

FLUORIDE INTOXICATION IN FISH: A REVIEW

Authors: SIGLER, W. F., and NEUHOLD, JOHN M.

Source: Journal of Wildlife Diseases, 8(3) : 252-254

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-8.3.252>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

FLUORIDE INTOXICATION IN FISH: A REVIEW

W. F. SIGLER and JOHN M. NEUHOLD, Department of Wildlife Science,
Utah State University, Logan, Utah, U.S.A.

Abstract: A wide range of environmental and genetic factors cause fish to respond differently to given levels of fluorides, but they do display characteristic fluoride intoxication signs. Some of the variation can also be explained by postulating a chloride-fluoride excretion mechanism over the epithelial tissues. Such a mechanism would explain variations in toxicity correlated with different chloride concentrations and the survival of natural populations of fish at fluoride concentrations which are lethal under laboratory conditions.

FLUORIDE IN THE AQUATIC ENVIRONMENT

Fluorides are widely distributed in the rivers, lakes, and seas of the world. The U.S. Geological Survey¹⁰ reports that fluorides are common in the waters of the United States, especially in the west. Concentrations of 0.1 ppm are common and concentrations exceeding 1.0 ppm are not rare. Water samples from Walker and Pyramid Lakes in Nevada contain up to 13 ppm fluoride. The Madison and Firehole Rivers in Yellowstone National Park have fluoride concentrations ranging from 12-14 ppm. Kobayashi⁵ reports 1.5 - 5.5 ppm of fluorides in wells in Japan. The natural thermal waters of New Zealand (pH 5-9) contain from 1-12 ppm of fluorides⁷.

Most of the fluorides occur naturally. They are leached from fluoride, cryolite, apatite, and sedimentary phosphate rocks by precipitation and ground water. Pollution, both aerial and hydric also contribute fluoride to the aquatic environment.

Aquatic organisms would be expected to contain fluoride concentrations proportional to those in their environment. Neuhold and Sigler⁶ reported mean concentration up to 1600 ppm in the bones of brown trout taken from the Madison River system. Lee and Nilson⁸ recorded high concentrations of fluorides in the bones of canned salmon and mackerel. Similarly, Fisher¹ noted high concentrations of fluorides in fish meals used in the manufacture of prepared feeds.

FLUORIDE INTOXICATION

Signs of Fluorosis

Rainbow trout, carp and goldfish become apathetic and evidenced anorexia with the introduction of sodium fluoride to their medium. Goldfish suffering from anorexia lose weight⁸, go through a period of violent movement which degrades into aimless wandering, and finally lose their equilibrium. The loss in equilibrium is accompanied by tetany and ends in death. Mucus secretion in all the fish tested increases with introduced fluoride. The increased mucus secretion is accompanied by proliferation of mucus-producing cells in the respiratory and integumentary epithelium⁸.

The embryos of rainbow trout display much the same signs as adults when intoxicated with fluoride. Violent movement within the confines of the egg often cause the vitelline membrane to rupture prematurely. This phenomenon frequently is followed by death and an immediate coagulation of the yolk protein. Embryos that survive a fluoride-induced premature emergence have a high incidence of deformed spines⁸.

Blood Changes

Changes occur in the blood of rainbow trout suffering from fluoride intoxication¹. The level of total serum protein appears to drop in both trout and carp during intoxication^{1,2}. The gamma and beta globulins specifically appear to change. Both serum alkaline phosphatase

activity and plasma magnesium levels increase, while calcium decreases with increasing fluoride concentrations.

Differentiation from Thermal Death

In thermal death, fish first show marked apathy then loss of orientation and equilibrium and finally gasping for breath, reduction of swimming ability, darkening of color, and then death. Occasionally there is limited muscular contraction just before death. After death the fish is limp. The difference in signs between fluorosis and thermal stress is that in the former, fish activity is much more intense in the early stages; in the final stage fluoride-induced death invariably results in some degree of tetany, generally quite intense and the skin does not discolor.

TOXIC LEVELS

The effects of fluorides in the environment, as reported by literature, all lead to the conclusion that fluorides above certain levels have a profound toxic effect on the physiology of animals.

The response of fish to moderate fluoride concentrations (1.5 to 5.0 ppm) is related to environmental acclimatization and is species dependent. It is difficult to assign specific values as toxic levels because so many factors in the environment, including the physiological state of the fish, the species, even the race or strain, govern the response of fish to intoxication. Various chloride concentrations in the medium affect the reactions of rainbow trout to fluorides⁹. Minnows subjected to simultaneously raised concentrations of chlorides and fluorides succumb to lower levels of fluorides than do fish subjected to only fluoride⁶. The amount of calcium in the medium also affects fish response. Higher than normal concentrations of calcium in the medium or the food tend to enhance the resistance of fish to fluorides⁶.

The time necessary for rainbow trout eggs to hatch decreases with an increase in the concentration of fluoride in the medium. It is concluded that larger fish, subjected to a given level of fluoride, tend to succumb last; that is, the larger the fish the more resistant they are to a given level of fluorides. The size of the fish has an effect on the length of the experiment only. No effect on the LD₅₀* or the sensitivity is apparent. The LD₅₀ for rainbow trout, 10-20 cm in total length, in a medium low in both calcium and magnesium and at a temperature of 13C, is between 2.7 and 4.7 ppm. The LD₅₀ for small carp falls between 75 and 91 ppm of fluoride when the same low calcium and magnesium concentrations are present⁶.

