Ref ID: 70852

JOB COMPLETION REPORT

INVESTIGATIONS PROJECTS

State of Montana

Project No. F-8-R-3 Work Plan I Job No. I-A

Title of Job: Development of a manual on trout culture for the Montana Fish and Game Department.

Abstract:

A manual on trout culture is being prepared primarily for the use of fish-culturists in Montana. A bibliography of approximately 2100 references on trout disease and nutrition has been assembled to serve as source material for the manual which will be issued as a series of literature reviews with complete bibliographies. As sections are completed they will be issued in loose leaf form and revisions or supplements will be prepared whenever significant additions to the subject appear in the literature. Sections on fin rot, blue-sac, gas bubble disease, and vitamin A have been completed.

Objectives:

To summarize and assemble material from the available literature into a manual on trout culture with emphasis on disease and nutrition.

Techniques Used:

Available publications pertaining to trout disease and nutrition were cross-indexed by subject and bibliography cards were filed by author. Summaries of the literature on specific subjects such as fin rot, vitamin A, blue-sac disease of trout fry, etc. are being prepared for a hatchery manual which is being distributed to trout hatcheries and fish biologists in Montana. The manual is written specifically for the use of fish-culturists but comprehensive bibliographies accompany each section for the use of workers who wish to make further study of the subject. Revisions or supplements to sections already prepared will be issued as new information becomes available in the published literature.

Findings:

A bibliography of approximately 2100 references on trout culture and related subjects has been compiled and typed on 3 x 5 cards for the author index. About 1350 of these references are contained in the trout disease and nutrition library of reprints, microfilm, technical journals, and text books. Only the texts and microfilm were purchased by D-J funds.

A copy of the completed sections of the hatchery manual is submitted with this completion report.

A subject index has been prepared on 3 x 5 cards and now covers 1500 of the references. There are 290 subject headings used in this index and each reference is cross-indexed by an average of about three subjects. An additional 300 references are contained on FAO World Fisheries Abstracts cards which are filed by subject. The remaining 300 references not indexed are on the present

"want list" many being out of print or otherwise unavailable.

Recommendations:

As a general rule there is considerable time lag between the publication of significant research findings on trout disease and nutrition and the practical use of this information by production hatcheries. Since the literature is often written in a form not easily understood by the fish-culturist or may be published in technical journals which are seldom read by hatchery personnel, the hatchery manual should be completed and kept up to date with frequent revisions and supplements. Every effort should be made to see that it is written in the language of the fish-culturist to assure actual use of the contained information.

Summary:

It was originally intended that the project leader should spend considerable time collecting a library on trout disease and nutrition in order to familiarize himself with the subject and apply the information to Montana trout hatcheries. A bibliography of 2100 references was compiled and 1350 publications in the form of reprints, microfilm, text books and technical journals were collected. In order to make this wealth of information on trout cultural subjects available to the fish-culturist, a manual is being prepared and distributed to Montana hatcheries and fisheries biologists. Sections have been completed on blue-sac, gas bubble disease, fin rot and vitamin A.

Prepared by Jack E. Bailey Approved by _____

Date May 1, 1954

1212.1 FIN ROT

1212.11 Symptoms. Davis (1928) gave the following description of fin rot. "In small fingerlings the pectoral fins are usually affected first, the dorsal fins being attacked next and the disease may eventually spread to the other fins although they are rarely affected as the pectorals and dorsals. In older fish there does not appear to be the same sequence in the order of infection and in many instances the dorsal or caudal fins are apparently affected before the pectorals.

Usually the first noticeable indication of the disease is a faint white line along the outer margin of the fin. This white streak gradually moves toward the base of the fin while at the same time the outer margin becomes badly frayed owing to the disintegration of the tissue between the fin rays. This process continues until eventually the entire fin may be destroyed. In late stages of the disease, sores filled with a glistening white pus may develop at the base of the fins and occasionally such lesions are found on the body at some distance from the fins. Due to the discharge of pus into the surrounding water these lesions may appear as small, circular depressions which extend for some distance into the underlying muscles. The sides of such lesions are so clearly defined as to give the impression of a small piece of tissue having been removed by some sharp instrument." Davis further states that such lesions are rare in younger fish as they usually die before reaching this stage. In a later publication (Davis 1946) the small circular lesions on the backs of advanced cases are not mentioned in the description of typical fin rot and he notes that several different diseases such as ulcer disease and furunculosis have at times been confused with fin rot.

