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LYMPHOCYSTIS DISEASE OF FISH
(Revised)

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Viruses are submicroscopic absolute parasites which are utterly dependent upon living cells for their reproduction and which in fact cannot be propagated in the absence of living cells. Most viruses freely pass through filters which retain most bacteria; hence they have been termed "filterable viruses". All known viruses are infective agents; they are often specific in their host, and in some instances they have high specificity for certain tissues. There are many inapparent viral infections, but when viruses produce clinical disease, the predominant effect is usually one of two major types. Some viruses evoke an inflammatory response or destroy host tissue; others cause it to proliferate abnormally.

Lymphocystis is a virus-caused disease of fishes and is unique in several ways. It occurs only among several of the higher orders of fishes. It is a chronic disease and is seldom if ever fatal. Host cells which become infected are stimulated to abnormal growth and commonly undergo a 50,000 to 100,000-fold increase in volume (fig.1). Their increase in size is probably the greatest of any virus-stimulated growth. Lymphocystis is easily transmitted between species, but less easily between genera. This specificity, the chronic nature, and lack of mortality appear to be features of host/parasite relationships of very long standing.

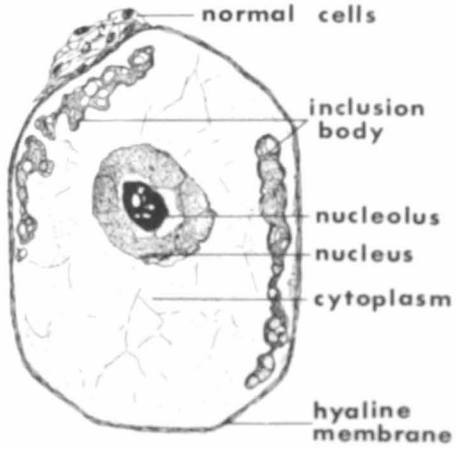

Figure 1:--Drawing of a section through part of a lymphocystis lesion. One of the tremendously enlarged infected cells is shown in detail, and adjoining normal sized cells are shown for comparison. The infected cell has a length of about 140 μ.

IDENTIFICATION

The external lesions of lymphocystis are raised growths of generally granular, wart-like or nodular tissue which is composed of several to many greatly enlarged host cells and their covering membrane. Mature infected cells usually have diameters of 100 microns or greater and may attain a size of a millimeter or more. Infection sites resemble a spattering of farina, and the larger masses have a raspberry texture.

Individual lesions, especially early stages, may be barely visible, but infected cells enlarge with development, and in advanced cases in some fishes much of the skin surface may be grossly involved. Among centrarchids, lymphocystis is often limited to the fins, and the caudal fin is commonly the principal site of infection. Color of the lesion is usually light and may be white, gray, or cream-colored. There is a tendency towards opalescence, and larger lesions may show pink due to the vascular network. Mature lesions may become slightly hemorrhagic, but they seldom become necrotic. Lymphocystis cells may occur internally, but the infection is characteristically a disease which involves the skin. Lymphocystis has been incorrectly identified as an infestation by copepods, as sporozoan parasites, and as eggs of helminth parasites. Careful examination under low magnification (hand lens or dissecting microscope) will often reveal great size differences between cells. This lack of uniformity will help distinguish the lesion from the more uniformly sized copepods or eggs. The histologic picture is unmistakable (fig.1).

CAUSE OF THE DISEASE

Lymphocystis disease is firmly established as a viral infection. The virus is filterable, glycerolsensitive, and preservable by lyophilization, simple desiccation, and by freezing at -20°C. or lower. It was the first virus of fishes to be seen, and recent electron micrographs show that it occurs only in the cytoplasm of infected cells. The virus is a regular polyhedron having a mean diameter of 200 microns and occasionally occurring in crystalline array.

SOURCE AND RESERVOIR OF INFECTION

In all probability the disease is maintained in susceptible host fishes. Viruses are released when mature infected cells burst. Healthy fish have been experimentally infected by exposing them to water which contained emulsified lymphocystis cells.

MODE OF TRANSMISSION

Infected host cells are stimulated to abnormal growth, and the cytoplasmic inclusion enlarges with time. Host cells ultimately burst and/or slough, and virus particles are released. This can occur intermittently through the duration of infection, or it can b massive as upon death (other causes) and decomposition of the host fish.

INCUBATION PERIOD

Under natural conditions incubation peri is probably long, being on the order of seve weeks or more. Under experimental condit incubation has varied with the nature of the infectious material, the route of inoculation the host species, and especially the tempera Among centrarchids which had been injected subdermally and held at 25°C., minimal inc bation was 12 to 15 days, whereas at 12.5°C. the minimal incubation was 37 days. In European perch (Acerina cernua) the minim time for lesion development was 9 days, and Stizostedion incubation time was about 2 wee

PERIOD OF COMMUNICABILITY

Within limitations imposed by season or temperature, host specificity and the like, lymphocystis is communicable while lesions present.

SUSCEPTIBILITY AND RESISTANCE

Many species of marine and fresh-wate fishes are susceptible. Among the artifical propagated fresh-water species (excluding exotics and aquarium fishes) the walleye and many of the Centrarchids are susceptible. A yet the disease has not been reported among salmonids. Host specificity is strong but no absolute. It has not been possible to infect Lepomis with material from Stizostedion. Weissenberg's early attempts were unsucce and only more recently he has been able to infect Fundulus with Stizostedion. The perc age of success was low, and the results wer atypical. Between members of the genus Lepomis the disease could be readily transmitted. Among susceptible species reinfect is possible.

RANGE

Lymphocystis has been reported from Europe, South America, and North America

Its presence in Pacific waters of North America suggests a possibility that it will be shown to occur throughout the world.

