may rarely result in starvation of an animal; but usually, if this is the only broken bone, the injury amounts to only a temporary loss of condition and weakening until the fracture mends. Impactions themselves seldom cause important permanent disability either. The long range effects of an actinomycosis infection on a deer are unknown, and the "lumpy jaw" anomaly merits further study.

LITERATURE CITED

BERGSTROM, ANDREA S., JOHN R. PALMATEER AND STUART L. FREE. 1971. Incidence of maxillary canine teeth in white-tailed deer from New York State. N.Y. Fish and Game Jour. 18(1):66-67.

COWAN, IAN M. 1946. Parasites, diseases, injuries and anomalies of the Columbian black-tailed deer, *Odocoileus hemionus columbianus* (Richardson) in British Columbia. Can. Jour. Res. 24(D):71-103.

FOWLE, C. DAVID, AND R. C. PASSAMORE. 1948. A supernumerary incisor in the white-tailed deer. Jour. Mamm. 29(3):301.

JOHNSON, CHARLES E. 1935. Abnormal mandible in white-tailed deer. Jour Mamm. 16(1):69-70.

KARSTAD, LARS. 1967. Flurosis in deer. Bull. Wildl. Disease Assn. 3:42-46.

LUETH, FRANCIS X. 1969. Some anomalies in Alabama deer. Presented at the 49th Annual Meeting of the American Society of Mammalogists. (mimeo.)

MECH, DAVID L., L. D. FRENZEL, JR., PATRICK D. KARNS AND DAVID W. KUEHN. 1970. Mandibular dental anomalies in white-tailed deer from Minnesota. Jour. Mamm. 51(4):804-806.

NEILAND, KENNETH A. 1970. Disease and parasite study. Alaska Federal Aid in Fish and Wildlife Restoration Project W-17-2 (annual report for Work Plan R). Alaska Dept. Fish and Game. (mimeo.)

NORDQUIST, GUS. 1941. Elk teeth in California deer. Calif. Fish and Game 27:39-40.

REES, JOHN W. 1969. Morphologic variation in the cranium and mandible of the white-tailed deer (*Odocoileus virginianus*). A comparative study of geographical and four biological distances. Jour. Morph. 128(1):95-112.

ROBINETTE, W. LESLIE. 1958. Unusual dentition in mule deer. Jour. Mamm. 39(1):156-157.

ROBINETTE, W. LESLIE, AND C. M. ALDOUS. 1955. "Parrot-mouth" in Rocky Mountain mule deer. Jour. Mamm. 36(2):308-310.

RINEY, THANE. 1951. Standard terminology for deer teeth. Jour. Wildl. Mgt. 15(1):99-101.

RYEL, LAWRENCE A. 1963. The occurrence of certain anomalies in Michigan white-tailed deer. Jour. Mamm. 44(1):79-98.

RYFF, J. F. 1953. Encephalitis in a deer due to *Actinomyces bovis*. Jour. Am. Vet. Med. Assn. 122:78-80.

SEVERINGHAUS, C. W. 1949. Tooth development and wear as criteria of age in white-tailed deer. Jour. Wildl. Mgt. 13(2):195-216.

SMITH, HILTON A., AND THOMAS C. JONES. 1966. Veterinary Pathology. Lea and Febiger, Phila., Pa.

SOUTHWICK, CHARLES H. 1954. Canine teeth in a Wisconsin white-tailed deer fawn. Jour. Mamm. 35(3):456-457.

STONE, WARD B. 1970. Investigation of reservoirs of disease in wildlife populations of significance to man and other animals. New York Federal Aid in Fish and Wildlife Restoration Project W-35-R (progress report for Job IV-1). N.Y.S. Dept. Environ. Cons. (mimeo.)

VANGELDER, RICHARD G., AND DONALD F. HOFFMEISTER. 1953. Canine teeth in white-tailed deer. Jour. Wildl. Mgt. 17(1):100.

VERME, LOUIS J. 1968. Possible hereditary defects in Michigan white-tailed deer. Jour. Mamm. 49(1):148.

VERME, LOUIS J., AND JOHN J. OZOGA. 1966. A white-tailed deer jaw with fourth molars. Pack-Pine Warbler. 44:148.

