Aquatic Herbicide Chronicity[1]

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ABSTRACT

Eurasian watermilfoil (*Myriophyllum spicatum* L.), wild celery (*Vallisneria americana* Michx.), and waterweed (*Elodea canadensis* Michx.) were subjected to long-term exposure of low concentrations of three herbicides to measure chronic effects in an effort to determine the feasibility of slow-release herbicide application. Matrix analysis of the data indicate that chronic effects can be determined in the laboratory, independent of plant species or herbicide, ranging from 10 to 31 days of exposure.

INTRODUCTION

The concept that there is no threshold below which a chemical agent does not induce a biological interaction is one theory which has received widespread acceptance. This concept is true in that, when one atom or molecule enters the reactive region of another, a chemical interaction will result. This would imply that the entrance of even one molecule into the protoplast will produce a biological effect that is deleterious. If the word "effect" is used in a strict sense, we agree with the probability that the interaction of one pollutant molecule with another molecule of a cell component could result in an "effect." It is our position, however, that stochastic determinants impose a lower limit on the dose-response relationship between molecules within the homogeneous mass of the protoplast. If the interaction is accumulative in terms of mass action, the reaction will move to the right and the plant will slowly die. Such a relationship at some threshold level, is postulated in slow-release herbicide treatment (1, 2, 5, 6).

Acute Toxicity. The effect of a herbicide depends on many complex factors. Although no single way of expressing potential effect is completely reliable, the most rapid and convenient indicator for most chemicals is the acute toxicity (the single dosage necessary to produce death). The usual way of expressing acute toxicity is by means of an LD_{50} (median lethal dosage) value. The LD_{50} is a statistical estimate of the dosage that would be lethal to 50% of a large population of the test species. Although LD_{50}'s give no information on the dosage that would be lethal to every individual of the species, or to treatment given in some other way than in the test, the LD_{50} value, with its confidence limits, is still probably the most convenient and reliable means available for comparing the inherent toxicity of chemicals. There is enough similarity between cases to establish rules of thumb relating lethal levels for a species in laboratory toxicity tests to potential effects from field exposure. One method is to predict the field effects of a new chemical by comparing its LD_{50} for the species of interest with the LD_{50} of a chemical whose field effects are known. Thus, if herbicide X has an LD_{50} of 10 $mg \cdot kg^{-1}$ and is known to kill plants in the field when applied at 2.24 $kg \cdot ha^{-1}$, herbicide Y with an LD_{50} of 5 $mg \cdot kg^{-1}$ might be expected to kill plants 1.12 $kg \cdot ha^{-1}$ (3, 7, 8).

Subacute Toxicity. An empirical value that represents the minimum repeated dosage ($mg \cdot kg^{-1} \cdot day^{-1}$) that is lethal has been determined. This was found by administering the chemical daily for the test species. Such groups were treated at geometrically spaced dosage levels until dosage levels were found that produced 25, 50, and 90 % mortality. This program to establish a subacute dosage or treatment from which the plant could not recover, is defined as the chronic dosage that would be suitable for slow-release herbicide application. Test levels within the 25 to 90% mortality range are approximately straight-line functions (1, 2, 5, 8).

METHODS AND MATERIALS

Three herbicides, 6,7-dihydrodipyrido[1,2-:2′,1′-*c*]pyrazindiinium dibromide salt (diquat) in water solution, (2,4-dichlorophenoxy)acetic acid butoxyethyl ester (2,4-DBEE) in water solution, and 2-(2,4,5-trichlorophenoxy) proprionic acid propylene glycol butyl ether ester (silvex) with sufficient acetone to keep in solution were prepared by serial dilution for 1.0, 0.1, and 0.01 ppm stock volume of each test container for the proper concentrations. Two basic experiments were performed. In one, the herbicide was added daily so that the dose was accumulative for each rate of application. In the second case, the testwater (Hoaglands Solution) was changed every other day so that the concentration would be constant for each rate of application. Watermilfoil, wild celery, and waterweed were grown in 4-liter jars in the laboratory. A photoperiod of 14 hr of normal laboratory light was used in all the treatments. Plants were conditioned 4 weeks prior to the addition of the herbicide. All treatments were run in replicates of four. Plant mortality was subjectively rated based on a rating of 0 for a green healthy leaf and 100 for a brown

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dead leaf. Controls were run for all tests for comparison with the herbicide treatments.[2]

RESULTS AND DISCUSSION

In Tables 1, 2 and 3, it is observed that differences related to percent mortality, concentration of herbicide, specific herbicide applied, method and time of exposure, do exist. Matrix analysis of the data indicates that those differences due to plant species as such are relatively small. The amount of toxicant, i.e., rate of application, the concentration and specific herbicide, and the method and time of exposure are highly significant. For a 90% mortality, the estimated values are given in Table 4, ranging from 10 to 31 days exposure. Under laboratory conditions, it was possible to demonstrate the chronic effects of herbicides at levels of 1, 0.1, and 0.01 ppm concentration. It is thought that a field application of 0.1 ppm would represent a level feasible for operational control. The exposure time required is estimated at 19, 23, and 27 days for diquat, 2,4-DBEE, and silvex, respectively, under accumulative treatment conditions. However, suitable field trials would be necessary to determine more exactly the levels required under an operational program.

