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Exophthalmia in Trout-Fry.

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(Introduced by Frederick L. Hisaw.)

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H. B. Ward and J. Mueller[1] reported exophthalmia in the trout-fry. Their examination showed the presence of larvae of an unidentified trematode in the muscles of the eyes, at the base of the fins and in some cases in the optic nerves of the protruding eyes. Exophthalmia is prevalent amongst the fish at the State Fish Hatchery at Madison, Wisconsin. Those affected are found to be free from parasites and only those that have been injured are infected with fungus. The condition does not appear until the yolk sac has been absorbed and the fry begin to eat. Large numbers die at this stage of development, and a high percentage of those that die show exophthalmia. The number that die, and the number showing exophthalmia as well, decreases as they grow and summer approaches, only to increase again the following winter and spring when they are a year old. Histological and experimental study show the exophthalmia to be the same in the adult trout and in the fry.

Numerous papers have appeared in the literature describing thyroid diseases of the trout and other fishes, both wild and domesticated. Bonnet[2] first described a tumor of the region of the gill junction, and Scott[3] first described such a tumor as a carcinoma of the thyroid. M. Plehn,[4] L. Pick,[5] Jaboulay,[6] Gaylord and Marsh,[7] Marine and Lenhart,[8] Müller[9] and Johnstone[10] have described similar conditions. In all cases reported, hyperplasia of the thyroid has been indicated externally by a tumor or by areas of hyperaemia. The

[1] Ward, Henry B., and Mueller, Justus F., *Arch. Schiffs-u. Tropen-Hyg.*, **1926**, xxx, 602. (*Biol. Abstr.*, 1927, vii and viii, 1126.)

[2] Bonnet, R., *Bayerische Eisch. Ztg.*, Munchen, 1883, vi, 79.

[3] Scott, *Tr. and Proc. N. Zealand Inst.*, 1891, xxiv, 201.

[4] Plehn, M., *Allg. Fisch. Ztg.*, 1902, vii, 117.

[5] Pick, L., *Berl. Klin. Woch.*, 1905, xlvi-xlix, 1435.

[6] Jaboulay, *J. Med. et Chir. prat.*, 1908, lxxix, 235.

[7] Gaylord, H. R., and Marsh, Millard C., *Bull. U. S. Bur. Fisheries*, **1912**, **xxxii**, 367.

[8] Marine, David, and Lenhart, C. H., *J. Exp. Med.*, 1910, xii, 311.

[9] Müller, F. W., *Virchow's Arch. Path. Anat. u. Physiol.*, 1926, cclx, 405.

[10] Johnstone, J., *Proc. and Tr. Liverpool Biol. Soc.*, 1926, xl, 59.

fry examined by the writers showed no external indication of enlargement of the thyroid.

Exophthalmia was either unilateral of bilateral, the extent of protrusion of the eyes varying from an indication to complete protrusion. When exophthalmia is unilateral, the fry show the phototropic reactions described by Main,[11] in that they tilt along their long axis with the injured eye upward and turn toward the injured eye. If the eyes are equally affected, the tilting reaction is compensated, they swim in a normal manner and turn toward either side with equal frequency. In extreme cases of exophthalmia they are also emaciated and muscularly weak and may be found in 3 stages of the diseased condition: first, indications of exophthalmia, muscular weakness and emaciation; second, more pronounced exophthalmia, and more pronounced muscular weakness in that they remain motionless on the bottom and swim but a short distance when disturbed; third, loss of ability to maintain equilibrium in that they come to rest on their side and respond to stimuli with slow and weak lashings of the tail. They may live in the last condition for some time, the only indications of life being the respiratory movements.

Microscopic sections of the head region show a hyperplastic condition of the thyroid, the alveoli being much enlarged and plicated. The epithelium is definitely columnar and the alveoli lack stainable colloid material. The fry with extreme exophthalmia show a much greater degree of thyroid hyperplasia than those with only an indication of exophthalmus, which indicates a close relation between the 2 conditions.

In order to determine the effect of iodine on the young trout-fry, the fry at the hatchery were divided into 2 groups, one numbering 25,000 and the other 7,000. The larger group received sodium iodide in the water in the proportion of 100 parts to a billion parts of water for 10 days and then 30 parts per billion for the following 8 weeks. The iodine treatment was begun about the time that they began to feed, because, as stated above, the exophthalmic condition does not appear before they have reached this stage of development. The second and smaller group received no iodine in the water but were kept in pools fed by the same spring stratum.

Exophthalmia did not appear in the iodine treated group. The treated fry grew more rapidly than the control fry and at the age of 6 months were found to be 4 times as large as the non-treated fry of the same age. Only occasional fry died, whereas in the 20 years previously practically all the fry died which were hatched at this hatchery unless they were moved to other waters. The thyroid

[11] **Main, R. J., *Z. f. vergl. Physiol.*, 1928, vii, 611.**

from the fry of the treated group was found to be small, and limited to the region immediately about the ventral aorta. The alveoli were small and distended with stainable colloid material, and the epithelium was of a very low cubical type.

The alveoli of the fry not receiving iodine and seemingly normal were slightly larger than the alveoli of the fry having received iodine. Likewise, the alveoli were found to be only incompletely filled with stainable colloid material, the walls smooth and free from plications and the epithelium of a cubical type. Gaylord and Marsh describe the thyroids of the trout from Michigan and Wisconsin as being slightly hyperplastic. The fry not receiving iodine, and that died, showed the same microscopic conditions as the exophthalmic fry, indicating that the hyperplastic thyroid was more common than the normal thyroid in the control group.

From the present study it is concluded that iodine prevents the appearance of exophthalmia in trout-fry. The minimum iodine required to prevent the appearance of exophthalmia has not been determined. The iodine as total iodine from the water from the spring stratum was found to be 8.9 parts per billion parts of water. Since very few died that received iodine, it is assumed that the fry not receiving iodine, and that died without exhibiting exophthalmia, died from the same cause for they have been found to lack only the one characteristic. Iodine causes a decrease and a disappearance of exophthalmus, involution of the thyroid gland, the appearance of colloid material in the alveoli as well as an increase in physical vigor. Since the characteristics of the disease do not recur as long as they receive iodine, indications are that it is due to a lack of iodine.

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Effect of Foreign Protein and of Insulin Administered by Mouth After Ox-Gall.

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While in the midst of a series of agglutination and precipitin tests on rabbits and guinea pigs the writers came across several news