Rotenone, Piscide, Potassium Permanganate, trout, Streams

KINETICS OF ROTENONE-POTASSIUM PERMANGANATE REACTIONS AS APPLIED TO THE PROTECTION OF TROUT STREAMS

4743

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ABSTRACT

Rates of rotenone detoxification by potassium permanganate were estimated by arresting the reaction with tannic acid and estimating rotenone residues by bioassay. At rotenone concentrations up to 50 ppb (1 ppm 5 per cent emulsifiable product), detoxification occurs at a negative exponential rate directly proportional to the potassium permanganate concentration. Other rates are characteristic of higher concentration ranges. At any given rotenone concentration, detoxification time is inversely proportional to potassium permanganate concentration. Temperature has little effect on detoxification rates. Rates for given potassium permanganate and rotenone concentrations are slightly slower in hard than in soft water. Colloidal rotenone in saturated or freshly prepared dispersions is more difficult to detoxify than dissolved rotenone. Correction factors are given for hard water and for water containing organic material. Detoxification criteria are those for trout. Detoxification need not progress as far for centrarchids. Sodium thiosulfate may be used to treat toxic concentrations of potassium permanganate in streams.

Potassium permanganate ($KMnO_4$) detoxifies rotenone in water. This fact, discovered by Lawrence (1956), has been used in numerous stream-reclamation projects. It is assumed that a chemical reaction takes place, but the equation for the reaction remains unknown. Of published data on reaction rates, a study by Jackson (1957) showed that 1 ppm potassium permanganate will detoxify an approximately equal concentration of 5 per cent rotenone formulation within 24 hours. Several published reports of field observations made during stream treatments suggest a practically immediate detoxification.

Other properties of potassium permanganate dictate that it be introduced into natural waters with due care. It, too, is toxic to fish. Maximum concentrations tolerated by trout are in the neighborhood of 3 ppm in soft water and 1.5 ppm in hard water. Most other fish species appear to be more resistant. Since potassium permanganate is a strong oxidizing agent, it is readily broken down by reducing agents. Such treatment renders it non-toxic to fish at ordinary concentrations and prevents possible nuisance situations arising from its red color and tendency to stain laundry and plumbing.

The usual objectives of rotenone-potassium permanganate treatment are (1) removal of unwanted fish from a given stream segment and (2) protection of downstream populations of desirable fish from the toxic effects of rotenone. These ends can be attained by introducing potassium permanganate at a measured rate sufficiently high to completely detoxify the rotenone within a relatively short measured distance below the detoxification station. If the required concentration of

potassium permanganate should be above the tolerance limit for the fish to be protected, it would be necessary to introduce a safe reducing agent at a point sufficiently far downstream from the detoxification station to insure complete detoxification of the rotenone. Use of such a procedure requires a knowledge of stream velocities and transport dynamics and accurate estimates of the rates at which potassium permanganate detoxifies rotenone at various concentrations and under various conditions. To obtain the latter information, a study was conducted at the Department's Fish Laboratory at Livingston Manor, New York. The present paper describes the study, its results and the possible application of these results.

METHODS

Rotenone-containing dispersions of various concentrations were treated with various concentrations of potassium permanganate for various periods of time. At the end of the allotted time, tannic acid ($C_{76}H_{52}O_{46}$) was added and trout were placed in the dispersion. Residual rotenone concentrations were estimated from turnover times on the basis of a concentration-response curve (Loeb and Engstrom-Heg, 1971). All tests were conducted in 5-gallon glass jars, each containing 10 liters of water. Jars were partly immersed in aquarium tanks held at constant temperature. Dispersions were prepared by dilution of concentrated stock dispersions to the appropriate concentration. Except in tests specifically related to the "aging" of freshly prepared dispersions, the dispersions were held overnight (17-18 hours) before each test, to insure complete solution of the rotenone. In each test, trout were also placed in untreated control dispersions to confirm the validity of the concentration-response curve for the fish and rotenone formulation used. Test animals were yearling brown trout of Catskill Hatchery stock, weighing about 75 grams each. The fish had been acclimated for at least a week at the experimental temperatures and were not fed during the 48-hour period prior to an experiment. Rotenone concentrations ranged from 0.025 to 0.25 ppm (0.5 to 5 ppm of 5 per cent emulsified product). Potassium permanganate concentrations ranged from 1 to 10 ppm. Rotenone preparations used included Noxfish [1] and 94 per cent crystalline rotenone dissolved in acetone. Preliminary tests to measure the effectiveness and pinpoint any toxic effects of reducing agents, either alone or in combination with potassium permanganate and/or rotenone, were conducted in the same vessels, with fish of the same stock and under essentially the same conditions.

