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Effect of Magnacide® H Herbicide Residuals on Water Quality Within Wildlife Refuges of the Klamath Basin, CA

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Widespread attention and concern is focused on the toxicity and persistence of chemicals that come in contact with the environment. Magnacide® H Herbicide (active ingredient: 92% minimum acrolein, inhibited) is used in agricultural canals as an injectable aquatic herbicide to control aquatic weeds. Acrolein contains two functional groups, a reactive double bond and an aldehyde grouping. These groups react rapidly with amine, alcohol and mercaptan groups of aquatic plants, destroying cell structure and thereby killing the plants (Parent *et al.,* 1992; Parent et al., in press). Acrolein reacts with the same nucleophiles in nontarget species producing a variety of adverse effects (Beauchamp *et al.,* 1985). Because acrolein is highly toxic to aquatic life (USEPA, 1986), it represents a potential for water quality concern near waterbodies where used.

Although the use of acrolein herbicide may present a hazard to nontarget receptors, information is limited regarding the persistence, fate and impact of acrolein on environmental waters. The available data indicates that when acrolein is applied to agricultural canals at the recommended concentration, its presence is transient with a dissipation half-life of approximately 7.0 hours (O'Loughlin and Bowmer, 1975; Bowmer and Higgins, 1976; Bowmer and Sainty, 1977). During the summer irrigation months, Magnacide® H is used routinely in the agricultural canals of the Tulelake Irrigation District (TID), California. The aim of this cooperative study between Baker Performance Chemicals (BPCI) and California Regional Water Quality Control Board (CRWQCB) was to determine whether acrolein was reaching downstream natural receiving waters of the Tulelake sump and adjacent wildlife refuges and to assess the potential for an adverse impact on nontarget aquatic species. A "worst-case" scenario was used in which the farthest downstream application site was monitored. This was determined to be the site most likely to result in introduction of acrolein into the Tulelake sump.

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MATERIALS AND METHODS

Acrolein (95.9% purity) and the reagents for the polarograph and the Magnacide® H Monitor were supplied by Baker Performance Chemicals, Inc. (BPCI). Pentafluorophenylhydrazine and P-5 (5% methane in argon) were supplied by both Baker Performance Chemicals, Inc. and Agricultural and Priority Pollutants Laboratories (APPL) (Fresno, CA). Acrolein-pentafluorphenylhydrazone was synthesized by BPCI and APPL. All other reagents were of analytical grade or better.

The test site was located in Siskiyou County, California, within the TID. The study site consisted of a 2.7 km length of the J-1 irrigation canal connected to a 2.4 km drain (40-A). Water from the canal enters the drain, travels its length where it reaches pump 3 and is periodically pumped into Tulelake sump. Under current management practices employed by TID when applying acrolein to canals, flows from the treated canal to drains are closed. The route of acrolein reaching the drain under our sampling conditions would have been through minor incidental leakage common to earthen irrigation systems at locations where the canal and drain meet.

Three separate applications of acrolein were made to the J-1 canal with different target concentrations of acrolein and at different water flow rates, giving the opportunity to monitor under three different operating scenarios. The application data are summarized in table 1. Each application was made by forcing the acrolein from a 168 kg net portable steel cylinder with oxygen-free nitrogen and introducing the acrolein directly into the irrigation canal through a 15.25 m injection hose.
Water samples were taken from 13 sampling points along the length of the J-1 Canal

Table 1. Details of the three Magnacide® H Herbicide applications.

Application Number	Date	Time of Treatment	Water Flow (L/sec)	Target Conc. (ppm)
1	7/13/94	0600-0648	142	7.5
2	7/21/94	0840-0927	283	11.6
3	8/10/94	0855-0938	453	10.4

