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SACRAMENTO RIVER CHINOOK DISEASE (SRCD)

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INTRODUCTION

Coleman National Fish Hatchery, Anderson, Calif., has experienced annual epizootics in fingerling chinook salmon (Oncorhynchus tshawytscha) since the station was established in 1941. Various causes were postulated until 1957, when a virus etiology was demonstrated. Since 1959 the disease has been under intensive investigation. The results of this investigation are given in this leaflet.

IDENTIFICATION

The appearance of the disease is temperature dependent and is anticipated in young fingerlings after water temperatures reach approximately 56°F. Newly feeding fingerlings are the usual victims, but it is commonly observed in fish that are 3 or more inches in length. The disease manifests itself by an abrupt, rapid increase in mortalities. After the acute phase, usually about 7 days, gross external symptoms appear. These symptoms are exophthalmus, distended abdomen, and very characteristically the appearance of a dull red area on the dorsal surface anterior to the dorsal fin. The most striking gross internal observation is of "white"

visceral organs. The stomach and intestines are a bright white and devoid of food. The liver, kidney, spleen, gills, and heart are pale. The previously mentioned dorsal patch appears to be cherry red when the skin is peeled away from the area. All of these, including the dorsal patch, are manifestations of anemia, a condition caused by the destruction of the hemopoietic tissue of the kidney.

Histologic examination of prepared specimens presents the following information: Kidney -- destruction of the parenchymal cells of the hemopoietic tissue with some involvement of the epithelial cells of the tubules; Pancreas -- some limited necrosis, but primarily characterized by inclusions of various sizes in the cytoplasm and nuclei of acinar cells; Musculature of the dorsal patch -- infiltration of the myotomes by red blood cells released by the destruction of the kidney; other organs do not show any obvious pathology.

CAUSE OF DISEASE

A virus of extremely small size is the etiologic agent of this disease.

This leaflet supersedes FL 497, A "Virus" Disease of Chinook Salmon, March 1960.

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SOURCE AND RESERVOIR OF INFECTION

The source of infection and reservoir for the etiologic agent are the spawning adults. This has been proved for females and is speculated to be true for males.

MODE OF TRANSMISSION

Transmission is effected directly by the egg from carrier females. It is speculated that the egg is bathed with the virus at the time of stripping the female and enters the egg at the time of fertilization. Experimental infection can be effected by inoculation. Exposure of healthy fish to moribund fish will not produce the disease, contrary to previous reports. Indirect evidence indicates that susceptible fish can be infected at the time infected eggs hatch. This aspect of transmission is being investigated further.

INCUBATION PERIOD

Precise data are not available for the incubation period in an epizootic. Laboratory studies indicate that the incubation time is 3 days for fish injected interperitoneally with the virus.

PERIOD OF COMMUNICABILITY

The initial period of communicability is limited by the mechanics of spawning. A second period is postulated to occur at the time infected eggs hatch. Indirect evidence supports this hypothesis.

SUSCEPTIBILITY AND RESISTANCE

Natural epizootics have been observed exclusively in chinook salmon. Experimentally, the host range has been extended to many other species. Susceptibility or, conversely, resistance seems to be a function of size-age of the animal and the accident of exposure. True resistance is shown by the silver salmon (O. kisutch).

GEOGRAPHIC DISTRIBUTION

Natural epizootics have been observed only at the Coleman National Fish Hatchery. One outbreak was noted at Nimbus Hatchery, California Department of Fish and Game, in fish from Coleman eggs placed at the State station as part of the experimental program. Both the Battle Creek and Keswick Dam stocks of the Sacramento River chinooks spawned at Coleman are susceptible and are carriers.

METHOD OF CONTROL

Identification and elimination of the carrier adults are the ultimate means of controlling the disease. Epizootics can be limited by manipulation of water temperatures, particularly at the time of hatching. Treatment of fish with antibiotics and other chemotherapeutic agents has no effect.

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