Hard water used in some tests was prepared by bubbling compressed carbon dioxide through a bed of calcite chips at the bottom of an aquarium tank of laboratory water, and subsequently removing free carbon dioxide by vigorous aeration.

[1] S.B. Penick and Company, Jersey City, N.J. Noxfish consists of: rotenone: 5%; other cube extractives: 10%; solvents and emulsifiers: 85%.

RESULTS

The results of the various tests are presented separately.

PRELIMINARY TESTS ON REDUCING AGENTS

Preliminary tests on reducing agents lead to the following conclusions:

1. Tannic acid and sodium thiosulfate ($Na_2S_2O_3$) both reduce potassium permanganate quickly and efficiently. At a ratio of 2 parts tannic acid to 3 parts potassium permanganate, reduction to tetravalent manganese occurs within 15 seconds. Sodium thiosulfate, applied at a 1:1 ratio, is somewhat slower, but for field use it would be the preferred reducing agent because of its lower price.
2. Reduction of potassium permanganate immediately halts detoxification of rotenone. Noxfish dispersions to which potassium permanganate and tannic acid were added simultaneously did not differ measurably in toxicity from untreated dispersions.
3. Treatment with 6 ppm tannic acid or 10 ppm sodium thiosulfate does not measurably increase or decrease the toxicity of 0.1 ppm Noxfish dispersions.
4. Brown trout tolerate up to 10 ppm of tannic acid as well as the reaction products of at least 50 ppm of potassium permanganate treated with tannic acid at a 2:3 ratio.
5. Brown trout tolerate at least 100 ppm of sodium thiosulfate as well as the reaction products of at least 50 ppm of potassium permanganate treated with sodium thiosulfate at a 1:1 ratio.

TESTS ON NOXFISH IN SOFT WATER WITH NEGLIGIBLE POTASSIUM PERMANGANATE DEMAND

The tests were made with 1 ppm Noxfish (0.05 ppm rotenone). The water used was laboratory spring water (pH, 6.8; TDS, 23 ppm; alkalinity, 14 ppm) held at 65° F.

It was immediately evident that detoxification was not instantaneous. An extensive series of tests showed clearly that at any given potassium permanganate concentration, the logarithms of the residual rotenone concentrations formed a decreasing linear series when plotted against the potassium permanganate-rotenone contact time. Figure 1 shows a typical set of data for a series of contact times at a given potas-

sium permanganate concentration. It was evident that for each concentration, detoxification had occurred at a single negative exponential rate. Rates were computed for each concentration by curvilinear regression, using the original Noxfish concentration of 1 ppm at zero contact time as a fixed point of origin. Regression equations had the form $ln\ [n] = -kt$, where $[n]$ is the residual Noxfish concentration in ppm, t is the contact time in minutes and k is the rate constant. In terms of reaction kinetics, the reactions are of first order with respect to rotenone.

At a given potassium permanganate concentration, the contact time (T) required for detoxification to $[n] = 0.03$ ppm, the appropriate threshold concentration for brown trout (Loeb and Engstrom-Heg, 1971), can be obtained from a semilogarithmic plot by reading the intercept of the regression line with the time axis at $[n] = 0.03$ or by using the equation

$$(1) \quad T = \frac{ln\ ([N]/0.03)}{k} = \frac{ln\ 33.3[N]}{k}$$

where $[N]$ is the *original* Noxfish concentration in ppm.

When the rate constants (k) are plotted against corresponding potassium permanganate concentrations, the relationship is linear, indicating a direct proportion (Figure 2). Regression through a fixed origin, at $[KMnO_4] = 0$ and $k = 0$, yielded the equation

$$(2) \quad k = 0.067[KMnO_4]$$

where $[KMnO_4]$ is expressed in ppm. Equations (1) and (2) may be combined to give

$$(3) \quad T = \frac{ln\ 33.3[N]}{0.067[KMnO_4]}$$

Thus, at initial Noxfish concentrations up to 1 ppm, the rate constant is directly proportional, and the required contact time for detoxification is inversely proportional to the potassium permanganate concentration. Doubling the potassium permanganate concentration halves the required contact time. This simple, elegant relationship depends at least in part on the large excess of potassium permanganate over rotenone (20:1 to 200:1), which creates a situation in which the potassium permanganate concentration remains nearly constant throughout the reaction period. It would no doubt be invalid at low potassium permanganate-rotenone ratios. The equation is clearly applicable to Noxfish concentrations lower than 1 ppm, since a detoxifying dispersion must run through the full range of concentrations from 1 ppm to 0.03 ppm, and in all instances the log-linear relationship was maintained.