OCCURRENCE

It is difficult to generalize on available information about occurrences of lymphocystis. Weissenberg reported peak occurrences both in winter and in summer but stated that among centrarchids winter seemed to be the season of greatest prevalence. He was unable to infect bluegills after spring and attributed the failure to the warm season. Nigrelli stated that among fresh-water species the disease was more common in summer and that it disappeared in fall and winter. Other reports tend to agree with Nigrelli's statement. Similarly, experimental infections have been given to bluegills at the Eastern Fish Disease Laboratory through 4 consecutive years. There is general agreement that lymphocystis lesions are persistent; they commonly remain for several months and may continue for a year or more.

METHODS OF CONTROL

Weissenberg recommends that all tumor-bearing fish be removed from the waters in which they occur and be destroyed. If high percentages of artificially propagated Centrarchids are found to carry the disease it may be possible to eliminate it by using only brood stock which are free of the disease, and by using water from a supply known to be free of tumor-bearing fish. No method of treatment is known.

ANNOTATED BIBLIOGRAPHY

Literature on lymphocystis disease is extensive. For additional references the interested reader or researcher is referred to bibliographies included in Weissenberg's publications.

Alexandrowicz, J.S.

1951. Lymphocystis tumors in the red mullet (Mullus surmuletus L.). Jour. Mar. Biol. Assoc., U.K., Vol. 30, No. 2, pp. 315-332, illus.

Report of lymphocystis in a new species. Detailed histological findings are presented. Four plates of excellent photomicrographs are appended.

*Davis, H.S.

1953. Culture and diseases of game fishes. U. of Calif. Press, Berkeley and Los Angeles, 332 pp., illus.

(pp.293) The author gives a short description of the disease and several facts regarding its occurrence; an illustration is included.

*Nigrelli, Ross F.

1954. Tumors and other atypical cell growths in temperate fresh-water fishes of North America. (Symposium. Research on fish diseases: A review of progress during the past 10 years). Trans. Am. Fish.Soc., Vol. 83 (for 1953), pp. 262-296, illus.

(pp. 277-279) Macroscopic and especially microscopic characters of lymphocystis disease are included. A list of susceptible fish is given. Facts of its cause, course, and occurrence are briefly discussed. A good illustration is given.

*Snieszko, S.F.

1953. Virus diseases in fishes: Outlook for their treatment and prevention. The Progressive Fish-Culturist, Vol. 15, No. 2, pp. 72-74.

The nature, biology, and challenge of viruses as causes of disease are discussed. Problems which fish viruses present in research and fish culture are indicated. Recommendations are made for control of virus-caused diseases.

Walker, Roland.

1962. Fine structure of lymphocystis virus of fish. Virology, Vol. 18, No. 3, pp. 503-505.

Lymphocystis lesions from the walleye (Stizostedion vitreum) were sectioned and studied by electron microscopy. Virus was found in the cytoplasm. Particles were regular hexagons and pentagons having a mean diameter of 200 microns. The work is illustrated.

*Watson, Stanley W.

1954. Virus diseases of fish (Symposium. Research on fish diseases: A

review of progress during the past 10 years). Trans. Am. Fish. Soc., Vol. 83 (for 1953) pp. 331-341.

(pp.332-333 and 338) The history, hosts, description, and cause of lymphocystis disease are briefly reviewed.

Weissenberg, Richard.

1939. Studies on virus diseases of fish. III. Morphological and experimental observations on the lymphocystis disease of the pike perch, Stizostedion vitreum. Zoologica, N.Y. Zoo. Soc., Vol. 24 (Part 2), pp. 245-253, illus.

Experimental transmission of lymphocystis was achieved by feeding. Growth stages of affected cells are described; the author states that the disease is similar to lymphocystis in European perch. Previous literature is reviewed, and a section on control is included. Microscopic anatomy of lymphocystis cells is illustrated.

1945. Studies on virus diseases of fish. IV. Lymphocystis disease in Centrarchidae. Zoologica, Vol. 30, Part 4, pp. 169-184, illus.

Occurrences among Centrarchids are reported. The course of the infection is described, and the detailed cytology is given in words and illustrations. Experimental infections were effected within a species and between two species of Lepomis. Infection of Lepomis was not transmitted with material from Stizostedion. Illustrations of lesion and infected cells are included.

Weissenberg, Richard.

1949. Studies on lymphocystis tumor cells of fish. I. The osmiophil granules of the cytoplasmic inclusions and their interpretation as elementary bodies of the lymp cystis virus. Cancer Research, Vol. 9, No. 9, pp. 537-542, illu

Uniformly small granules which show a grea affinity for osmic acid stain increased in nu with age of the infected cell. They are thou to be infective units of the virus (elementary bodies). Illustrations of infected cell anatomy are included.

1951. Studies on lymphocystis tumor cells of fish. II. Granular structures of the inclusion substance as stages of the developmental cycle of the lymphocystis virus. Cancer Research, Vol. No. 8, pp. 608-613, illus.

Primordial granules in inclusion bodies are thought to be vegetative units which multiply by fission and give rise to elementary bodie which are the individual viruses (virus parti cles). Infected cell anatomy is illustrated.

Wolf, Ken.

1962. Experimental propagation of lymphocystis disease of fishes. Virology, Vol. 18, No. 2, pp. 2 256.

A means of experimental transmission of lymphocystis disease was sought and found. The infection was maintained among centrar chid fishes through a period of more than 2 years. The virus was clearly shown to be filterable, glycerol-sensitive, and preserv- able by desiccation and by freezing at -20°C Fish tissue cultures inoculated with the viru failed to develop lymphocystis cells.

* Papers marked with asterisks are of special importance to practical fish culturists.