Increasing the temperature of the medium increases the sensitivity of rainbow trout to fluoride intoxication. This relationship appears to be a function of the metabolic rate, which increases with temperature⁶. The relationship between temperature and metabolism is one of size and sensitivity of fish to fluorides. Small rainbow trout and small carp appear to be less resistant to fluoride intoxication than large ones.

Fish populations vary widely with respect to their ability to live in specific concentrations of fluorides. Healthy, growing populations of trout exist in the Firehole River in Yellowstone National Park where fluoride concentrations reach 14 ppm. The same is true of Pyramid and Walker Lakes in Nevada where the concentrations reach 13 ppm. Yet, rainbow trout that have been reared in low concentrations of fluoride display LD₅₀ of approximately 3 ppm.

FLUORIDE UPTAKE

Fluoride uptake occurs in both soft tissues and bone in carp, goldfish and rainbow trout. The uptake in muscle tissue is highly variable and can be ascertained only between extremes. Osseous tissues are particularly good indicators of

*The LD₅₀ is the dose lethal to 50 percent of the experimental animals. The dosage schedule is predetermined. Although the time is not predetermined, experiments are generally terminated between 48 and 240 hours.

uptake of fluoride. Fish collected from the Madison River system showed a positive correlation between bone fluoride concentration and size of fish.

Goldfish subjected to chronic levels for 90 days accumulated fluoride at a rate similar to those for carp under acute levels^{3,9}. The rate of uptake from the medium and its incorporation into the bone is by an apparent second order mode, suggesting active transport⁶. Fluoride concentration in the tissue is directly correlated with the amount of fluoride in the medium and with the duration of exposure. Fluorides accumulate more readily in osseous tissues⁷. When goldfish were subjected to low concentrations of fluorides (0.34-2.95 ppm) in soft water for 90 days there was an increase in bone fluoride concentration that correlated with both the median fluoride concentration and the duration of exposure⁸.

TISSUE CHANGES

In one experiment, the epithelium of the gill filaments of rainbow trout displayed an increase in population density of mucus-producing cells from 0.31 at

0.0 ppm fluoride to 0.52 at 25.0 ppm fluoride. The epithelial tissue in the head region of rainbow trout fry subjected to two ranges of fluoride concentrations (0 to 25 ppm and 250 to 335 ppm) also indicated an increase in mucus cells. The tissue upon which these population density determinations were made was integumentary epithelial tissue located between the eyes⁵.

Aside from the accumulation of fluorides in the tissues, other changes occur as well. The ultimobranchial gland (parathyroid function) hypertrophied in trout subjected to high levels of fluoride^{1,2}. In the gill lamellae of goldfish, an edematous condition of the epithelium (described as a focal, non-specific, cytoplasmic enlargement, with the nuclei of the cells remaining unchanged) also occurs during fluoride intoxication³. If the mucus cells in the epithelium are assumed to act as a fluoride secretion mechanism, and if their proliferation is assumed to be a function of the fluoride concentration in the blood, the tendency would be toward an increased concentration of blood fluorides with increased concentration of medium fluoride.

LITERATURE CITED

1. ALGER, R. H. 1960. The effects of fluoride ion on some blood constituents of rainbow trout. Utah State Univ. M.S. Thesis. p. 55.
2. ANGELOVIC, J. W., W. F. SIGLER, and J. M. NEUHOLD. 1961. Temperature and fluorosis in rainbow trout. J. Wat. Pollut. Control Fed. (April, 1961). p. 371-381.
3. deROOS, C. C. 1958. The effects of sodium fluoride on the weight gain and gills of the common goldfish. Utah State Univ. M.S. Thesis. p. 40.
4. FISHER, H. J. 1951. Common toxicologic findings in Connecticut. J. Amer. vet. med. Assoc. 118: 309-311.
5. KOBAYASHI, D. 1951. The fluoride content of underground water of Shippo Village Aichi Prefecture (Japan). Kagaku (Science), 21: 367-368.
6. LEE, C. F., and H. W. NILSON. 1939. Study of the metabolism of naturally occurring fluorine in canned salmon and mackerel. U.S. Bureau of Fish., Invest. Rpt. 44. p. 15.
7. MAHON, W. A. J. 1964. Fluorine in the natural thermal waters of New Zealand. New Zealand Jour. of Science 7: 3-28.
8. NEUHOLD, J. M., and W. F. SIGLER. 1960. Effects of sodium fluoride on carp and rainbow trout. Trans. Am. Fish. Soc. 89: 358-370.
9. NEUHOLD, J. M., and W. F. SIGLER. 1962. Chlorides affect the toxicity of fluorides in rainbow trout. Science 135: 732-733.
10. U.S. GEOLOGICAL SURVEY. 1957. Quality of surface waters of the United States. 1952. U.S. Geol. Surv. Water-Supply Paper 1253. p. 353.

Received for publication November 1, 1971