MONTANA STATE DEPARTMENT OF FISH AND GAME

FEDERAL AID IN FISH RESTORATION SECTION

HELENA, MONTANA

JOB COMPLETION REPORT INVESTIGATIONS PROJECTS

State of Montana										
Project No.	F-8-R-3	Job No.	III-A							
Title of Job	Diagnosis of Montana h	natchery and wild	trout diseases.							
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Abstract:

Diagnoses of trout diseases at Montana hatcheries were attempted in response to reports of sick or dying fish. Usually the examinations were completed on the spot by microscopic examination of the fish, their tissues or smears from diseased tissues. In some cases stained slides of diseased tissues and bacterial cultures were used to assist further in making the diagnoses. Recommendations were made for continuance of this trouble-shooter type of work on regular state fish hatchery funds rather than as a Federal Aid project. The need for designed and controlled research on diseases and nutritional problems now present in the Montana hatchery system is also pointed out.

Objectives:

The project leader endeavored to diagnose occurrences of diseases as they were reported in Montana during the period covered by this project. In the case of diseases caused by organisms such as bacteria, protozoa, etc., the causative organisms were to be identified. Contributing factors such as faulty diets and unfavorable environmental conditions were recorded in each case.

Techniques Used:

Several trips were made to various hatcheries in response to reports of sick and dying fish. An attempt was made in each case to determine the cause of the trouble. The behavior and general appearance of the fish, the conditions of the environment including diet as well as microscopical examination of fresh and stained slide preparations, and in some cases bacterial cultures were used in making the diagnoses.

Findings:

Anaconda:

A myxobacterial infection of yearling rainbow trout was investigated during the summer of 1953 at the Anaconda hatchery. The bacterial organism observed alive in smears from diseased gill tissue exhibited the columnar behavior as described in literature on columnaris disease. The water temperature in the ponds concerned was 56° F.; however, part of this water came from a duck pond in which the surface temperature was 70° F.

Symptoms of the diseased fish included shortened gill covers, gills slightly swollen and gill filaments, usually on one side only, with lesions that gradually ate away all epithelial tissue leaving a thin strand of what appeared to be cartilaginous tissue. The lesions were covered by a ball of fungus and the myxobacteria were found by pulling off the fungus and smearing the lesions on glass slides. Body muscles became quite tense or rigid before death.

Recovery of the diseased fish was affected by shutting off the water from the duck pond source with a consequent reduction in water temperature from 56° to 53° F.

Other myxobacterial infections occurred at Anaconda during the spring and early summer. The sick fish appeared to be suffering from bacterial gill disease but in most cases PMA treatments did not effect complete or lasting cures probably due to continual reinfection from the duck ponds and other shallow ponds used for the hatchery water supply.

Arlee:

The popeye disease caused mortalities at the Arlee hatchery in March, 1954. Only cutthroat trout of several different strains were affected at the time of this report. The exterior appearance of these fish was very similar to that of the popeye disease which occurred at Emigrant in 1952 and 1953. Abdominal cavities were swollen from the accumulation of watery fluid and the eyeballs protruded markedly. Although the kidneys of the Arlee fish had large amounts of pigment there were none of the granular concretions that were deposited in such abundance in the kidney tubules of the Emigrant fish. Short rod-shaped bacteria were observed in the abdominal fluid but no myxobacteria or myxosporidians were found in kidney smears. The intestines contained a pale yellowish fluid, livers were pale with a slight yellowish cast, and gill color varied from light red to pink. There was some indication of bacterial gill disease which is a common condition at Arlee but is kept under control when necessary by PMA treatments. Pectoral fins appeared somewhat frayed and under the stereoscopic microscope it appeared that epithelial proliferation was producing small lumps near the extremities of the rays while epithelial sloughing was also occurring over large areas of fin.

The popeye condition and increase in mortality rate to eight times normal came on very suddenly after the fish had been fed two weeks on a diet of 40 pounds of lungs, 20 pounds each of spleens, livers and horsemeat, and 5 pounds of dry mix. The dry mix contained equal parts of flavonne, herring meal, cottonseed meal, and wheat shorts. A few hog livers which had been stored for some time were included in the liver component. It is believed that these hog livers may have contributed a toxic factor which caused the losses. The fish were taken off feed for three days and losses dropped to near normal five days after the popeye condition had so suddenly appeared. The symptoms seem to indicate clearly fatty degeneration of the liver but the picture is not clear especially concerning the rapid recovery. It would be interesting to know what part was played by the hog livers.

