Histopathology of "Gas-Bubble" Disease in Salmon Fingerlings[1,2]

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ABSTRACT

Chinook salmon fingerlings were diagnosed as having "gas-bubble" disease. Gross symptoms were similar to those described by other investigators. The histopathology associated with this disease has not been previously reported. The following 10 tissues and organs were examined: gills, liver, spleen, heart, kidney, intestine, stomach, roof of the mouth, skin, and muscle. All exhibited histopathological changes with the exception of the hearts and stomachs, which did not differ from the control fish. The most striking pathological changes occurred in the roof of the mouth of all fish examined.

INTRODUCTION

Gas-bubble disease was first observed in aquaria fish by Gorham (1898). This disease was also noted in aquaria fish by Marsh (1903), which led to a more detailed description of the cause, symptoms, and mechanics of gas-bubble disease (Marsh and Gorham 1904).

Rucker and Hodgeboom (1953) briefly discussed the gas-bubble disease problem in the Puyallup trout hatchery. The gas-bubble disease problem and its probable causes at Cultus Lake trout hatchery were described by Harvey and Smith (1961). Rucker and Tuttle (1948) described a method of removing supersaturated nitrogen from hatchery water supplies. Westgard (1964) noted the problems associated with gas-bubble disease and methods for correcting the problems in the adult salmon spawning channel at McNary Dam. Alikunhi et al. (1951) found that carp fry, cultured under laboratory conditions, were extremely sensitive to gas-bubble disease, possibly due to excessive oxygen production. This disease is usually encountered in hatcheries, spawning channels, aquaria, or at low plunge basin water falls or dams, when water falling over the spillways becomes supersaturated. Special methods of controlling the amount of dissolved gases in the water are required to prevent excessive mortalities in these situations (Harvey and Smith, 1961; Westgard, 1964).

Although rare, some naturally occurring mortalities of fish due to gas-bubble disease have been recorded in black crappies, northern pike, walleyes, and cyprinids from Lake Kaubesa, Wisconsin (Woodbury, 1941); in trout from spring ponds of the River Bosna, Yugoslavia (Rukavina and Varenika, 1956);

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[1]Received for publication September 20, 1966.
[2]This report is based on studies sponsored by the Washington State Department of Fisheries by contract (No. 13565-B) with the Pacific Northwest Laboratory of Battelle Memorial Institute.

J. Fish. Res. Bd. Canada, 24(4), 1967.
Printed in Canada.

in large numbers of seatrout and in small numbers of several other marine fish from upper Galveston Bay, Texas (Renfro, 1963). Mortalities observed in the hatcheries, spawning channels, and aquaria are thought to have been due to supersaturation of nitrogen or oxygen caused by leaks in the hydraulic pipes or by heating cold water for temperature control. The mortalities observed in nature are thought to have been caused by extremely high concentrations of dissolved oxygen in the water produced by abnormally large algae blooms.

The literature pertaining to gas-bubble disease and its effects on fish has been reviewed by Bishai (1960). In a recent book dealing with diseases of lower vertebrates (Reichenbach-Klinke and Elkan, 1965), gas-bubble disease is only mentioned briefly.

Chinook salmon fingerlings, *Oncorhynchus tshawytscha*, that were dying in a public aquarium at Rocky Reach Dam, Washington, were diagnosed as having "gas-bubble" disease, with obvious large and numerous gas bubbles present on their bodies and heads. Most investigators that have encountered gas-bubble disease have described in detail the gross pathology associated with the diseased fish. However, there have been no previous records of the histopathology associated with this disease.

MATERIALS AND METHODS

Several fish, approximately 6 inches long and 1 year old, that exhibited a light, but detectable, case of the disease were fixed for histopathological examination to determine if it affected specific organs or tissues in its early stages. Control fish of similar size and age were sampled from the hatchery at the Pacific Northwest Laboratory, because all fish in the Rocky Reach aquarium were afflicted with the disease. Samples of the following tissues and organs were preserved in methanol for histopathological examination: gills, liver, spleen, heart, kidney, intestine, stomach, roof of the mouth, skin, and muscle. Tissue sections were stained with Mayer's hematoxylin and eosin. Diseased and control tissues were fixed similarly and stained together to eliminate the occurrence of artifacts. The young fingerlings at the Rocky Reach Dam aquarium were originally obtained from L. R. Donaldson's stock at the University of Washington, Seattle.