(upstream, 1-6) and drain 40-A (6A - drain leakage, 7 - drain entry, 7A,B,C,D - every 75 ft from point 7). The detection of the approach of the acrolein wave at each sampling point was determined by semi-quantitative analysis using a Magnacide® H Monitor. This employs a calorimetric method to compare 5 ml canal water samples to a Magnacide® H-free water sample (blank) following the reaction of the sample with 1 ml 2,4-dinitrophenylhydrazine, 1 ml NaOH and 3 ml isopropyl alcohol. The peak acrolein concentration of the wave was determined quantitatively by differential

pulse polarography. A model 264A polarographic analyzer (E. G. and G. Parc) with a Model 303 static mercury drop electrode was used with the following instrument settings: scan range = -0.90 to -1.40 V (vs. Ag/AgCl); drop time = 1 sec; pulse height = 100 mV; mode = differential pulse. Sensitivity was 100 mA for acrolein concentrations of ≥ 1000 ppb and 100 µA for ≤ 1000 ppb, which required a 2 and an 8 minute purge with nitrogen, respectively, to avoid signal interference from oxygen. Analysis was performed using 10 ml of canal water spiked with 1 ml of a phosphate buffer (pH 7.0). Rhodamine and fluorescein dyes were also used as general visual tracers. At each sampling time, samples of 250 ml were taken from well mixed areas of the canal and drain for off-site analysis. These samples were split for independent analysis by APPL and BPCI.

All samples for off-site analysis were filtered and derivatized in the field immediately after they were collected and then stored on ice. Approximately 50 ml of the sample were withdrawn into a plastic syringe and filtered through a 5.0 um Millipore filter into a silanized amber bottle. A 2.5 ml aliquot of a buffer solution (1M KH_2PO_4, pH 5.0) was then added followed by a 1.0 ml aliquot of pentafluorophenylhydrazine (PFPH) (2.658/l methanol). The bottle was shaken, capped, sealed and placed on ice in a cooler for shipment. Field derivatization with hydrazone prevented degradation from occurring after the samples were removed from the canal.

Derivatized aqueous samples were extracted twice with 25 ml volumes of ethyl acetate in a 125 ml separatory funnel. After each extraction the ethyl acetate layer was passed through a 2 - 2.5 cm layer of anhydrous Na_2SO_4 which was rinsed with two 5 ml portions of ethyl acetate. A Hewlett Packard 5890 gas chromatograph was used with an electron capture detector at 350°C. Using a split/splitless injector, the injection volume was 1.0 µl with the injector purge off for the first minute of the chromatographic period, then opened. The column was a DB-1701, 30 m x 0.32 mm I.D. with a 1.0 micron film thickness (J and W Catalogue #123-0733) and maintained at 350°C. Following an initial oven temperature of 120°C for one minute, the temperature was raised l0°C/minute to 220'C with a second ramp rate of 30°C/minute to a temperature of 280°C and then held for 2.0 minutes. The column flow rate was 15 - 20 ml/min of helium into the injector which provided excess flow for the purge and a column head pressure of 1.0 atmosphere. The detector flow rate was 55 ml/min of P-5 (5% methane in argon). Method validation indicated an acceptable detection limit (MDC) of 5.0 ppb for acrolein.

RESULTS AND DISCUSSION

Tables 2-4 list the peak acrolein concentrations at the various sampling points and times for the three applications. Using polarographic analysis, peak concentrations of 3310, 7630 and 7100 ppb were detected at sample point 1 for applications 1, 2 and 3, respectively. At sample point 6, peak acrolein concentrations had fallen to 127, 2120 and 3100 ppb for applications 1, 2 and 3, respectively. This represents dissipation rates of 96, 72 and 55% within a 12.5, 6.9 and 8.8 hour period. For all three

applications, regression analysis using sampling data from the J-l canal only indicates that the decay of acrolein can be modeled as a first-order decay process. The dissipation half-life for acrolein was 2.6 (r = -0.974), 3.3 (r = -0.959) and 7.3 (r = -0.962) hours. Corresponding rate constants are 0.267 hr^{-1}, 0.21 hr^{-1} and 0.095 h r^{-1}.

Samples selected for PFPH analysis were concentrated in the area of canal leakage (6A) and in the downstream drain (7 - 7D) leading to Tulelake sump. Detectable concentrations of acrolein were found in the canal leakage water following all three applications. The PFPH analysis resulted in acrolein concentrations of 5.9, 17-23.4 and 160-804 ppb at sample point 6A for applications 1, 2 and 3, respectively. Despite this, acrolein concentrations in all water samples from drain 40A (7 - 7D) immediately downstream of the canal were non-detectable.