Big Timber:

During the winter of 1952 a shipment of two cases of brown trout eggs from Pennsylvania was received at the Big Timberhatchery. The eggs were dry on arrival but had not begun to shrink and their temperature was 52° F. Total

egg and fry loss up to the first feeding was 24 percent which was not considered alarming. Shortly after hatching the weak and dead fry pickoff included a small percentage of the usual blue-sac fry. The starting diet was a mixture of horse hearts, livers, and brewers yeast ground together, mixed with water and fed soup style by spoon. The resulting milky cloud seemed to irritate the fry causing considerable activity until after it had passed from the trough when the fish would again settle to the bottom. It was also noticed that a very high percentage of the fry were bloated with a bluish watery fluid. This bloated condition did not appear until the yolk sacs were nearly or completely absorbed and the fish were beginning to feed. It should also be noted that this condition had only a superficial resemblance to the common "blue-sac" loss which was picked off within a very few days after hatching. A portion of this egg shipment that was relayed from Big Timber to Emigrant hatchery did not develop the dropsical condition. The loss was 100 percent at Big Timber.

A shipment of eastern brook trout eggs from Washington were hatched at Big Timber just following the experience with the brown trout fry. The brook trout also suffered nearly a 100 percent loss exhibiting the same symptoms at the same stage of development as the brown trout. Brook trout from the Washington source developed no such condition at the Emigrant station.

An experiment was conducted at the Big Timber hatchery with healthy brook trout from Emigrant and some of the sick brook trout. It demonstrated that healthy fish held in water that had passed over sick fish contracted the disease while healthy fish held in an uncontaminated portion of the hatchery did not develop the dropsical condition. Since the disease appeared to be definitely contagious, effort was made to sterilize the contaminated wooden troughs by scrubbing, exposure to sunlight and painting.

Brown trout obtained from South Dakota the following winter (1953-54) began to develop a similar appearing dropsical condition but losses were not high and the fish did not accumulate as much of the watery fluid. These fish were treated frequently with salt and when last observed were apparently healthy. It was later learned that these fish had suffered a mechanical shock during the incubation period of the eggs in South Dakota.

No new information was learned about the cause, prevention or cure of dropsy in trout fry. These observations merely illustrate the existance of two or possibly three different diseases which are often referred to by the collective term "blue-sac". The need for organized research on blue-sac, particularly the contagious type which can cause 100 percent losses, is realized; however, adequate facilities are not presently available for this work in Montana.

Big Timber:

Early in September, 1953, rainbow trout, two to three inches long, in dirt ponds began dying with symptoms of severe external protozoan parasitization. The fish were dark colored, emaciated, off feed, had gone through a short period of flashing, and many were floating listlessly near the edges of the ponds. A few Trichodina were found but not enough to explain the almost total loss in one pond. The large protozoan, Ichthyophthirius, was found on the gills and anteriodorsal surfaces of all fish examined. The gills appeared somewhat swollen. It was concluded that the latter parasite was responsible for the trouble. The affected ponds were receiving part of their water supply from a swampy area and the pond first affected and which suffered the highest loss was getting the larger share of this poor water supply. A treatment with formalin using malachite green for tracer was administered to the least affected pond and the water level drawn down to permit flushing out the free swimming stages of the parasite. The swampy water supply was closed off as it was believed to be a contributing factor in starting the epidemic. The fish made a gratifyingly rapid recovery.

Big Timber:

In March, 1953, Oregon rainbow trout in Big Timber hatchery troughs suffered a small loss with the only consistent symptom being a reddish tinge in the abdominal region. Fungus mycelia were abundant inside the stomach and intestine of moribund trout. In fish that had been dead for some time, the mycelia had penetrated the body wall and appeared on the outside.

Standard remedy for <u>Saprolegnia invaderis</u>, removal of sick and dead fish, was recommended.

Bluewater:

The Bluewater hatchery continues to be plagued by a fin rot condition which destroys the dorsal fin and sometimes attacks the skin and musculature at the base of the dorsal fin. The progress of the disease resembles descriptions of so-called bacterial fin rot. Rod-shaped bacteria were found in smears from diseased fins but more thorough bacterial examinations of these fish have not been made as the project leader is not aware of any literature that positively links a specific organism with the disease notwithstanding the many papers mentioning a diplo-bacillus with this condition.

Phemerol dip treatments at 1:20,000 for 20 to 30 seconds have been of considerable benefit in clearing up the diseased fins but the source of the trouble remains untouched.