RESULTS

Gross examination of the fingerlings showed gas bubbles on the body, head, the peritoneal lining of the body cavity, and the oral cavity. The hearts and stomachs of the diseased fish appeared normal upon histological examination, not differing from the controls. However, all other tissues of the diseased fish exhibited definite histopathological changes.

The diseased fish possessed swollen, edematous gill filaments (Fig. 2, compare to Fig. 1), which showed hydropic degeneration of the lining squamous epithelium in many instances. Hemolyzed red blood cells were common in the gill filaments of all diseased fish.

The most striking pathological changes occurred in the roof of the mouth where the squamous epithelium had undergone hydropic degeneration and the dense connective tissue beneath it was edematous, greatly hypertrophied, and possessed karyolytic (faintly stained and lacking basophilism nuclei (Fig. 4 and 5, compare to Fig. 3). In some cases, large vesicles were present between the connective tissue and the epithelial layer, resulting in a separation of the epithelium from the underlying connective tissue (Fig. 5).

The spleens of all infected fish showed an apparent reduction of the white pulp and an abnormal hemolysis of red blood cells.

The diseased animals all possessed kidney tubules with epithelial cells exhibiting vesicular, lightly stained cytoplasm and pyknotic (shrunken and darkly staining) nuclei (Fig. 7, compare to Fig. 6). Many of the epithelial cells were beginning to slough away from the basement membranes. The kidneys had an increased number of hemolyzed red blood cells.

Necrosis was apparent in the intestines of the diseased fish where the epithelial cells possessed vesicular, lightly staining cytoplasm, faded nuclei, and in many cases the distal ends of these cells were ruptured. Beneath the intestinal epithelium, the granule cells of the granulosum possessed vesicular cytoplasm and lightly stained nuclei. In some fish this layer of cells was non-existent.

Skin and muscle samples were taken as a single unit from the lateral line region of the fingerlings. The muscle fibers were atrophied, edematous, and separated from the sarcolemma. The muscle cells exhibited a light staining cytoplasm which had lost its characteristic striated appearance. The muscle nuclei were pyknotic and, in many instances, completely separated from the muscle fibers. In many respects, the epithelial layer of the skin showed pathologic changes similar to those observed in the epithelium of the roof of the mouth.

The liver cells had undergone degenerative changes, with vesicular, hypertrophied cytoplasm and pyknotic nuclei. In some instances, the liver cells were so enlarged that the normal architecture of the organ was obscured and the red blood cells normally present in the liver sinusoids were absent (Fig. 9, compare to Fig. 8).

DISCUSSION

The gross pathology observed in the fingerling salmon from Rocky Reach Dam has been noted by other investigators. However, some additional symptoms of the disease have been reported by other workers, including swollen air bladders and gas bubbles in the gill arch mucosa, the intestine, the bulbus arteriosus of the heart, the eyes, and between the membranes connecting the fin rays. Microscopic examination of the gills has shown the branchial arterioles completely filled with gas bubbles (Renfro, 1963).

One puzzling aspect of the malady is the apparent recovery ability of fish in advanced stages of the disease (Rukavina and Varenika, 1956; Fujihara, personal communication, 1965). This is extremely perplexing upon observing the systemic destruction of cells and tissues by this disease in its early stages.

However, this apparent ability to recover is probably related to the supersaturation level of the water, the water temperature, the duration of exposure to the supersaturated water, and the size and species of the fish involved. Small fish appear to be highly susceptible to gas-bubble disease (Alikunhi et al., 1951; Harvey and Cooper, 1962). Westgard (1964) has discussed the physical and biological problems associated with gas-bubble disease and some possible solutions. However, the disease's etiology, including the stage of the disease at which desirable species of fish will not recover, has not been thoroughly studied.