1212.12 Cause of the disease. A rod-shaped bacterium is said to be the causative organism. The bacteria are found on the outer surface of the fins, in the connective tissue, and in the lymph channels. Other factors that have been associated with fin rot by various authors are: vitamin C deficiency (Hewitt 1937a.), fungus growing on sides of tanks (Hewitt 1937b), presence of traces of zinc and iron in the water supply (Hewitt 1937b), and underfeeding (Wolf 1938). Savage (1936) objects to the use of gravel in raceways as it constitutes an obstruction to treatments, accumulates decaying organic debris, and is a source of bacterial infection. He says fin rot is milder and easier to deal with in the absence of gravel. Leach (1939) describes a fin disease similar in appearance to fin rot but the causative organism (Gyrodactylus) is easily found and identified with the aid of a microscope. Overcrowding is sometimes said to cause fin rot but Davis (1946) observes that the fin condition resulting from overcrowding has a smooth contour and lacks the ragged appearance of fin rot. No infection is involved and the whitened edges of such fins are composed of scar tissue, probably the result of irritation.

1212.13 Control measures. Until the causative organism has been isolated and identified and the relationship of the various contributive factors worked out, it would be advisable to consider all the above-mentioned causes in the prevention of fin rot.

Typical fin rot apparently starts on the outer surfaces of the fins and consequently most treatments involve chemical baths designed to kill the bacteria before they can invade too deeply into the tissues. Davis (1946), Wright (1936) and Fish (1935) advise dipping the fish for 1 or 2 minutes in a 1:2,000 copper sulphate solution. Several treatments at intervals of 24 hours are usually necessary. McLaren et al (1947)

recommends a 30-second dip in 1:20,000 solution of phemerol. Burrows (1949) treated infected fish with 1:4,000 formalin for one hour. The formalin killed the sickest fish and cured the rest. A dual treatment using formalin and malachite green is described by Eicher (1947). The dual treatment was used as a flush in troughs by pouring 2 ounces of formalin and 2 ounces of 1/40 malachite green into the head end of each trough. Chlorine was used to disinfect troughs in the prevention of fin rot by Hewitt (1937b) and as a treatment for infected fish by Connell (1937). The procedure in treating fish with chlorine is given by Connell as follows:

1. The pools must be clean.
2. Estimate the volume of water in cubic feet.
3. High test calcium hypochlorite (HTH) is weighed out at the rate of 0.086 grams per cubic foot of water to be treated and dissolved in several pails of water. Sodium thiosulphate (Hypo) is weighed out at the rate of 0.36 grams per cubic foot. This too is dissolved in several pails of water.
4. Without even stopping the flow of water through the pool, the hypochlorite solution is spread quickly over the area to be treated and then mixed with brushes. Mixing is continued throughout the treatment. After 30 seconds test the chlorine concentration with a chlorine comparator using ortho-tolidine indicator. The concentration should be 2 ppm. If it is more the hypo solution should be mixed in immediately. If it is near 2 ppm the hypo is added after 2 minutes have elapsed and another test made to assure that all chlorine has been removed. The effect of chlorine treatments are cumulative over short periods of time and no more than one treatment of even a weak solution should be given in any 24-hour period. Healthy fish will stand 2 ppm for 4 minutes or more even in warm summer temperatures. A sick fish will not.

Although all of the above treatments have proven successful in certain instances, universal success cannot be expected. Davis (1946) notes that the disease varies greatly in severity and other authors have observed that treatments which are effective under certain conditions may not be

effective under different conditions or at different hatcheries.

It remains therefore for each hatchery to work out the most effective control for its own particular set of conditions. The sulfa drugs and the modern antibiotics should not be overlooked as possible controls. Attention should also be given to cleanliness, balanced diets, and the possible presence of toxic metallic ions in the water supply.

1212.14 Literature.

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Prog. Fish-Cult., No. 27: 11-15.

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Trans. Wisc. Acad. Sci. 37: 265-274. (1945).

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Prog. Fish-Cult., No. 23: 20.

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Prog. Fish-Cult., No. 24: 1-26.