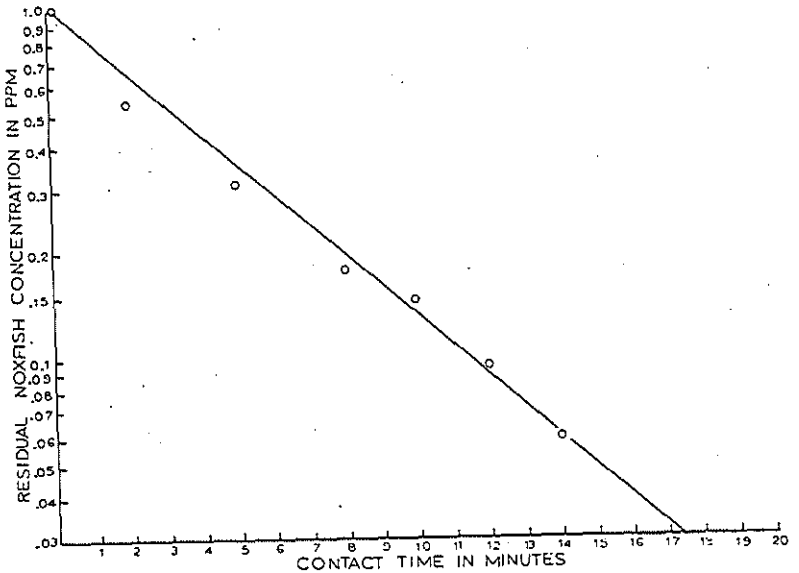

Figure 1. Detoxification of 1 ppm Noxfish by 3 ppm potassium permanganate. Equation for line: $ln[n] = -0.2t$, where $[n]$ is residual Noxfish concentration in ppm and t is contact time in minutes.

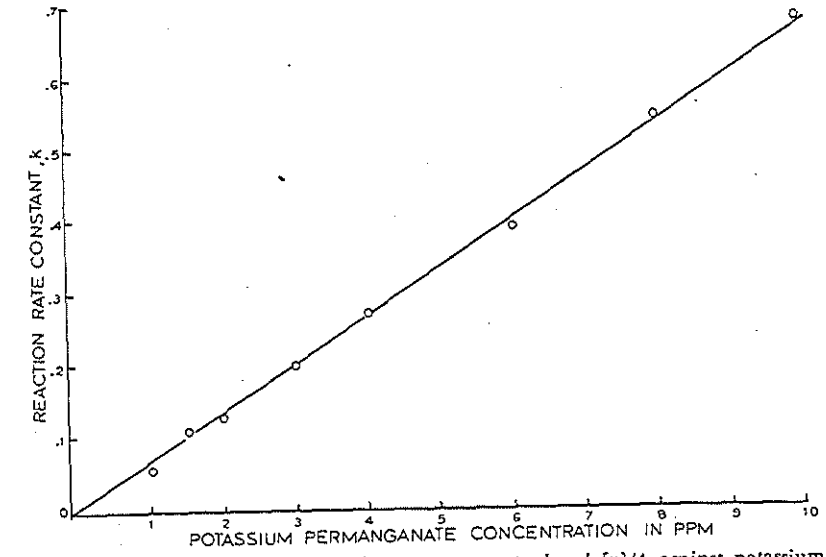

Figure 2. Regression of detoxification rate constants, $k = ln[n]/t$, against potassium permanganate concentration, $k = 0.067[KMnO_4]$.