The histopathological changes observed in the salmon fingerlings due to gas-bubble disease are similar to the pathological changes that have been observed in other fish due to chemical toxicity (Christie and Battle, 1963; Andrews et al., 1966), which include congested and edematous gill filaments, edematous muscle fibers, and degenerative liver changes with variations in staining intensity and distortion of the liver cells. The striking pathological changes that resulted in the "swollen" roof of the mouth appear to be unique to gas-bubble disease and we are using this as the diagnostic characteristic for detecting gas-bubble disease in its early stages. Death in fish suffering from gas-bubble disease is apparently caused by necrosis of vital organs and tissues, probably due to anoxia resulting from capillary occlusion by gas emboli.

ACKNOWLEDGMENTS

The cooperative efforts contributed by Messrs D. E. Schoeneman, T. K. Meekin, A. C. Moser, and G. Tangen of the Washington State Department of Fisheries are gratefully acknowledged.

We are grateful to Dr William J. Clarke, Manager of the Pathology Section of the Pacific Northwest Laboratory, for his assistance and interpretation of many of the histopathological results and critical review of the manuscript. Collection of the samples was made by Mr P. A. Olson of the Pacific Northwest Laboratory. Technical assistance of Mr J. D. Maulsby is gratefully acknowledged.

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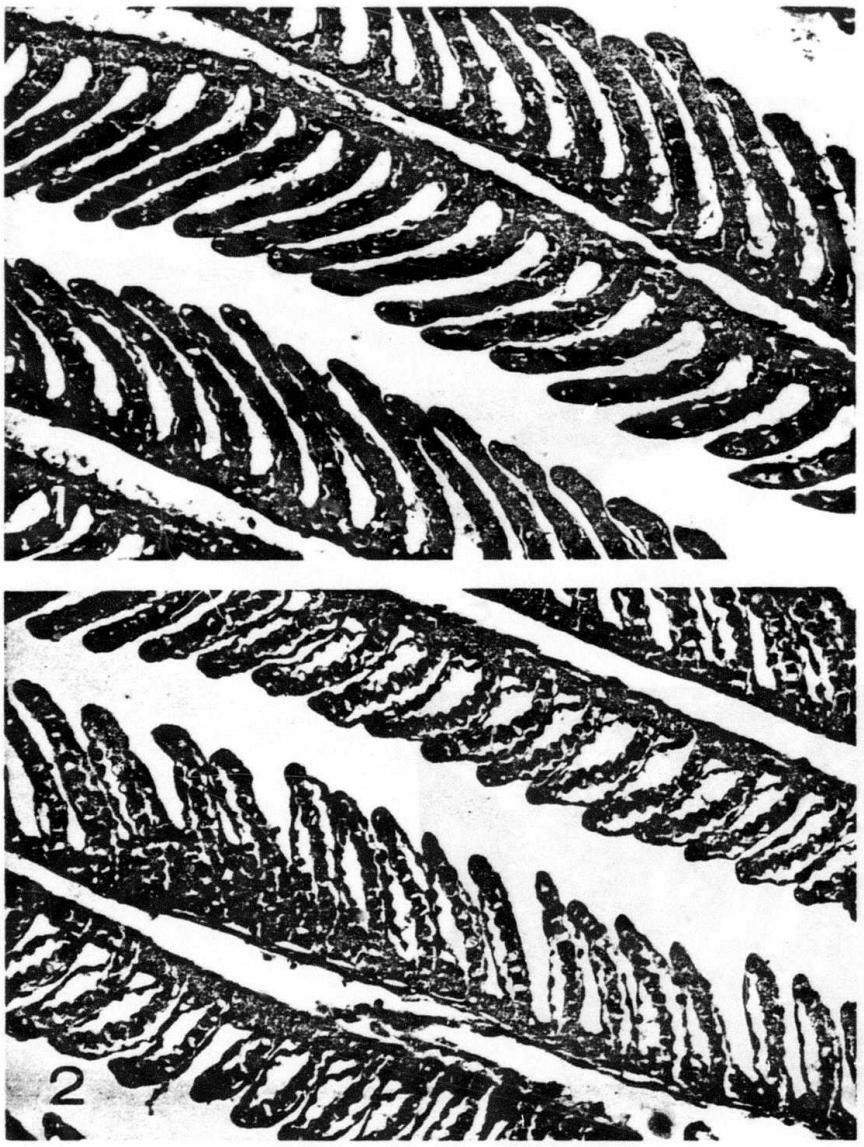