It is perhaps significant that the water source at Bluewater has no oxygen and despite the use of aerators which allow the water to fall in a thin sheet over a vertical cylinder of $\frac{1}{2}$ -inch mesh hardware cloth, the dissolved oxygen content in the cement raceways has never tested over 6.5 ppm and drops to as low as 4.1 ppm at the lower end of raceways containing yearling trout. Water temperature is a constant 56° F. at this station. Total alkalinity as parts per million calcium carbonate is 1600 at Bluewater and ranges from 55 to 225 at the other state hatcheries.

Emigrants

For two consecutive years a high loss was observed among rainbow and cutthroat trout at the Emigrant hatchery. The fish had a severe popeye condition resulting from the gradual accumulation of granular concretions in the kidney tubules which in turn caused an accumulation of watery fluid in the body cavity. Brown trout and brook trout at this station developed the concretions in their kidneys to a lesser extent and did not show any of the external popeye symptoms. Chemical analyses of the concretions was not made due to their extremely small size. They appear white and crystalline under the stereoscopic microscope. Excessive amounts of pigment were present in the kidneys and other internal organs. Most of the moribund fish showed symptoms quite like fatty degeneration of the liver. Bacterial examination of internal organs revealed mixed myxobacterial infections.

These trout had been on a 100 percent heart diet for their entire life and it is suggested that a vitamin deficiency might be the main causative factor. Addition of cod-liver oil and brewers yeast to the diet had no noticeable beneficial effect. Beef liver has been added to the heart diet for approximately seven months in feeding the present lot of trout; however, at least three months

more will be required before the results will be known as past experience has shown that the trout are about one year old before the popeye condition becomes apparent and losses begin.

Hamilton:

Golden trout yearlings being held for brood stock were examined in July, 1953, when a small loss occurred. Some of the fish were quite listless. This is unusual for golden trout which generally scurry rapidly for cover when approached. Dead and dying fish floated at the water surface and there were large gas bubbles in the stomachs of dead and dying fish. The foreman expressed a belief that the fish had been overfed during a temporary change in the hatchery routine and that the fish might have had the opportunity to eat "soured" food from the bottom of the tank. The condition soon cleared up and the percentage loss was quite small.

Bozeman:

Another case of large amounts of gas in the stomachs of hatchery trout was observed in March, 1954, at the Federal hatchery in Bozeman. The fish were two-month-old brown trout which had been fed a diet of beef liver with 30 percent pork liver. These fish had also had some salmon viscera but this item was only included in the diet for a few days some two weeks prior to the increase in mortality rate. Gall bladders of these fingerlings were considerably enlarged and contained clear green fluid. Losses were not too high.

Although 50 percent beef liver with 50 percent pork liver is said to be a very successful starting diet for salmon, it seems possible that trout cannot handle the pork as well as a starting food.

Recommendations:

Faulty diets and feeding practices directly or indirectly cause a major share of the trout hatchery losses in Montana. The need for designed and controlled feeding experiments is illustrated by the circumstantial evidence indicating pork livers were a cause of losses at two hatcheries. Circumstantial evidence, however, does not constitute conclusive proof and carefully executed experiments can only be conducted in the absence of pressure from production schedules.

Certain losses at Bluewater, Big Timber and Anaconda hatcheries are not so easily traceable to diet problems. Improved aeration of the water supply at Bluewater should increase the production capacity of that station and help eliminate the fin rot problem. The Big Timber water supply can easily be altered in such a way as to eliminate the swampy portion of the water source. Extensive changes in the Anaconda water source cannot be so easily done because of other uses of the water supply which comes out of a city park. Prophylactic treatments along with good diets and feeding practices offer the best means of disease control at this station.

The trouble-shooter work of this job has been of considerable value in providing the project leader first hand experience with various hatchery problems. In some cases where the problem was correctly diagnosed, the hatchery personnel have benefited by learning how to avoid further recurrences of the same difficulties. It is apparent that with increasing experience of the project

leader this job is becoming progressively more an accepted tool of the hatchery production system and should therefore be continued in the future on regular state funds rather than as a Dingell-Johnson project.

Summary:

Hatchery losses investigated and reported include myxobacterial infections, fin rot, and Ichthyophthirius infestation, two different types of popeye disease, "blue-sac", and gas on the stomach of trout at two hatcheries. It is recommended that this job be discontinued as a Federal Aid project and be carried on regular state funds for operation of production hatcheries. The need for designed and controlled experimentation with trout diets and diseases is also pointed out.

Prepared	by		Jack	E.	Bailey		Approved	bу	
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