A SUDDEN MORTALITY OF FISHES ACCOMPANYING A SUPERSATURATION OF OXYGEN IN LAKE WAUBESA, WISCONSIN

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ABSTRACT

A heavy loss of fish occurred during April 1940, in the south end of Lake Waubesa, Wisconsin, and in the Yahara River below the lake. The fish affected were mainly black crappies, although bluegills, northern pike, yellow pikeperch, common suckers, and carp, were killed to a lesser extent. A heavy algal bloom composed principally of *Chlamydomonas* was concentrated in the area of fish loss and was accompanied by an extremely high (30-32 p.p.m.) oxygen content in the surface water.

Characteristic lesions were present in the fish and consisted primarily of gas emboli in the gill capillaries and gas bubbles in the subcutaneous tissues. Death of the fish was attributed to the blocking of the circulation through the gills by the gas bubbles with consequent respiratory failure. A suggestion is made that oxygen was the gas forming the bubbles.

INTRODUCTION

During the latter part of April 1940, a heavy loss of fish occurred in Lake Waubesa, Wisconsin. No estimate was made of the fish that died but at least several thousand were observed. Conversation with people familiar with the lake indicated that minor losses occurred every spring but never reached as serious proportions as in 1940. Since the loss was from a rather unusual cause it was felt that a description would be of value.

FISH AFFECTED

Black crappies (*Pomoxis nigro-maculatus*) were the predominate fish affected. However, other fish such as bluegills (*Lepomis macrochirus*), northern pike (*Esox lucius*), yellow pikeperch (*Stizostedion v. vitreum*), suckers (*Catostomus commersonnii*), and even an occasional carp (*Cyprinus carpio*), were found dead or dying. Nearly all of the dead crappies were of the 1936 year class, that is, fish hatched in 1936. Very few crappies of other ages were noted. In general all fish found were large mature adults. No small or young fish of any species were observed.

SYMPTOMS

The distressed fish were observed only in the last stages of the disease shortly before death so that the early symptoms could not be recorded. In the final stages the fish showed partial to complete loss of

equilibrium, and passed gradually into a coma or stupor which grew deeper until death.

General Appearance and Pathology

The general appearance of the fish was normal except for the presence of small gas bubbles, visible to the naked eye, under the scales, skin, and between the rays of the fins. This was the most prominent pathological manifestation. The bubbles gave the fins, and occasionally the skin, the appearance of having a large number of blisters and were

