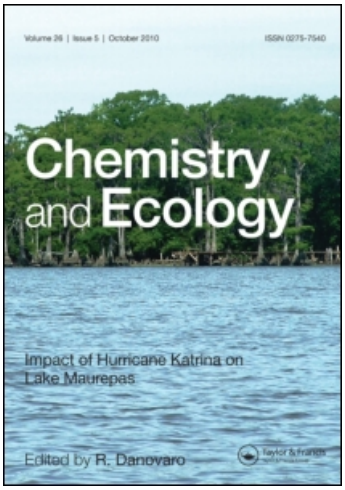

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Organophosphate Residues in Grasshoppers from Sprayed Rangelands

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Grasshoppers (Orthoptera) were collected in pastures that had been sprayed with malathion and acephate to estimate the secondary exposure of insectivorous birds to those pesticides. Residues of malathion were below 3 ppm at 30 and 54 hours after spraying and no malaoxon was detected. In contrast, acephate was found at 8 and 9 ppm 4 hours after spray; 3–5 ppm of the toxic metabolite methamidophos were also detected at that time. By 53 hours postspray, acephate levels declined to 2 ppm and methamidophos to less than 1 ppm. These results suggest that although malathion may not be a hazard to insectivorous species, acephate may be hazardous through metabolic transformation to methamidophos.

The replacement of chlorinated hydrocarbon insecticides by organophosphate and carbamate insecticides has generated extensive laboratory testing of the toxicity of these compounds to non-target organisms (e.g., Tucker and Crabtree, 1970; Hill *et al.*, 1975). Interpretation of the results of avian toxicity tests is complicated by the paucity of data on the actual dietary exposure of birds under field conditions. For seed-eating birds and seed treatment pesticides, direct calculation of potential exposure can be based on known application rates (Stromborg, 1977). However, most bird species are insectivorous during the months of heaviest pesticide application and there is no simple procedure for estimating pesticide intake from the insects eaten. Instances of avian secondary poisoning (eating animal foods contaminated with poisons) by organophosphates have occasionally been reported (Mendelssohn and Paz, 1977; White *et al.*, 1979) but the

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extent of such poisoning is largely a matter of speculation (Mills, 1973; Stickel, 1975).

Several alternative approaches for indirect assessment of potential secondary poisoning hazard can be suggested. Although the literature on toxicity of organophosphate pesticides to insects (e.g., MacDonald, 1967; Smith, 1977) could be utilized, the toxic mode of action results in an irreversible deactivation of the insecticide (Murphy, 1975:423). These data therefore reflect the amount of insecticide needed to kill insects, not the amounts of unreacted insecticide, or its active metabolites, which are present in the insects and constitute the secondary hazard. Model ecosystems (see Metcalf, 1977 for review) have been used to predict the environmental fate of insecticides in terrestrial systems (Cole *et al.*, 1976). These studies are designed to identify the products of complex environmental interactions over time compared to the relatively rapid effects on birds observed under field conditions (White *et al.*, 1979). The conditions are also greatly simplified and may not adequately represent actual field applications. Dittrich (1966) poisoned nestling birds by feeding them caterpillars that had been previously given a sublethal dose of phosphamidon. This was a direct demonstration of secondary poisoning hazard; whether the doses used represented field conditions was not specified.

All of the preceding indirect approaches to the problem of secondary hazard lack one key feature: an estimate of the amount of active insecticide and/or active metabolites present in avian insect prey items during the first few days after an operational insecticide application. This report supplies such information gathered during grasshopper control programs in Colorado and Montana.

METHODS

Malathion-poisoned grasshoppers were obtained from two separate 64.8-ha sites within a 20,250 ha block of weedy mixed prairie rangeland in southwest Weld County, Colorado, that had been sprayed with 0.614 kg/ha (8oz/acre) malathion in an ultra-low-volume (ULV) formulation. The Range Insect Laboratory of the U.S. Department of Agriculture made these applications to test grasshopper control efficacy. Active grasshoppers were collected with sweep nets; dead, dying and immobilized grasshoppers were picked up by hand. Grasshoppers were grouped by apparent health status, placed on dry ice soon after

collection and stored in a frozen condition until processed for chemical analysis. Samples were taken 30 h and 50 h posttreatment. Grasshoppers were also collected from acephate-sprayed mixed prairie range located in Sweet Grass County, south-central Montana. These grasshoppers were taken from the central portions of two 16.2-ha plots treated with 0.614 kg/ha (8oz/acre) acephate in a low-volume water formulation. Samples were collected 4 h and 53 h posttreatment and individuals were grouped by physical status and time of collection.

Weights of individual composite samples of grasshoppers ranged from about 6 to 14 g. An average weight for an adult grasshopper of the common rangeland species is 50 mg. Therefore, the number of grasshoppers ranged from about 120 to 280 individuals per sample.

