Ultra-violet light affords new opportunities for the control of fish diseases. It is unique in providing a means for the simultaneous treatment of both the fish and the water in which it swims. Since no experiments have been reported which deal with the ultra-violet radiation of normal fish we have summarized in the accompanying table some of our data. Before being radiated the trout were placed in a wire cage twelve inches square. This cage contained a screen bottom and top so that the fish could be confined in a water stratum at a definite distance from the surface. Ten trout about 2 to 3 inches in length were used in each experiment. During the radiation the trout were kept in their usual habitat of flowing spring water at a temperature of 10° C.

TABLE I

THE LETHAL DOSE OF ULTRA-VIOLET RADIATION FOR BROOK TROUT

Depth of water in inches	Distance of lamp from water in inches	Time of exposure in minutes	Time between exposures in hours	No. of exposures	No. trout killed	Results
1	6	5	24	3	4	Two died after third exposure. Two more died next day.
1	12	5	24	4	1	One died fifth day—others appeared grayish as if burned.
1	6	3	24	5	10	Three dead after fourth exposure. Four more dead after fifth exposure. Rest died next day.
1	12	3	24	16	1	No deaths until tenth day. Had not appeared normal or eaten well for a few days before.
1	12	3	18	5	0	No deaths. Held for 6 days after last exposure.
3–4	12	1	24	10	0	Apparently no detrimental effect.
3–4	6	90		1	3	Very grayish in appearance when removed—two died next day, one some time later. All appeared burned.

Our table shows that trout are killed by one long period of radiation or by a series of short periods at daily intervals. It also shows that trout can withstand a certain amount of radiation without injury. This affords a zone for further experiments in attempts to destroy the parasites without injuring the fish. No reliable comparison can be made between the sensitiveness of fish and the higher animals to ultra-violet radiation since the penetration of water by ultra-violet light seems unsettled. If water is readily penetrable, trout are less sensitive to radiation than man; if the reverse is true they are probably more sensitive.

The lamp used in these experiments was the Uviarc poultry lamp which was furnished us through the courtesy of the General Electric Company.

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ON THE OXIDATIVE NATURE OF THE NERVE IMPULSE[1]

A YEAR or so ago I succeeded in demonstrating[2] that although nerve conduction may go on in nitrogen for some time, for which chemically bound oxidative reserves were presumably used, no excess carbon dioxide was given off as is the case in aerobic conduction. The tentative view was adopted that the initial phase of conduction, manifested by the action potential as usually recorded, was conditioned by if not caused by a union of some substance in nerve with oxygen, rather than a complete oxidation with the accompanying production of carbon dioxide. I attempted at that time to determine manometrically whether this oxygen, required for stimulation, had first to be activated or whether molecular oxygen sufficed. Owing to inadequacies in the technique, the results were only suggestive and were not published. This work was made the starting-point of a program of research aimed at a clearer elucidation of the physical chemical processes responsible for the propagation of the impulse in nerve; the purpose of the present notice is to report a few of the results thus far obtained.

For this work I have turned to the theory of Warburg as being the most promising experimentally. Warburg believes to have shown that the respiratory enzyme is an iron-containing, hemin-like substance which can be poisoned by cyanides, hydrogen sulphide and carbon monoxide. Neither Warburg nor his collaborators have ever worked on nerve, however. Hence it became our first task to see whether nerve behaves towards these poisons as does Warburg's yeast.

To be very brief, it appears that sodium cyanide may inhibit nerve respiration very completely. In dilute solutions, *e.g.*, N/1000 NaCN, the inhibition is

[1] From a paper presented at the Marine Biological Laboratory, Woods Hole, Massachusetts, on July 25, 1930, and summarized in *The Collecting Net*, 5: 145, 1930.

[2] F. O. Schmitt, *Biochem. Zeitschr.*, 213: 443, 1929.

complete only for an hour or so, thereafter wearing away to a certain residual amount. In strong solutions, *e.g.*, N/10 NaCN, the inhibition is practically constant from the first and amounts to 80 to 95 per cent. The explanation of the escape from inhibition is to be found, perhaps, in the expulsion of HCN from the inner fibers by the lactic acid as a result of the anoxemia produced since lactic acid is stronger than HCN by some six orders; the effect may also be due somewhat to a relatively slow outward diffusion of the lactic acid.

Similarly it was found that nerve respiration may be inhibited fairly completely by carbon monoxide in the dark. The constant expressing the relative affinities of the carbon monoxide and oxygen for the iron catalyst represented by the equation $n/1 - n \cdot CO/O_2 = k$ was found to approximate closely to the value of 10 as was found for other cells by Warburg. Furthermore, illuminating the nerve causes a marked decrease of the carbon monoxide inhibition of resting metabolism.

By far the most striking results were obtained when the effect of carbon monoxide on the action potential was studied by means of the cathode ray oscillograph. In mixtures containing from 1 to 3 per cent. of oxygen in carbon monoxide it was found that the height of the action potential decreases progressively to extinction, this decrease being considerably faster than in a similar mixture of oxygen with nitrogen. If during this decline in carbon monoxide the nerve be illuminated by means of an arc-light, the height of the action potential rises immediately and may return to, or even exceed somewhat, the original value. It is important to note that the potential rises immediately with illumination but does not drop at once when the illumination is turned off; the return to the original extinction curve usually takes from 20 to 30 minutes. That the effect is not one of temperature rise or of photo-oxidation is shown by the fact that a companion nerve in nitrogen failing along a similar curve is quite unaffected by the illumination. There is some evidence of small rises in potential in illuminated nerves in presumably *pure* carbon monoxide; the explanation of this is at present not yet clear.

The work is not sufficiently far along to warrant any sweeping generalizations, but it seems clear that the action potential is produced by an oxidation or oxygenation of a substance or substances in nerve, and that for this purpose, activation of the oxygen by a respiratory enzyme similar to that of Warburg's is essential. Since nerves usually do not fail in pure carbon monoxide any faster than in pure nitrogen it appears that the function of the iron catalyst is chiefly to make active oxygen available to the irritable mechanism which when stimulated is then capable of producing the action potential. For the further elucidation of the rôle of the iron catalyst and of the oxidations required for the production of the action potential I am attempting to bring together two distinct lines of research: that of manometric measurement of metabolism, and that of the measurement of the electric potential of nerve. Indeed, some progress has already been made in this direction; I refer to the fact that it is now possible in our hands to obtain accurate records of the height, shape, duration, etc., of the action potential of nerves with the cathode ray oscillograph whilst measuring simultaneously their metabolism manometrically. Only by such a union of methods will the questions raised in this report be adequately answered.

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