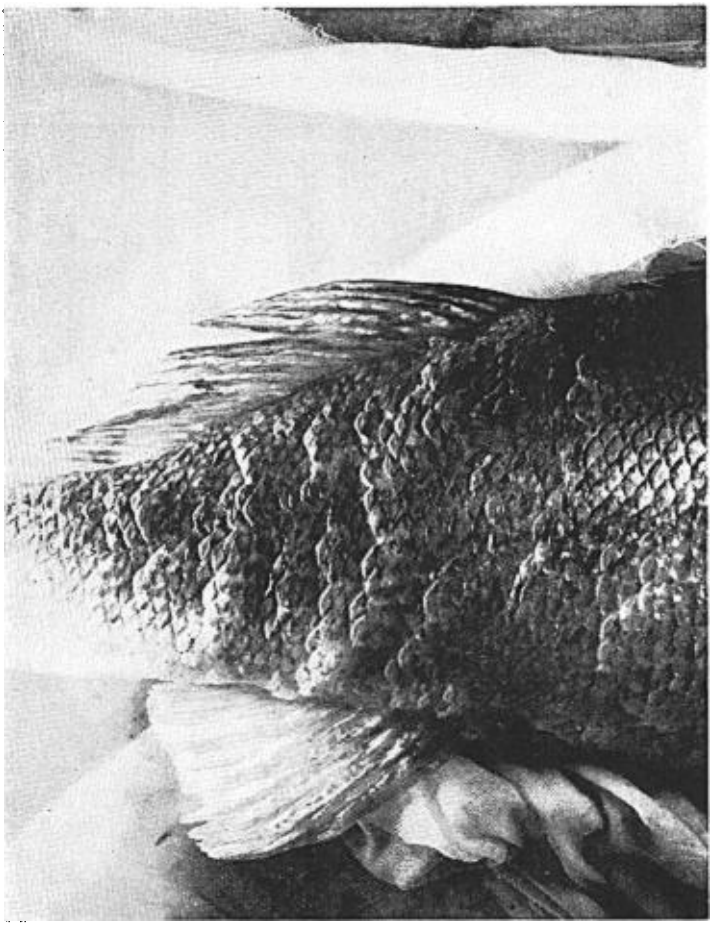

Figure 1.—Portion of yellow pikeperch showing gas bubbles in scale pockets and between rays of dorsal and anal fins

present on nearly all the fish observed. The condition is illustrated in Figure 1 which shows the general appearance of the skin and anal fin of a yellow pikeperch found dead in the lake. The gas bubbles may be seen between the fin rays very distinctly, especially in the anal fin. Certain of the scales have been raised by the formation of bubbles in the scale pockets.

The gills also were damaged severely, moreso than other parts of the body. The primary cause of the damage was the presence of gas emboli in the capillaries of the gill filaments. Most of the filaments examined contained gas emboli of such a size that they completely blocked the flow of blood through the capillaries. Figure 2 shows a gill filament in which the gas bubbles can be seen blocking the capillary. The obstruction of blood circulation in the capillaries resulted eventually in the death of the filament with consequent degenerative

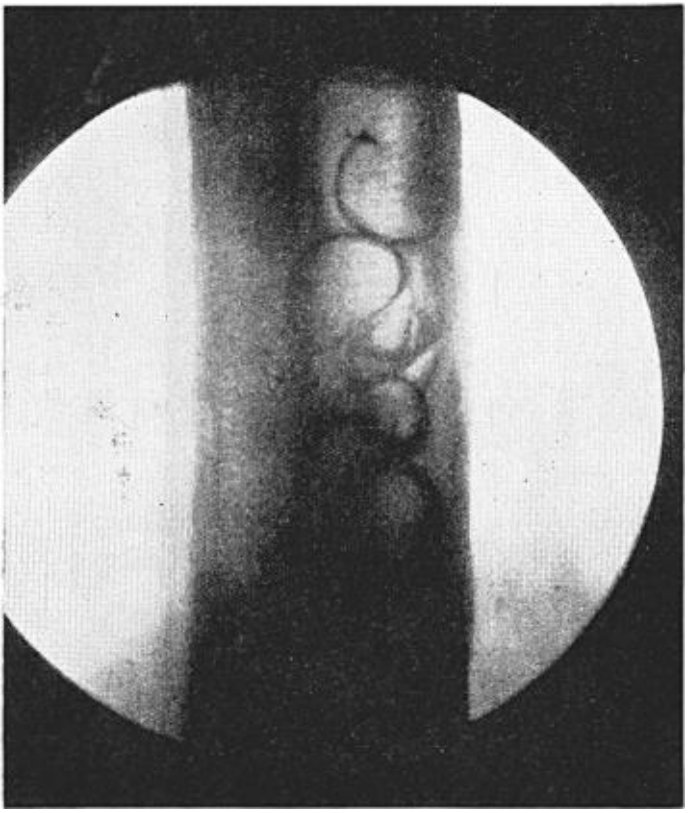

Figure 2.—Gill filament of yellow pikeperch showing gas bubbles in capillary

changes. In many of the fish examined large areas of the gills were necrotic and overgrown with fungus, in others the gill tissue had sloughed away leaving only the supporting strip of cartilage. Blood clots were seen between the lamellae of many of the gill filaments. Occasionally an eye would be damaged by the formation of bubbles within the eyeball.

Water Conditions

At the time of examination the water along the entire south (windward) shore of the lake had a greenish appearance due to an algal bloom, and a thick layer of algae was present in a band about 10 feet wide along the shore line. Upon identification the major part of the algal layer was found to consist almost entirely of *Chlamydomonas*, with small amounts of Protozoa, diatoms, desmids, and filamentous algae constituting the remainder.