TESTS ON HIGHER ROTENONE CONCENTRATIONS

Tests at 2 and 3 ppm Noxfish (0.1 and 0.15 ppm rotenone) in laboratory water showed that Equation (3) was not applicable at these concentrations. The early stages of detoxification were as predicted, but there was a low residual toxicity that did not disappear in the predicted time. The plot of this residual "tail" was linear on semilogarithmic paper, again indicating detoxification at a negative exponential rate, but at a slower one than that applying to the main body of toxicant (Figure 3). These test data can be explained by assuming the existence, in each part per million of Noxfish, of a second source of toxicity, having an initial toxicity value equal to 0.07 ppm Noxfish and undergoing detoxification at a rate described by the equation

$$(4) \quad T = \frac{ln\ 2.33[N]}{0.0154[KMnO_4]}$$

Such a toxicity source would be detoxified before rotenone at concentrations less than 1 ppm, but would be the factor controlling detoxification time at higher concentrations. Equation (4) corresponds well with data for 2 and 3 ppm Noxfish and for 0.1 ppm of crystalline rotenone. It was thought at first that this second source of toxicity might be one of the rotenoids present in the "other cube extractives" portion of Noxfish. However, the fact that crystalline rotenone followed the same pattern suggests that rotenone may detoxify in a stepwise manner, and that the first intermediate product may be a slightly toxic substance whose effects are evident at 2 ppm, but not at 1 ppm, Noxfish.

Gimlette (1923) found the solubility of rotenone in water to be approximately 0.16 ppm (equivalent to 3.2 ppm Noxfish). Data obtained by Cohen et al. (1960) tend to support the validity of this figure. At 4 ppm Noxfish (0.2 ppm rotenone), it might be assumed that rotenone would be present in both a dissolved and a colloidal phase. Tests on 4 and 5 ppm Noxfish dispersions indicated the presence of a much more persistent residual toxicity than occurred at 2 and 3 ppm (Figure 4). If it is hypothesized that dispersions more concentrated than 3.2 ppm Noxfish contain colloidal rotenone, and that colloidal rotenone is both less toxic (Loeb and Engstrom-Heg, 1970a) and less easily detoxified than dissolved rotenone, then the data for 4 and 5 ppm may be satisfactorily explained. Differences between residual toxicities at 4 and 5 ppm indicate a toxicity for colloidal rotenone equivalent to 0.14 ppm for each part per million of Noxfish. Using this value, a solubility of 3.2 ppm and the apparent detoxification rate for the residual "tail" at 4 ppm, the equation

$$(5) \quad T = \frac{ln\ 4.66\ ([N] - 3)}{0.00513\ [KMnO_4]}$$

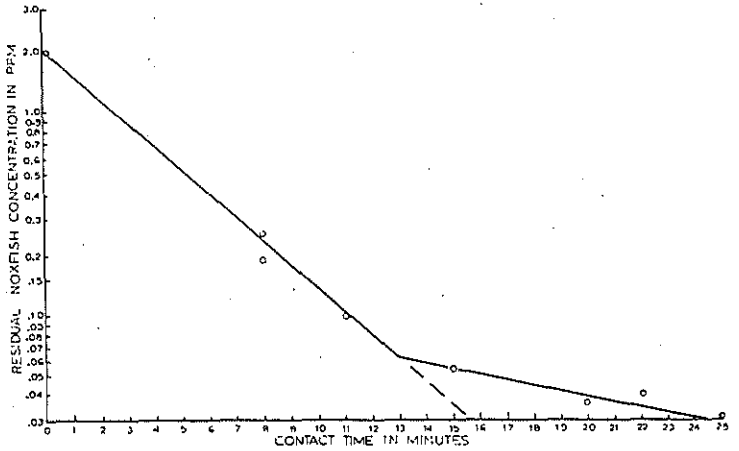

Figure 3. Detoxification of 2 ppm Noxfish by 4 ppm potassium permanganate. Detoxification at 15.7 minutes would be predicted from Equation (3).