Analytical Methodology

Chopped grasshoppers (5.0 g) were placed in a centrifuge bottle and extracted three times with 70 ml of methylene chloride by blending in a tissuemizer for 30 seconds, centrifuging and then decanting. The three extracts were combined and concentrated with a rotary evaporator to 10 ml. The concentrated extract was analyzed for malathion and malaoxon, or acephate and methamididophos on a Hewlett Packard 5840 gas chromatograph equipped with a flame photometric detector in the phosphorus mode. Operating conditions for malathion and malaoxon were: flow rate—60 ml/min nitrogen; 5% OV-101 column; oven 205°C; detector 225°C; detector flow rates—150 ml/min hydrogen, 50 ml/min air, and 20 ml/min oxygen. For acephate and methamidophos, operating conditions were: flow rate—30 ml/min nitrogen; 1% Reoplex 400 column; detector 225°C; detector flow rates—150 ml/min hydrogen, 50 ml/min air, and 20 ml/min oxygen; temperature programmed at 20°C/min from 130°C to 190°C. The lower limits of reportable residues were: malathion, malaoxon, and acephate—0.5 ppm; methamidophos—0.1 ppm. Average recoveries from spiked grasshoppers were: malathion—97%, malaoxon—99%, acephate—106% and methamidophos—100%. Reported residues were not corrected for recovery.

RESULTS AND DISCUSSION

Malathion residues ranged from 1.3 to 2.8 ppm in insects collected

TABLE I

Malathion residues (ppm wet weight) in grasshoppers collected from malathion-sprayed rangeland

Time of collection (hours post-spray)	Insect condition		
	Healthy	Sick	Dead
30	2.4	1.3	2.8
	2.2	1.3	2.8
		2.2	2.8
		1.4	2.6
mean	2.3	1.6	2.8
54		0.8	2.8

30 hours post-spray (Table I). The 3 subjective categories of insect status (healthy, sick, and dead) seemed to be related to measured residues with little overlap of residues between categories. The residue levels were different between categories (ANOVA, $P < 0.05$), but the limited number of samples dictates caution in interpreting the results. Apparently healthy grasshoppers had intermediate residues whereas sick grasshoppers had the lowest levels. Whether this reflects metabolic detoxification of the insecticide and impending recovery of these sick grasshoppers is unknown. The 2 samples collected 54 h postspray contained residues similar to the 30-h samples with somewhat lower residues in the sample of sick insects. No malaoxon was detected in any sample. These residues are somewhat higher than the maximum of 0.37 ppm reported in dead bees from a spray area (Levin *et al.*, 1968) and they exceed the average diazinon residues found in insects collected in sprayed tobacco fields (Stromborg *et al.*, 1982).

Grasshoppers collected in the acephate plots very shortly after spraying (4 h) had higher insecticide levels than any of the malathion-treated grasshoppers (Table II). Sick and dead insects had higher residue levels than healthy insects. By 53 h postspray, insecticide levels had decreased to approximately the amounts in the malathion insects. Dead and sick grasshoppers still had higher residues than healthy insects at this time. The major difference between the acephate and malathion samples was the presence of measurable quantities of metabolites in the former. The toxic metabolite methamidophos was detected in all 4 acephate samples in amounts ranging from 32 to 49% of the amount of parent compound. In light of the much greater vertebrate toxicity of methamidophos (Zinkl *et al.*, 1981), this metabolic transformation of acephate represents a significant increase in hazard to animals secondarily exposed on an area treated with acephate.

TABLE II
Acephate and methamidophos residues (ppm wet weight) in grasshoppers collected from acephate-sprayed rangeland

Hours post-spray	Acephate		Methamidophos		Methamidophos/Acephate	
	Healthy	Sick[a]	Healthy	Sick	Healthy	Sick
4	8.2	9.4	2.6	4.6	0.32	0.49
53	1.5	2.2	0.5	0.7	0.33	0.33

[a] Includes fresh dead grasshoppers.

Residues are reported on a wet weight basis (Tables I and II). Six samples of grasshoppers averaged 64% (S.D. 3%) moisture, whereas the moisture content of laboratory diets is normally about 10% (J. Serafin, personal communication). Assuming that birds would eat an equivalent amount of each of these foods on a dry weight basis, the reported residues must be multiplied by a factor of 2.5 for comparability between field and laboratory diets. At 30 h after malathion spray, grasshoppers had malathion residues corresponding to 4.0 to 7.0 ppm on this basis. Corresponding acephate residues ranged from 3.8 to 23.5 ppm whereas methamidophos ranged from 1.3 to 11.5 ppm.

Although these data are limited, they provide preliminary information on the interpretation of laboratory toxicity data in relation to effects observed in the field. Residues of malathion in grasshoppers were far below the dietary levels required to produce effects on birds in the laboratory (Hill *et al.*, 1975). This is supportive of the lack of reports of deleterious avian effects associated with malathion. Acephate levels also were lower than those affecting birds in laboratory toxicity tests (Zinkl *et al.*, 1981). However, the methamidophos residues at 4 h may explain the significant cholinesterase inhibition observed in birds collected from areas sprayed with acephate (Zinkl *et al.*, 1979). Birds in the field may also acquire additional pesticide exposure through dermal contact, in drinking water, and by inhalation.

Data of the type reported here are needed to integrate laboratory results and field observations. Use of this procedure does not depend on locating sick or dead birds that may have moved some distance from the site of exposure and may be very difficult to find. We think that these results offer a useful supplementary tool for assessing the avian effects of insecticide use.

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