FIG 1. Normal gill of a salmon fingerling. 155×.

FIG. 2. Gill of salmon fingerling afflicted with gas-bubble disease. Note the swollen, edematous appearance of the individual filaments. 155×.

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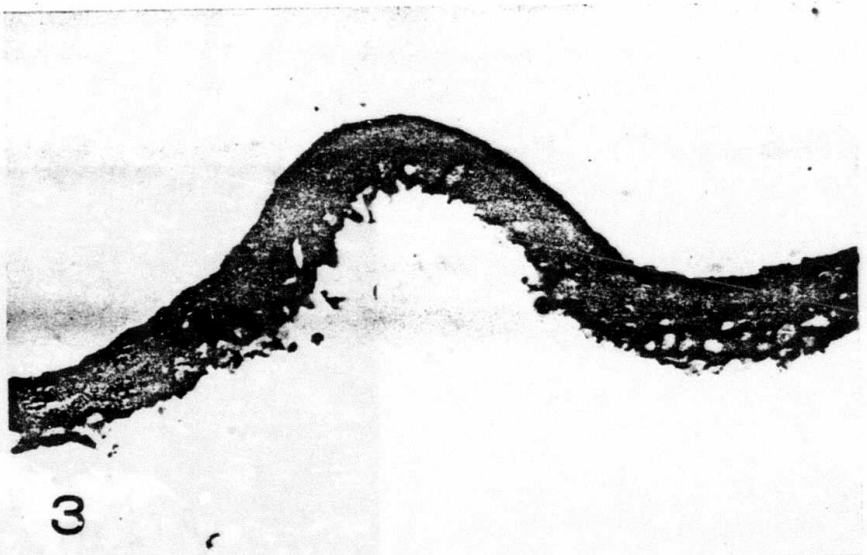

FIG. 3. Normal tissue from the roof of the mouth of a salmon fingerling. 155×.

FIG. 4. Tissue from the roof of the mouth of salmon fingerling possessing gas-bubble disease. Note the swollen appearance of both the epithelial and connective tissue layers. 155×.

FIG. 5. Tissue from the roof of the mouth of salmon fingerling possessing gas-bubble disease. Note the swollen appearance of the tissue and the vesicles between the epithelium and connective tissue. 155×.