1220.1 GAS BUBBLE DISEASE

1220.11 Symptoms. Easily visible bubbles of gas in the fins, under the skin, in the gills, kidneys, and heart. May cause popeye if gas bubbles collect behind the eyeballs. Trout of all ages may be affected.

1220.12 Cause of the Disease. Supersaturation of nitrogen or oxygen gas in the water. Rucker (1953) concludes that trout can stand 110 percent supersaturation of nitrogen gas and that 115 percent is probably too high. Woodbury (1941) found gas bubble disease asscciated with a supersaturation of oxygen (30-32 ppm). The disease caused the death of several yearling rainbow trout in a western Montana farm pond where an algal bloom following chemical fertilization increased the oxygen content to over 23 ppm at the surface (Temperature 46° F, altitude 3500). Wiebe (1932) was unable to cause gas bubble disease in rainbow and eastern brook trout which he subjected to oxygen concentrations of 21-33 ppm or 209-328 percent saturation.

1220.13 Control Measures. Use aerating devices to remove excess nitrogen from water supplies. Beware of excess vegetation in ponds where the rate of change of water is not sufficient to keep the oxygen concentration below approximately 200 percent at the surface on bright sunny days.

1220.14 Literature.

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Trans. Am. Fish. Soc. 62: 267-274.

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1220.2 BLUE-SAC DISEASE

Hydrocoele embryonalis, Yolk-sac dropsy,
Dropsy of the vitelline sac

1220.21 Symptoms. Affects salmonid sac fry before the yolk is absorbed. The yolk sac enlarges due to accumulation of a watery fluid between the inner and outer vitelline sac membranes. The fluid often has a bluish tinge. Sick fry lie quietly on their sides being unable to support the weight of the enlarged sac. The appearance of a white spot or spots in or on the yolk may be noticed in some cases. Fat droplets may appear within the yolk following enlargement of the outer sac.

1220.22 Cause of the Disease. The following have been listed as possible causes:

1. Injury to the eggs by pressure or mechanical shock during shipment (Schaeperclaus, 1933 and others).
2. Injuries to the brood stock prior to spawning (Schaeperclaus, 1933).
3. Bacterial infection--Diplobacillus and Proteus hydrophilus have been associated with the disease by several workers (Guberlet, Sampson, and Brown, 1931 and others).
4. Mineral content of the water may be a factor (Davis, 1946).
5. Yolk sac dropsy may be a symptom of a goiter condition of the fry (Schereschewsky, 1935).

1220.23 Control Measures. Although the evidence is inconclusive, it appears that the blue-sac condition can result from several widely different factors, therefore no treatment or control measures can be specifically recommended. Treating the eggs with a 1:2,000 solution of acriflavine for 25 minutes is reported to have reduced mortalities in an epidemic believed to have been caused by a diplobacillus (Atkinson, 1932). Care should always be exercised in the handling of brood stock and eggs to avoid mechanical injuries.

1220.24 Literature.

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1314.1 VITAMIN A

1314.11 Functions. Certain red and yellow pigments called carotenoids are intimately related to vitamin A. These pigments form an integral part of the chromatophores (color containing cells) of the skin and also occur in the flesh and some of the internal organs of trout (Tunison, et al., 1942; Goodwin, 1951). It has been shown that one of the carotenoids, astaxanthin, which occurs in considerable quantities in salmonid eggs, is a true fertilization hormone (Goodwin, 1951). Thus, even though carotenoids may not be necessary for normal development of sac fry (Steven, 1949), they may serve a vital role during the fertilization process (Goodwin, 1951).

Vitamin A prevents night blindness of animals (Phillips and Brockway, 1948). It also aids in maintaining resistance to infections, is essential for a healthy condition of epithelial cells, and is a stimulus for new cell growth (Wooster and Blanck, 1950).

Hypervitaminosis A (too much vitamin A in the diet) may cause the following symptoms described by Burrows and co-workers (1952): "Fish thin, almost emaciated; usually one operculum hemorrhagic (blood-shot); one or more gill arch bearing fungus; gill lamellae malformed, some swollen, some curled, some proliferated, some entirely eroded away; livers pale; intestinal fat slight or lacking." These symptoms appeared in blueback salmon on a whale liver diet which has a very high vitamin A content.

1314.12 Deficiency symptoms. Several authors have reported on the results of feeding a 100 percent spleen diet to trout. The results of feeding this vitamin A deficient food are tabulated in Table 1.