We have demonstrated that the use of Magnacide® H, when applied under the conditions of this study, as an aquatic herbicide in agricultural canals does not result in the introduction of acrolein into natural receiving waters at a distance of approximately 2.7 km downstream. In other field experiments, the kinetics and half-life of acrolein concentrations in irrigation canals were similar to those observed in this study (O'Loughlin and Bowmer, 1975; Bowmer and Higgins, 1976; Bowmer and Sainty, 1977). The mean first order rate constant from eight canals was 0.163 hr^{-1} (SD = ±0.039) and varied between 0.104 and 0.211 hr^{-1} at pH 7.1 to 7.5 and water temperatures of 16 - 24°C (Bowmer and Sainty, 1977). The mean value corresponds to a half-life of 4.25 (SD = ±1.18) hours at a mean temperature of 21°C (SD = ±2.62).

Table 2. Concentration of acrolein in canal J-1 and drain 40A following application 1 of Magnacide® H Herbicide.

Sample Point	Distance From Application Point (km)	Study Time (Hours)	Peak Conc. (ppb)	ln (Conc.)
1	0.2	0.8	3310	8.1
2	0.9	3.6	1970	7.6
3	1.7	6.3	1040	7.0
4	2.1	8.3	648	6.5
5	2.5	11.1	268	5.6
6	2.7	12.5	127	4.8
6A	2.7	12.5	6*	1.8
7-7D	+2.7	12.5-14.7	ND*	-

*Concentrations calculated using PFPH analysis.

Table 3. Concentration of acrolein in canal J-1 and drain 40A following application 2 of Magnacide® H Herbicide.

Sample Point	Distance From Application Point (km)	Study Time (Hours)	Peak Conc. (ppb)	ln (Conc.)
1	0.2	0.9	7630	8.9
2	0.9	2.0	7080	8.9
3	1.7	3.6	5350	8.6
4	2.1	5.1	4000	8.3
5	2.5	6.6	2710	7.9
6	2.7	6.9	2120	7.7
6A	2.7	7.7	23*	3.1
7-7D	+2.7	7.7-8.8	ND*	-

*Concentrations calculated using PFPH analysis.

Table 4. Concentration of acrolein in canal J-1 and drain 40A following application 3 of Magnacide® H Herbicide.

Sample Point	Distance From Application Point (km)	Study Time (Hours)	Peak Conc. (ppb)	ln (Conc.)
1	0.2	0.5	7100	8.9
2	0.9	2.1	6600	8.8
3	1.7	4.0	5500	8.6
4	2.1	7.2	4400	8.4
5	2.5	8.5	3400	8.1
6	2.7	8.8	3100	8.0
6A	2.7	9.1	804*	6.7
7-7D	+2.7	8.3-11.7	ND*	-

*Concentrations calculated using PFPH analysis.

Similar kinetics for acrolein degradation have been shown previously in the laboratory where it was also demonstrated that the rate constants are independent of the initial acrolein concentrations but increase with decreasing pH (hydration) and increasing pH (condensation) (Pressman and Lucas, 1942; Hall and Stern, 1950; Bowmer and Higgins, 1976). The half-life of 46 hours (0.015 hr^{-1}) calculated using dilute buffered solutions of acrolein in distilled water at 21 °C and pH 7.0 is considerably longer than the half-life observed in this and other field experiments. In laboratory experiments, however, the half-life is influenced by the presence of the acrolein reaction products. Equilibrium is reached with 8 % of the original acrolein and 85 % of total aldehydes still present, but these do not persist in environmental waters where other methods of dissipation exist (Bowmer *et al.,* 1974; Bowmer and Higgins, 1976). The results of field studies do not indicate such an equilibrium and are more consistent than the laboratory data with the results of bioassays with bacteria and fish which show the loss of biocidal activity of aged acrolein solutions after approximately 120 to 180 hours at a pH of 7.0 (Kissel et al., 1978).

The rate of decay of acrolein in environmental waters is influenced by processes other than hydration such as volatilization, adsorption and dilution (Frank, 1970; Bowmer *et al.,* 1974). It has been demonstrated that acrolein would be readily degraded by mixed microbial populations (Stover and Kincannon, 1983) while others have indicated that its toxicity to microorganisms may prevent biodegradation (Chou *et al.,* 1978; Bridie *et* al., 1979). It has been suggested (Bowmer and Higgins, 1976) that the fall in pH observed during the decline of the acrolein reaction product in laboratory experiments is indicative of the formation of a carboxylic acid which is also produced during the microbial oxidation of aldehydes (Meikle, 1972).