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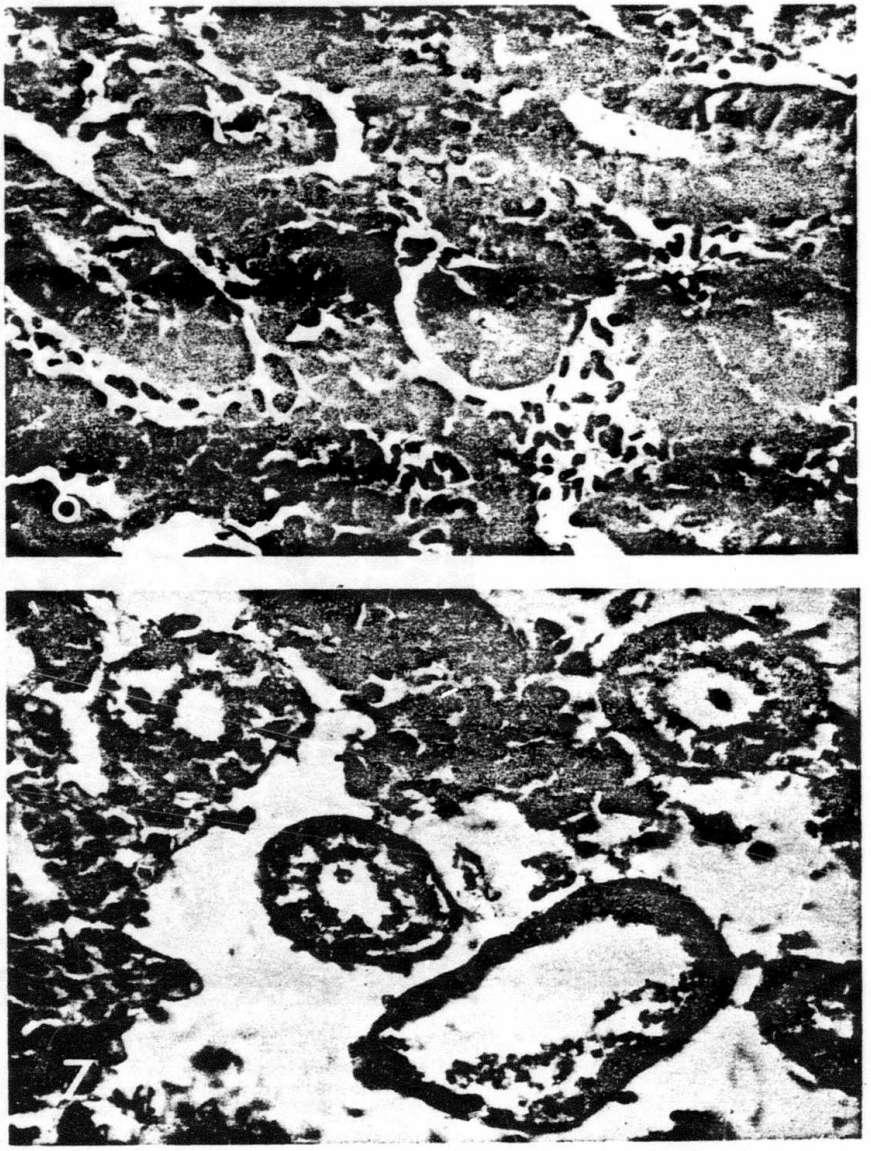
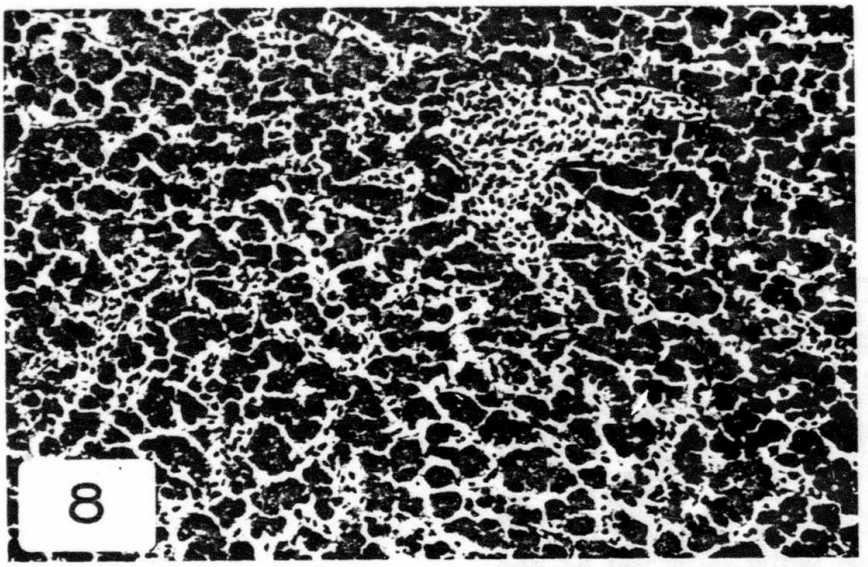


FIG. 6. Normal kidney from salmon fingerling. 390×.