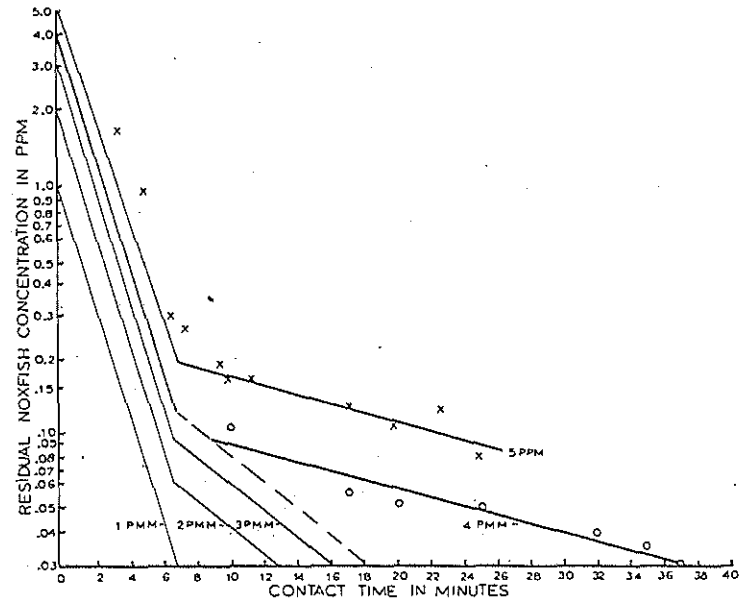

Figure 4. Detoxification of 4 and 5 ppm Noxfish by 8 ppm potassium permanganate. Slopes of residual "tails" correspond with Equation (5). Detoxification times for 1, 2 and 3 ppm Noxfish are also shown. Detoxification of 4 ppm in 18 minutes would be predicted from Equation (4).

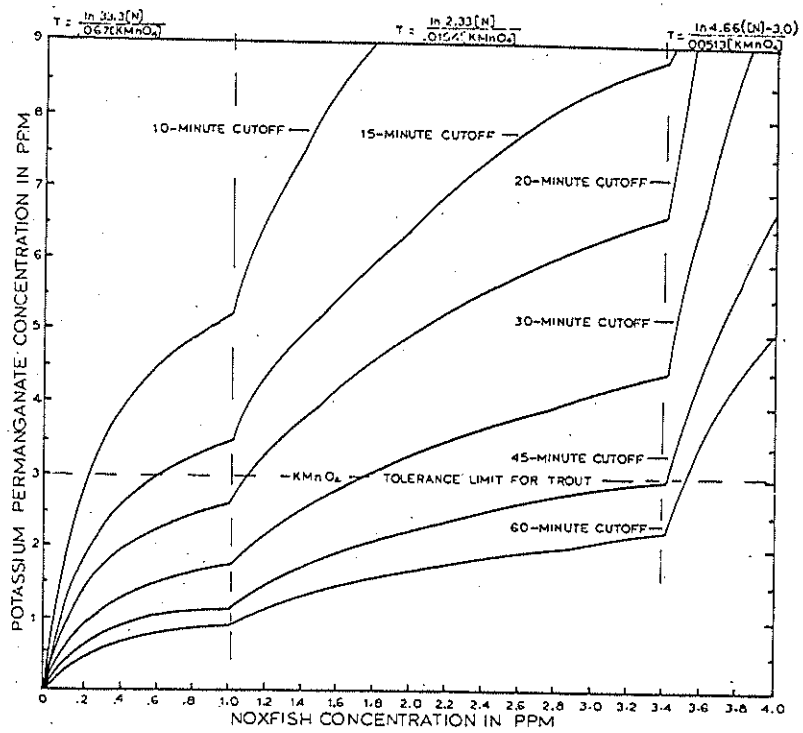

Figure 5. Relationship between Noxfish concentration, potassium permanganate concentration and contact time required for detoxification.

may be derived. This equation predicts that the colloidal rotenone concentration will become the factor controlling detoxification time at all concentrations above 3.5 ppm Noxfish (0.17 ppm rotenone).

Figure 5 shows the relationship between Noxfish concentration, potassium permanganate concentration and contact time required for detoxification to the threshold level for trout, over the full range of values studied. This chart may be used to find the potassium permanganate concentration needed to detoxify a given Noxfish concentration in a given period of time in soft water at temperatures at or near 65° F.

Tests on Freshly Prepared Dispersions

It was found that freshly prepared Noxfish dispersions did not detoxify completely within the predicted contact times for given concentrations of Noxfish and potassium permanganate. The detoxification pattern at 1 ppm resembled that for higher concentrations, indicating the presence of colloidal rotenone as hypothesized by Loeb and Engstrom-Heg (1970a). In still water, a lapse of 3 to 5 hours from

addition of Noxfish to addition of potassium permanganate was needed before detoxification could be obtained in the predicted contact time. When an electric stirrer was used to simulate the turbulent flow of a stream, solution was apparently complete in 1 to 2 hours for 1, 2 and 3 ppm Noxfish, as shown by detoxification within the predicted contact times. Subsequent tests in streams have shown that an hour of "aging" is sufficient to insure detoxification within the predicted time by a given potassium permanganate concentration for Noxfish concentrations up to 2.7 ppm in a pool-and-riffle stream of moderate gradient.

