

Copy to: Lincoln
R. Marks
Walcott
Zalsman
Palmer
Tubbs
Shust
J. Marks
6-19-30

INSTITUTE FOR FISHERIES RESEARCH
UNIVERSITY MUSEUMS
UNIVERSITY OF MICHIGAN
ANN ARBOR, MICHIGAN

June 14, 1930

Report No. 14

INVESTIGATION OF CAUSE OF DEATH OF FISH AT
THE ODEN HATCHERY FEEDING STATIONS

In response to a telephone statement of loss of fish in the feeding stations under the management of the Oden Hatchery, and on accompanying request for an investigation, Fish Pathologist Wendell H. Krull spent June 4 and 5 on this investigation, in company with Superintendent of Hatcheries A. B. Cook. A considerable loss of fingerlings had been reported by Overseer Guy Lincoln, in the lower ponds of three feeding stations.

A complete internal examination of small sick fingerlings was made at the Jordan Creek Feeding Station. These in splendid condition as far as general appearance and parasites are involved.

Gill smears were made from numerous sick fish at the Creeks and hatchery. In every specimen bacteria in great numbers were found on the gill's. These bacteria are of the bacillus (rod) type and occur attached end to end producing long filaments, which no doubt makes them more serious. In fish showing the first symptoms of the disease the mucus secretion is abundant and when examined is found to contain many of the bacilli, as well as foreign material collected from the water. The fish cease to eat and appear to be sluggish and weak. Suffocation because of collections of

bacteria, mucus and foreign material seems to be a plausible explanation of death. Fungus (Saprolegnia) had attacked quite a number of the fingerlings, and was growing from the dorsal part of the gill opening along the region of the back, indicating a possible origin in the gill region.

Certain characteristics of this outbreak incidentally support the conclusion that the death of the fingerlings^G was to gill infection. Some of these characteristics will be related in the following paragraphs.

The outbreak occurred almost simultaneously at the three places, about one month after the fish were taken from the hatchery. A fourth feeding station not visited, at Ricker's Dam was also supplied with fish from this same hatchery and to the present (June 5, 1930) the fish there were not dying. As a matter of fact, those fish were taken from the hatchery. We shall be very glad to learn whether or not these fish suffered any loss.

In both creeks the loss of fish began in the last of the series of ponds. At Jordan Creek, a heavy loss was reported in the last pond and the fish in the next to the last pond were not eating. In the first two ponds, the fish which had been treated with copper sulphate about ten days previous to our visit, were apparently normal. At Moyer Creek the loss before our visit in the last pond took place over night. At this same creek one pond located between ponds having heavy losses was not affected up to the time of our visit. The water running through them was the same, and as far as we were able to tell, the depth of water, condition of the bottom, and vegetation was very similar. These peculiarities of the sick fish suggest a bacterial disease governed by some very peculiar environmental factors, or by differential resistance on the part of the fish.

If it were a question of food all ponds in the infected region, as also the Ricker's Dam station, should be affected.

If it were a question of water the same would hold true. The larger fish in

areas of the stream between the ponds would also, no doubt, be affected.

If it were due to the construction of the new ponds, the Moyer Creek Station and ponds at the hatchery would not be affected.

The fact remains that the fish act and look sick, and the gill bacteria appear to be the cause.

We were unable to obtain any temperature correlations. But if what H. S. Davis says is true we may expect a temperature change in the water of only a few degrees to do a great deal of damage. This same investigator has observed irregularities in fish attacks even in protozoan infections.

At the Oden hatchery as soon as the loss took place in the ponds, the fish were returned to the troughs and the heavy losses ceased. Whether the disease had had its run or whether the temperature change affected the loss can not be determined.

Crowding is a possible cause of, and condition for, the rapid spreading of these bacterial diseases. If crowding is strongly guarded against, both at the feeding station and in the hatchery, a decrease in the death toll may be hoped for.

At the hatchery we suggested treatment of some of the troughs with copper sulphate. We also requested that accurate data be kept on these troughs and the controls. We shall be grateful for the receipt of the report on this experiment.

At the Jordan feeding station a treatment of some of the ponds was attempted and probably was not very successful. On the day after the treatment the fish were not eating but the fish in a pond not treated were also not eating so that this condition is not the result of the copper sulphate treatment. Mr. A. B. Cook instructed the employes to plant some of the fingerlings which is the logical thing to do. The fish at these stations were in splendid condition except for the gill disease.

This loss of fish in the rearing stations is of especial interest in that it confirms the view of Dr. Metzelaar, which we regard as well based, that the fine

success obtained at the new rearing stations is in part the success of a new environment. It is certainly true that newly introduced animals are apt to do very well at first, because the balance has been thrown in their favor by leaving behind the natural enemies and diseases. The diseases of course will creep into the new rearing stations, and we must expect more trouble with disease in the rearing stations in the future.

Carl L. Hubbs.

Carl L. Hubbs
Director