The extreme toxicity of acrolein to aquatic organisms represents a significant risk to the aquatic environments of natural receiving waters. In a 3-generation 64-day study with the crustacean *Daphnia magna,* the highest concentration that did not result in mortality was 16.9 ppb. Survival was reduced at levels in excess of 33.6 ppb, but the number of young per female was not affected even at the highest concentration tested, 42.7 ppb (Macek *et* al., 1976). In a 60-day test with fathead minnow (*Pimephales promelas*), the highest concentration without adverse effects was 11.4 ppb (Macek *et al.,* 1976). Increased mortality among offspring was demonstrated at 41.7 ppb. No adverse effects were found on survival and mortality of adults, number of spawnings and number of eggs per female, number of eggs per spawn, length of offspring, or hatchability. We have demonstrated that acrolein concentrations in all water samples from drain 40A immediately downstream of the canal were nondetectable using an analysis with an MDL of 5.0 ppb; thus, acrolein did not adversely impact the natural receiving waters of the Tulelake sump and surrounding wildlife habitats. When Magnacide® H is applied to agricultural canals under the conditions of this study, the presence of acrolein in water is transient. Biologically significant levels of acrolein do not reach downstream natural receiving waters and subsequently do not adversely impact natural aquatic environments.

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REFERENCES

Beauchamp RO, Andjelkovich DA, Kligerman AD, Morgan KT, Heck Hd'A (1985) A critical review of the literature on acrolein toxicity. CRC Crit Rev Toxicol 14(4):309-380

Bowmer KH, Higgins ML (1976) Some aspects of the persistence and fate of acrolein herbicide in water. Arch Environ Contam Toxicol 5:87-89

Bowmer KH, Lang ARG, Higgins ML, Pillay AR, Tchan YT (1974) Loss of acrolein from water by volatilization and degradation. Weed Res 14:325-328

Bowmer KH, Sainty GR (1977) Management of aquatic plants with acrolein. J Aquat Plant Manage 15:40-46

Bridie AL, Wolff CJM, Winter M (1979) BOD and COD of some petrochemicals. Water Res 13:627-630

Chou WL, Speece RE, Siddiqi RH (1978) Acclimation and degradation of petrochemical wastewater components by methane fermentation. Biotechnol Bioeng Symp 8:391-414

Frank PA (1970) Degradation and effects of herbicides in water. 1st FAO Int Conf Weed Control, WC/70: WP/28, p 22

Hall RH, Stern ES (1950) Acid-catalyzed hydration of acraldehyde. Kinetics of the reaction and isolation of B-hydroxypropionaldehyde. J Chem Soc 490-498

Kissel CL, Brady JL, Guerra AM, Pau JK, Rockie BA, Caserio FF (1978) Analysis of acrolein in aged aqueous media. Comparison of various analytical methods with bioassays. J Agric Food Chem 26(6):1338-1343

Macek KJ, Linberg MA, Sauter S, Buxton KS, Costa PA (1976) Toxicity of four pesticides to water fleas and fathead minnows, US Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Duluth, Minnesota (Ecological Research Series EPA-600/3-76-099)

Meikle RW (1972) In: Goring, CAI, JW Hamaker (eds) Organic chemicals in the soil environment, Dekker, New York, p 184

O'Loughlin EM, Bowmer KH (1975) Dilution and decay of aquatic herbicides in flowing channels. J Hydrol 26:217-235

Parent RA, Caravello HE, Long JE (1992) Two-year toxicity and carcinogenicity study of acrolein in rats. J Appl Toxicol 12(2):131-139

Parent RA, Caravello HE, San RHC (in press) Mutagenic activity of acrolein in S typhimurium and E coli. J Appl Toxicol

Pressman D, Lucas HJ (1942) Hydration of unsaturated compounds. XI. Acrolein and acrylic acid. J Am Chem Soc 64:1953-1957

Stover DL, Kincannon DF (1983) Biological treatability of specific olrganic compounds found in industry wastewaters. J Water Poll Control Fed 55:97-109

US EPA, Health Assessment Document for Acrolein, Washington, DC (1986), EPA/600/-8-86-014A, PB87-139960, pp 1-1 - 8-5