Influence of Temperature

At 47° F., a 1 ppm Noxfish dispersion was detoxified by 3 ppm potassium permanganate in 18.5 minutes. The corresponding figure for 65° F. was 17.5 minutes. Temperature evidently does not have an important effect on the detoxification rate. Since streams are usually reclaimed in the late summer or early fall, the equations based on data for 65° F. should be valid for practical field applications without any corrections.

Influence of Dissolved Electrolytes

Tests conducted at 65° F. with 0.8, 2.0 and 3.6 ppm Noxfish in hard water (pH, 7.8; TDS, 234 ppm; total alkalinity, 171 ppm; potassium permanganate demand, negligible) indicated (Table 1) that approximately 1¼ times as much potassium permanganate was needed to detoxify a given rotenone concentration in a given time in this water as in soft water. A satisfactory correction may be made by multiplying the required potassium permanganate concentration (from Figure 5)

Table 1. Detoxification of Noxfish in Hard Water *

Noxfish concentration in ppm	Potassium permanganate concentration in ppm	Contact time required for detoxification in minutes	Potassium permanganate concentration required for detoxification in same contact time in soft water in ppm	Ratio of concentrations required for hard water and soft water §
0.8	3.3	17.5	2.7	1.22
	4.0	15.0	3.3	1.21
2.0	6.5	20.0	5.0	1.30
	7.8	15.5	6.4	1.22
	8.45	15.0	6.6	1.28
3.6	9.4	30.0	6.8	1.38

* Total alkalinity, 171 ppm.
§ Mean ratio, 1.26.

by a factor equal to 1 + 0.002 (total alkalinity −20). Detoxification occurred within the predicted times when this correction was used to determine potassium permanganate concentrations needed to detoxify 1 ppm Noxfish in a series of dilutions of hard water with laboratory water.

INFLUENCE OF SUSPENDED ORGANIC MATERIAL

Organic materials in colloidal or coarse suspension often reduce potassium permanganate. This is particularly true of humic materials in brown-stained water originating in swamps and bogs. Reduction by such materials is relatively slow compared with the action of tannic acid or sodium thiosulfate. Chlorine demand (Loeb and Engstrom-Heg, 1970b) measured over a 10-minute period, and potassium permanganate demand (Engstrom-Heg, 1971) measured over a period equal to the desired contact time, provided useful indices to the amount of such reducing material present in the stream water.

Jackson (1957) recommended that the potassium permanganate concentration needed for detoxification in chlorine demand-free water be increased by an amount equal to the chlorine demand. Since reduction of potassium permanganate is not immediate, and since the final potassium permanganate concentration would be that needed to detoxify the rotenone in the given contact time, it seemed likely that a smaller allowance for potassium permanganate demand would be adequate. Tests on soft stream water having a 10-minute chlorine demand and 20-minute potassium permanganate demand of 1.6 ppm revealed that 1 ppm Noxfish treated with 4.6 ppm of potassium permanganate was detoxified in 14 minutes. Corresponding values in water with negligible potassium permanganate demand were 17.5 minutes for 3 ppm and 11 minutes for 4.6 ppm potassium permanganate. In this same water, detoxification of 1 ppm Noxfish in 17.5 minutes was achieved with 3.4 ppm potassium permanganate.

Further tests were conducted with laboratory water in which the potassium permanganate demand had been artificially increased by adding measured amounts of instant tea. At 15-minute potassium permanganate demands of 2 and 5 ppm, 15-minute detoxification of 1 ppm Noxfish was achieved with 4.5 and 6 ppm respectively of potassium permanganate, representing in both cases the addition of an amount equal to half the potassium permanganate demand. Since the tea tended to reduce potassium permanganate somewhat more rapidly than corresponding amounts of naturally occurring humic materials, this would seem to be a safe allowance.

DISCUSSION AND APPLICATION

Application of these findings to actual treatment of streams depends on fish management objectives, the physical and chemical characteristics of the stream to be treated, and the fish species present both in the treated section and in the protected area downstream from the detoxification station.