Analyses of the lake water revealed an extremely high dissolved-oxygen content and an unbalanced condition of the carbonate-bicarbonate content. The results of the analyses are presented in Table 1.

Table 1.—Results of analyses of oxygen and carbonate-bicarbonate content of Lake Waubesa water at time of the heavy fish loss

Station	Depth (feet)	Dissolved oxygen (p.p.m.)	Percentage saturation	Bicarbonates (p.p.m.)	Carbonates (p.p.m.)
20 feet offshore....	1	30.0	306	57.0	88.0
150 feet offshore..	1	32.1	327	.....	.....
150 feet offshore..	12	16.8	171	.....	.....

The large quantities of *Chlamydomonas* in the water undoubtedly account for the extreme unbalance of the dissolved gases. The weather preceding the outbreak of the epizootic had been clear and bright thus furnishing sufficient light for photosynthesis to proceed at a rapid rate. The high preponderance of carbonates in the water was probably due to the breaking down of the bicarbonates by the algae once the free carbon dioxide was exhausted. The supersaturation of oxygen was also present in the river below Lake Waubesa. Fish losses occurred in the river but not of the magnitude of those in Lake Waubesa.

Diagnosis and Cause of Death

Death of the fish apparently was caused by the formation of gas emboli in the gill capillaries and possibly in other critical areas of the body such as the heart chambers. The presence of gas bubbles in the gill capillaries of such a size as to prevent circulation would lead to necrosis of the gills and eventual death due to respiratory failure.

The process through which the gas emboli appeared in the capillaries

and the tissues can be only conjectured. Fish living for any length of time in water supersaturated with oxygen undoubtedly would soon have their blood and tissues likewise supersaturated with oxygen. A number of factors might have caused the release of oxygen bubbles into the blood stream or tissues. A slight rise in the body temperature of the fish due to increased activity might have been the direct agent since oxygen solubilities decrease with increasing temperatures. Again the water may have lost oxygen to the atmosphere due to wind agitation following a calm and a rise in temperature, thus rendering the fish blood and tissues supersaturated with respect to the water. Both of the above hypotheses predicate that the change occurred so rapidly that the oxygen escaped into the blood or tissues of the fish rather than being slowly diffused back through the gills into the water.

Examination of the literature indicates that the death of fish because of oxygen emboli in the capillaries is not an unique phenomenon. Schaperclaus (1933, p. 252) recorded a disease of young trout (Gasblasenkrankheit) in which gas bubbles appear under the skin especially on the head and gills. Plehn (1924) attributed the "gas bubble disease" to the supersaturation of the water by oxygen due to a rich plant growth.

Haempel (1928) discussed in some detail the effects of high oxygen concentrations on fish especially under conditions of artificial supersaturation with oxygen. He indicated that salmonid fishes exposed to high oxygen concentrations develop at first a marked uneasiness and dyspnoea followed by a loss of equilibrium and finally a stupor or oxygen narcosis. With further continuous supersaturation of the waters, the fish died. He recorded such pathological symptoms as opaqueness of the cornea, exophthalmia, and release of gas bubbles from the capillaries. Haempel's paper indicated that 2 or 3-year-old goldfish and the Zander (*Lucioperca sandra*, the European species of yellow pikeperch) do not succumb to the above conditions as easily as do the young fish, but do show symptoms of irritability.

Marsh and Gorham (1905) made a detailed study of a gas disease in fishes in which they described symptoms, pathology and etiology of this condition. The symptoms and pathology are in general the same as those described in this study and by Haempel and Schaperclaus. However, the disease as described by Marsh and Gorham accompanied an excess of nitrogen in the water and analyses of the gas in the skin blebs and heart chamber indicated that nitrogen was the gas responsible. Those authors discussed the possible role of oxygen in producing the gas disease but had no instances where oxygen was responsible.

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