FIG. 7. Kidney of salmon fingerling exhibiting gas-bubble disease. Note the pyknotic nuclei and lighty stained cytoplasm of the epithelial cells. 390×.



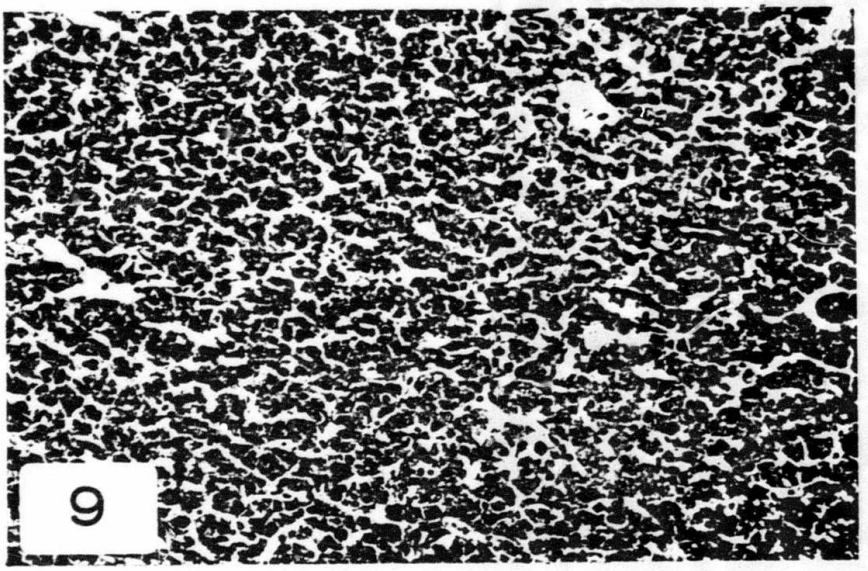


FIG. 8. Normal liver from a salmon fingerling. 155×.

FIG. 9. Liver from a salmon fingerling afflicted with gas-bubble disease. Note the lighty stained cytoplasm of the liver cells, the obscured normal architecture of the organ, and the almost complete lack of red blood cells. 155×.

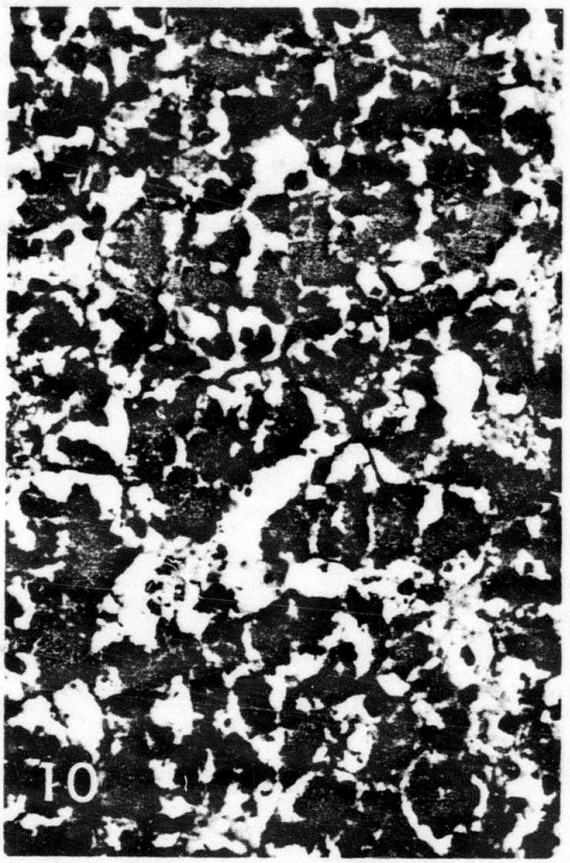


FIG. 10. A fingerling salmon afflicted with gas-bubble disease whose liver cells exhibit marked degenerative changes. Note the irregular staining cytoplasm and pyknotic nuclei. 390×.

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