Fenthion as a secondary poisoning hazard to American kestrels

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ABSTRACT

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FENTHION AS A SECONDARY POISONING HAZARD TO AMERICAN KESTRELS

The potential of fenthion to act as a secondary poisoning hazard to birds of prey was investigated using American kestrels (Falco sparverius) and house sparrows (Passer domesticus) as a representative model of a naturally occurring predator-prey interaction. Kestrels were presented with live sparrows previously exposed to perches containing Rid-A-Bird 1100® solution (Rid-A-Bird, Inc., Muscatine, IA), 11% fenthion active ingredient, under simulated field conditions. All 14 kestrels tested died following ingestion of fenthion-exposed sparrows. Decreased brain cholinesterase activity and residue analyses of kestrel gastro-intestinal samples confirmed secondary fenthion poisoning.

Prey selection trials were conducted in the laboratory to determine the response of kestrels to a mixed flock of contaminated and uncontaminated sparrows. Kestrels captured fenthion-exposed prey significantly more often (12 out of 15 trials) than normal, unexposed prey.

These results suggest that avian predators and scavengers in the wild are at risk from contact with fenthion-exposed prey in areas where Rid-A-Bird perches are in use.

RESUME

LE RISQUE D'EMPOISONNEMENT SECONDAIRE AU FENTHION CHEZ LA CRECERELLE D'AMERIQUE

Le risque d'empoisonnement secondaire au fenthion chez les oiseaux de proie a été étudié en utilisant la crécerelle d'Amérique (Falco sparverius) et le moineau domestique (Passer domesticus) comme modèle d'une relation prédateur-proie existant dans la nature. Des moineaux vivants, préalablement exposés à des perchoirs contenant une solution de Rid-A-Bird 1100® (Rid-A-Bird, Inc., Muscatine, IA), avec 11% de fenthion comme ingrédient actif, furent présentés aux crécerelles dans des conditions simulant une situation réelle sur le terrain. Les 14 crécerelles testées moururent suite à l'ingestion des moineaux ayant été exposés au fenthion. La diminution de l'activité de la cholinestérase du cerveau et l'analyse pour les résidus dans les tissus de crécerelle confirmèrent l'empoisonnement secondaire au fenthion.

La réponse des crécerelles à un groupe mixe de moineaux contaminés et non-contaminés fut testée en laboratoire lors d'une expérience de sélection de proie. Les crécerelles capturèrent les proies ayant été préalablement exposées au fenthion significativement plus souvent (12 essais sur 15) que les proies n'ayant pas été exposées au produit.

Ces résultats suggèrent que les oiseaux prédateurs et charognards sauvages courent le risque d'un empoisonnement secondaire au fenthion dans les zones où les perchoirs Rid-A-Bird sont utilisés.

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PREFACE

Organophosphorus (OP) pesticides have increasingly been developed and substituted for more environmentally persistent organochlorine pesticides. The OP fenthion was introduced in 1957 as an experimental insecticide (Spencer 1982). One of its many uses today is as an avicide to control nuisance birds.

An early field trial in an Oregon orchard demonstrated the efficiency of fenthion to control starlings (Sturnus vulgaris) and also provided some of the first circumstantial evidence that secondary poisoning to avian predators might be occurring (Larsen 1964). Early laboratory studies established that fenthion was 3 times as toxic to American kestrels (Falco sparverius) (and presumably other birds of prey) than to intended target birds (Schafer et al. 1964). This work also implicated fenthion as a possible secondary poisoning hazard. A subsequent report suggested that perch-killed prey might represent an even greater hazard to avian predators due to the presence of external contamination (Schafer and Cunningham 1965). However, following this research work on the possible secondary poisoning hazard of fenthion was essentially discontinued (Otis 1987).

More recently, birds of prey have apparently been secondarily poisoned following consumption of fenthion-contaminated prey from treated perches in nuisance bird control operations (Dominick et al. in prep.). Two additional published incidents involved falconers' birds (Wenneborg 1986; Keltsch-Richter 1989). Local public outcry

appears to have finally persuaded the appropriate agencies in at least one state to scrutinize the further use of fenthion-treated perches for nuisance bird control.

The purpose of this research was to conduct a study which objectively and realistically evaluated the secondary poisoning hazard of fenthion used as a perch toxicant to birds of prey. It should be noted that under field exposure conditions target birds coming into contact with treated perches would be expected to contain at least some surface contamination. Therefore, strictly speaking, the experimental design of this study does not test for true secondary poisoning, but rather incorporates a broader interpretation of secondary toxicity by taking into account predator-prey interactions which may transfer external contamination to the predator, thus contributing to the overall secondary toxicity. This is discussed further in Chapter 1.

This study contributes new information which should prove of key importance in the growing controversy surrounding the use of fenthion as a perch toxicant in terms of initiating the reregistration process. To my knowledge, this is the first study which investigates this hazard using live, perch-exposed prey. It is also the first to incorporate an all-avian model to examine predator-prey interactions where such prey has been exposed to an OP pesticide under controlled conditions. Preliminary results were presented at a State of Illinois public hearing of the Interagency Pesticide Committee on 13 April 1989, attesting to the relevance of this research towards policy-making decisions.