Table 1. - Some deficiency symptoms observed among trout on 100 percent spleen diet.

Author	Symptoms
Hess, 1937	opacities in eye lenses at age of six months or older, degenerative changes in the fins, scales, or iris of the eye. Fish fed a mixture of spleen-heart-liver did not develop the nutritional cataract.
McCay and Phillips, 1940	very little vitamin A in their storage depots while trout fed liver had rich supplies. Addition of cod-liver oil was not effective in increasing vitamin A storage.
McLaren, et al., 1947	fish broke at end of six weeks. Symptoms not described but furunculosis broke out and spread through the hatchery.

Since vitamin A may not be the only nutrient out of balance in the 100 percent spleen diet, these symptoms cannot be unconditionally accepted as the result of vitamin A deficiency. However, the symptoms described by Hess and the outbreak of a bacterial disease recorded by McLaren are not entirely out of line in light of what is known about the role of vitamin A in animal nutrition.

Heart tissue is also deficient in vitamin A and symptoms similar to those described by Hess (Table 1.) have been observed at our Emigrant station among rainbow and cutthroat trout and to a much lesser extent among brown trout being fed a 100 percent heart diet. These fish exhibited the cloudy eye lenses, degenerate fins, and scales. In addition, the urinary tubules seemed affected and crystals or concretions formed which blocked the tubules. The result among the rainbow and cutthroat trout was a popeye condition believed to be the same as that described by Davis (1946 and 1953). The body cavity filled with a clear watery fluid, considerable

pigment was present in the kidneys and other internal tissues and in late stages the fish became very dark in color. At Emigrant where water temperature ranges from 46 to 51°F, several months elapse before fish on the 100 percent heart diet begin to die from the described condition. Periodic microscope examinations of the fish revealed a gradual build-up of the symptoms. Symptoms of fatty degeneration of the liver and anemia were also present in a high percentage of the pop-eyed fish. Quite often the spleen would be the only spot of red color observed on opening sick fish. Myxobacteria were observed externally on the gills and internally in the kidneys of sick fish. Bacteriologists from the University at Missoula examined cultures from Emigrant trout and reported mixed myxobacterial infections internally as well as externally. The bacterial infections undoubtedly are factors in the mortalities but a vitamin deficiency resulting in lowered resistance to bacterial infection is probably the primary factor.

The symptoms here described are associated with vitamin A only through speculation with circumstantial evidence. It remains for the trained nutritionists, biochemists, and other experts to complete or correct the picture.

The exact amount of vitamin A necessary in trout diets is not known. Fresh beef liver contains approximately 19,200 I. U. per 100 grams of edible portion (Wooster and Blanck, 1950). Burrows and co-workers (1952) reported hypervitaminosis A symptoms in sockeye salmon fed diets containing over 50,000 spectrophotometric units of vitamin A while the control diet with 27,060 units per 100 grams of diet did not induce the symptoms. A safe course to follow would be to include a vitamin A source in the trout

diet but to avoid foods such as whale and tuna livers which contain too much vitamin A.

1314.13 Dietary sources. Contrary to popular belief, cod-liver oil apparently is not a good source of vitamin A for trout. McCay and Phillips (1940) failed to prevent the formation of nutritional cataracts in fish on a spleen diet by the addition of cod-liver oil and concluded: . . ."the trout can only utilize vitamin A in some special form such as that in beef liver." They further stated that the natural source of this special form of vitamin is unknown. Until more is learned about this phase of vitamin A in nutrition we will have to depend on animal products of proven value. Following is a list of recommended sources of vitamin A:

Beef liver, fresh	19,200 I. U./100 grams of diet (Wooster and Blanck, 1950)
Calf liver, fresh	20,500 (Wooster and Blanck, 1950)
Pork liver	14,200 (Watt, et al., 1950)
Dried whole cows milk	1,400 (Wooster and Blanck, 1950)

Other sources of vitamin A for which analyses are not presently available are: salmon viscera, and dried fish meals.

Paprika, at the 2 percent level in the diet has been used successfully to produce coloration in the skin of trout (Phillips, et al., 1945). Alfalfa leaf meal and tomato pomace are also sources of the carotenoids but trout appear unable to deposit vitamin A from these sources in their livers (Tunison, et al., 1942).

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