Rotenone concentrations used will depend on the target species. In general, it appears that 6-hour treatment with 1 ppm Noxfish (0.05 ppm rotenone) is sufficient to eradicate all common northeastern freshwater species except bullheads, goldfish and, possibly, large eels. In treating short sections of stream, there may be some advantage in using higher concentrations for a shorter period. However, there would seem to be no advantage in using concentrations above 3.5 ppm (0.17 ppm rotenone). Addition of more colloidal rotenone would not add greatly to the toxicity of the dispersion, but would make detoxification much more difficult. Streams should be treated in late summer or early fall because of the prevailing low stream flows and the greater effectiveness of rotenone at higher temperatures.

Potassium permanganate concentrations needed to detoxify a given rotenone concentration to the toxicity threshold for trout in a given time period can be obtained from Equations (3), (4), and (5) or from Figure 5. Appropriate correction factors can be applied for waters of high alkalinity and/or suspended organic content. Assume, for example, that a hard-water stream having an alkalinity of 220 ppm and a potassium permanganate demand of 2 ppm is to be treated with Noxfish that will arrive at the detoxification station at a concentration of 0.8 ppm. Detoxification in 20 minutes is desired. Reference to Figure 5 gives a value of 2.5 ppm potassium permanganate. The alkalinity correction is made by multiplying this figure by 1 + (200) (0.002) or 1.4, i.e., $2.5 \times 1.4 = 3.5$ ppm. The potassium permanganate demand correction is 2 ppm $\div$ 2 = 1 ppm. The desired concentration would then be 4.5 ppm of potassium permanganate. No temperature correction is needed. This concentration would, of course, require the use of a reducing agent.

The length of the transition zone below the detoxification site depends on stream velocity. Detoxification will be complete in a much shorter distance in a sluggish stream than in a rapid one. If a reducing agent is used, it is important that the downstream limit of rotenone toxicity be located and that the reducing agent be introduced below this point. This may be done by following the leading edge of a bolt of tracer material, either salt or dye. In the previous example, the reducing agent could be introduced at a point 20 minutes downstream from the potassium permanganate station, measured by this method. All rotenone passing this point will have had *at least* 20 minutes contact with potassium permanganate, and the residual rotenone concentration will be safely below the threshold value for trout.

Rotenone treatment of very short sections of trout streams for sampling purposes is probably impractical except where stream velocities are very low. In general, the last toxicant station in any treated section should be far enough upstream from the potassium permanganate station to allow the rotenone to dissolve completely. Presence of a colloidal residue could be troublesome.

Since trout are among the most rotenone-sensitive of fish, treatment of non-trout waters should entail fewer problems. Most centrarchid species tolerate about three times as much residual rotenone as do trout, and most other freshwater fish are still more tolerant. Hence, a given potassium permanganate concentration will detoxify a stream for bass and sunfish in a markedly shorter distance than for trout. It seems likely that, in centrarchid waters, the effects of low residual "tails" could be ignored and that an equation similar to (3), but based on the threshold concentration for the most sensitive species, could be used for all rotenone concentrations. In field tests, very few fish other than trout were observed to be in distress at any distance downstream from the potassium permanganate station.

LITERATURE CITED

COHEN, J. M., L. J. KAMPHAKE, A. E. LEMKE, C. HENDERSON AND R. L. WOODWARD. 1960. Effect of fish poisons on water supplies. Part I. Removal of toxic materials. Jour. Am. Water Works Assn. 52(12):1551-1566.

ENGSTROM-HEG, ROBERT. 1971. Direct measurement of potassium permanganate demand and residual potassium permanganate. N. Y. Fish and Game Jour. 18(2):00-00.

GIMLETTE, J. D. 1923. Malay Poisons and Charm Cures. Churchill, London.

JACKSON, C. F. 1957. Detoxification of rotenone-treated water. Tech. Circ. 14, N. H. Fish and Game Dept.

LAWRENCE, J. M. 1956. Preliminary results on the use of potassium permanganate to counteract the effects of rotenone on fish. Prog. Fish-Cult. 18(1):15-21.

LOEB, HOWARD A., AND ROBERT ENGSTROM-HEG. 1970a. Time-dependent changes in toxicity of rotenone dispersions to trout. Toxicol. and Applied Pharmacol. 17:605-614.

—— 1970b. Determination of chlorine demand. N. Y. Fish and Game Jour. 17(2):138-139.

LOEB, HOWARD A., AND ROBERT ENGSTROM-HEG. 1971. Estimation of rotenone concentration by bio-assay. N. Y. Fish and Game Jour. 18(2):129-134.

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