[2]Quinn, S. A. and Cardarelli, N. F. 1972. Aquatic Herbicides Chronicity Study, Ann. Rept., U.S. Army Corps of Eng., Wash., D.C., DACW 73-72-C0031, AD 903208.

TABLE 1. CHRONIC DOSE LEVELS FOR WATERMILFOIL AS DAYS TO MORTALITY.

Herbicide	Concentration ($ppm \cdot day^{-1}$)	Accumulative dose 25%	Accumulative dose 50%	Accumulative dose 90%	Constant dose 25%	Constant dose 50%	Constant dose 90%
Diquat	1.0	5	9	13	6	9	11
	0.1	6	9	13	6	9	13
	0.01	7	10	14	6	9	13
2,4-DBEE	1.0	10	10	11	7	8	11
	0.1	10	11	15	7	9	14
	0.01	10	11	14	7	11	22
Silvex	1.0	5	8	13	6	8	12
	0.1	11	17	23	13	14	34
	0.01	11	14	20	18	34	34

TABLE 2. CHRONIC DOSE LEVELS FOR WILD CELERY AS DAYS TO MORTALITY.

Herbicide	Concentration ($ppm \cdot day^{-1}$)	Accumulative dose 25%	Accumulative dose 50%	Accumulative dose 90%	Constant dose 25%	Constant dose 50%	Constant dose 90%
Diquat	1.0	4	4	5	4	4	4
	0.1	5	5	7	4	4	4
	0.01	8	11	18	10	16	50
2,4-DBEE	1.0	9	11	15	8	24	37
	0.1	11	17	31	10	16	38
	0.01	22	26	46	12	25	39
Silvex	1.0	15	23	27	11	19	29
	0.1	27	39	49	13	19	27
	0.01	51	51	51	16	19	24

TABLE 3. CHRONIC DOSE LEVELS FROM WATERWEED AS DAYS TO MORTALITY.

Herbicide	Concentration ($ppm \cdot day^{-1}$)	Accumulative dose 25%	Accumulative dose 50%	Accumulative dose 90%	Constant dose 25%	Constant dose 50%	Constant dose 90%
Diquat	1.0	9	15	20	9	11	13
	0.1	15	17	19	10	11	15
	0.01	17	22	35	10	12	22
2,4-DBEE	1.0	6	12	35	6	7	10
	0.1	9	31	35	6	7	11
	0.01	17	35	35	6	9	13
Silvex	1.0	10	11	14	8	10	12
	0.1	9	11	14	9	11	13
	0.01	28	29	46	8	9	13

Table 4. Estimated time in days to 90% mortality for herbicide chronicity treatments as the mean effect of the plant species studied.

Exposure	Herbicide	Concentration (ppm)	Time (days)
Constant	Diquat	1.00	10
Constant	Diquat	0.10	15
Constant	Diquat	0.01	16
Accumulative	Diquat	1.00	15
Accumulative	Diquat	0.10	19
Accumulative	Diquat	0.01	24
Constant	2,4-DBEE	1.00	15
Constant	2,4-DBEE	0.10	19
Constant	2,4-DBEE	0.01	24
Accumulative	2,4-DBEE	1.00	19
Accumulative	2,4-DBEE	0.10	23
Accumulative	2,4-DBEE	0.01	27
Constant	Silvex	1.00	18
Constant	Silvex	0.10	23
Constant	Silvex	0.10	28
Accumulative	Silvex	1.00	23
Accumulative	Silvex	0.10	27
Accumulative	Silvex	0.01	31

LITERATURE CITED

1. Cardarelli, Nate. 1975. Hypothesis concerning chronic intoxication of aquatic weeds, pp. 344-357. *In* International Controlled Release Pesticide Symposium. College of Science and Engineering. Wright State University, Dayton, Ohio 406 pp.
2. Dinman, B. D. 1972. "Non-concept of No-Threshold": Chemicals in the Environment. Sci. 175:495-497.
3. Elliston, R. A. and Steward, K. K. 1972. The response of Eurasian watermilfoil to various concentrations and exposure periods of 2,4-D, Hyacinth Contr. J. 10:38-40.
4. Gangstad E. O. 1971. Aquatic Plant Control Program. Hyacinth Contr. J. 9:46-48.
5. Hayes, Wayland J. 1967. The 90-dose LD_{50} and a chronic factor as measure of toxicity. Toxicol. and Appl. Pharm. 2:327-335.
6. Koish, MG F. P. 1971. Remarks relating to the current environmental crisis. Hyacinth Contr. J. 9:2-4.
7. Krawiec, Stanislaw and D. James Morre. 1968. Interactions of tordon herbicide applied in combinations. Down to Earth (Winter) pp. 7-10.
8. Stewart, K. K. and L. L. Nelson. 1972. Evaluation of controlled release PVC and attaclay formulations of 2,4-D on Eurasian watermilfoil. Hyacinth Contr. J. 10:35-38.