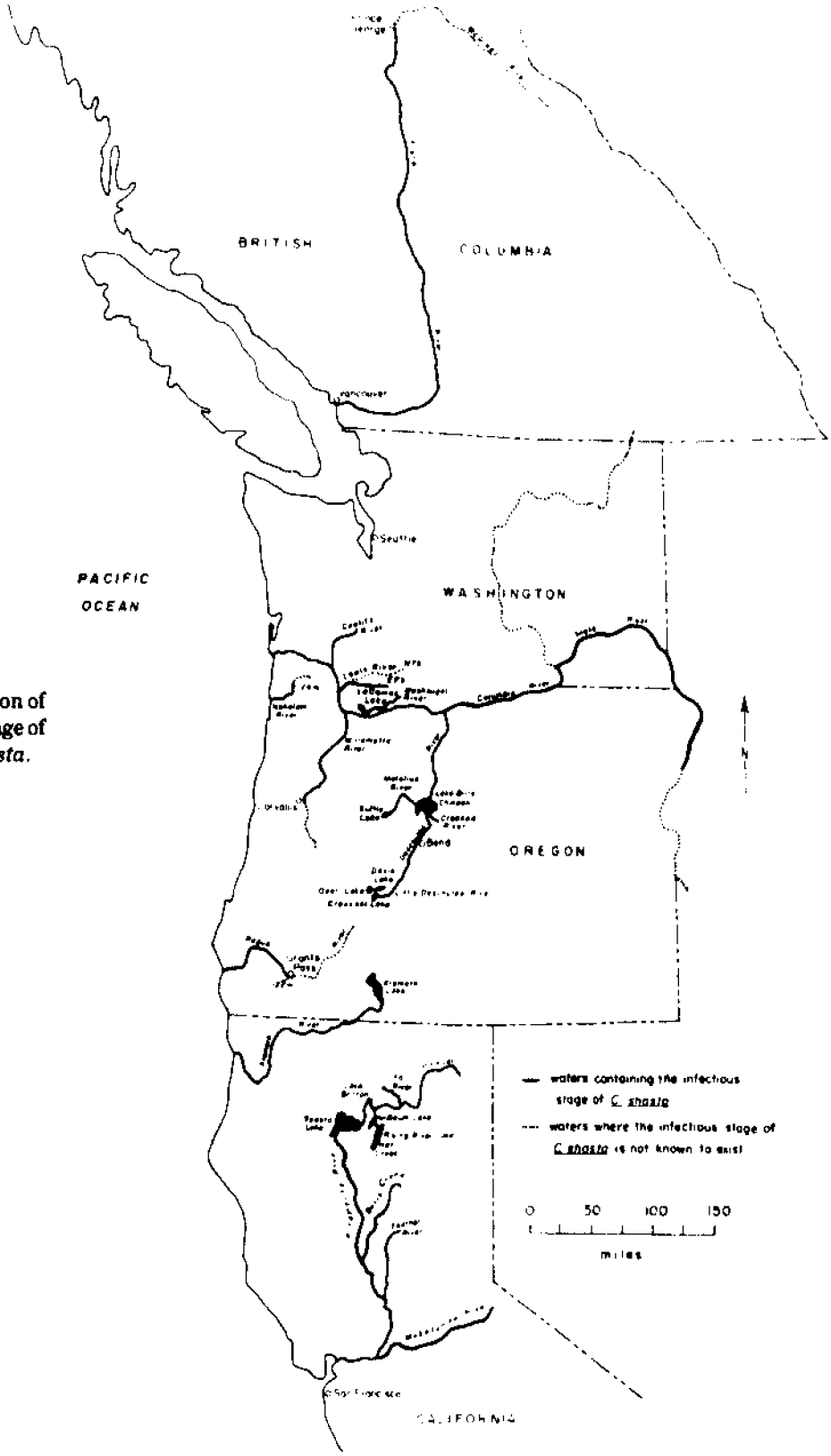

Fig. 3. Distribution of the infectious stage of *Ceratomyxa shasta*.

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A list of current *Fish Disease Leaflets* follows.

70. Brown Blood Disease (Methemoglobinemia) of Fishes, by P. R. Bowser. 1984. 6 pp.
71. *Edwardsiella* Infections of Fishes, by G. L. Bullock and R. L. Herman. 1985. 6 pp.
72. Columnaris Disease of Fishes, by G. L. Bullock, T. C. Hsu, and E. B. Shoots, Jr. 1986. 9 pp.
73. Channel Catfish Virus Disease, by J. A. Plumb. 1986. 7 pp.
74. Proliferative Kidney Disease in Salmonid Fishes, by R. P. Hedrick, M. L. Kent, and C. E. Smith 1986. 9 pp.
75. Epitheliocystis Infection of Fishes, by R. L. Herman and K. Wolf. 1987. 4 pp.
76. Annotated Bibliography of the Diseases and Parasites of Striped Bass, by K. W. McAllister, J. A Mann, and L. C. McKenzie. 1987. 16 pp.
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78. Bacterial Kidney Disease of Salmonid Fishes Caused by *Renibacterium Salmoninarum*, by G. L Bullock and R. L. Herman. 1988. 10 pp.
79. Systemic Noninfectious Granulomatoses of Fishes, by R. L. Herman. 1988. 4 pp.

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