As permitted by the McGill University guidelines concerning thesis preparation, the author has the option of submitting the thesis as an original paper or papers. Chapter 1 of this thesis entitled "Secondary Poisoning Hazard of Fenthion to American Kestrels" will be submitted to the Archives of Environmental Contamination and Toxicology. Chapter 2 entitled "Selective Predation of Fenthion-Exposed Prey By American Kestrels" will be submitted to the Journal of Applied Ecology. Each chapter has been written in the style required by its respective journal. David M. Bird of the Macdonald Raptor Research Centre, Laird Shutt of McGill University, and Pierre Mineau of the National Wildlife Research Centre of the Canadian Wildlife Service will appear as co-authors on both manuscripts for their contributions towards the initial conception of this research and their guidance throughout. Data collection, analysis, and manuscript preparation were completed independently by the senior author.

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CHAPTER 1

SECONDARY POISONING HAZARD OF FENTHION TO AMERICAN KESTRELS

ABSTRACT

SECONDARY POISONING HAZARD OF FENTHION TO AMERICAN KESTRELS

The possibility that fenthion, an organophosphorus pesticide, could represent a secondary poisoning hazard to birds of prey was tested using American kestrels (Falco sparverius) and house sparrows (Passer domesticus) as a representative model of a naturally occurring predator-prey interaction. Twenty kestrels (including 6 controls) were presented with live sparrows exposed previously to perches containing Rid-A-Bird 1100® solution (11% fenthion active ingredient). Eleven kestrels died within twenty-four hours after consuming 1 fenthion-exposed sparrow. Two kestrels died after consumption of a second fenthion-exposed sparrow on day 2, and a final kestrel died after partially consuming a third fenthion-exposed sparrow on day 3. Brain cholinesterase (ChE) activity in kestrels was depressed to levels diagnostic of poisoning by a ChE-inhibiting compound. The majority of fenthion contamination of sparrows was external, with the highest amounts measured on the feet. The detection of fenthion residues in kestrel gastro-intestinal tracts confirmed secondary fenthion poisoning. The results of this study identified fenthion as a secondary poisoning hazard to American kestrels.

INTRODUCTION

Organophosphorus compounds (OPs) trace their origins to World War II when they were heavily synthesized in Germany as potential chemical warfare agents (Murphy 1986). Fenthion (O,O-dimethyl O-[3-methyl-4-(methylthio) phenyl] phosphorothioate) is one of many organophosphorus pesticides in use today, primarily as an insecticide to protect livestock, domestic pets, various crops, and for mosquito control. Fenthion is also used as an avicide to control pest bird species in both urban and agricultural areas. The Rid-A-Bird® (Rid-A-Bird, Inc., Muscatine, IA)[1] system of perches and solution, 11% fenthion active ingredient, is federally registered as a contact avicide in both Canada and the United States. Fenthion, while of low toxicity to mammals (Francis and Barnes 1963), is highly toxic to various species of birds as evidenced by acute oral and dermal toxicity data for many species (Schafer et al. 1969; Schafer et al. 1973; Hudson et al. 1984; Smith 1987).

Species commonly targeted by this product include the rock dove (Columba livia), European starling (Sturnus vulgaris), and house sparrow (Passer domesticus). Fenthion has been shown to be three times more toxic to American kestrels (Falco sparverius) than to intended target species, suggesting a risk of secondary poisoning of avian predators which ingest exposed prey (Schafer et al. 1964; Schafer and Cunningham 1965). To date, only circumstantial evidence exists of this potential threat to wild raptors from perch-exposed prey. One such incident involved a falconer's red-tailed hawk (Buteo

jamaicensis) which consumed fenthion-exposed starlings from a Rid-A-Bird® eradication program and was diagnosed early enough to be saved (Wenneborg 1986). Several other apparent cases of secondary poisoning of raptors by perch-contaminated target species have also been reported (Dominick et al. in prep.).

Incidents of secondary fenthion poisoning occurring to avian species as a result of avicidal spray application and insecticidal use have been much more widely reported in the literature (Larsen 1964; Seabloom et al. 1973; Hanson and Howell 1981; Zinkl et al. 1981; DeWeese et al. 1983; Henny et al. 1987). A few amphibian models have also determined fenthion to have potential as a possible secondary toxicant (Hall and Kolbe 1980; Powell et al. 1982).

Other than Schafer and Cunningham (1965), no studies have investigated the secondary hazard of fenthion to birds of prey stemming from its use as a perch toxicant. Hence, a laboratory study using American kestrels as predators and house sparrows as prey was conducted under simulated field exposure conditions. The advantages of using the American kestrel in laboratory studies as well as its expanding role in toxicological research, including secondary exposure, are well documented (Bird 1982; Wiemeyer and Lincer 1987). House sparrows, being both abundant and a natural prey item of the kestrel (Palmer 1988), as well as a major target of nuisance bird control programs were considered the most suitable choice for the prey component of this model. The main objective of this study was to determine whether the perch exposure of an avian species to fenthion could lead to secondary poisoning in an avian predator preying upon

that species.

The toxicity of fenthion results from its oxidation in the body to several potent secondary metabolites (Francis and Barnes 1963). These metabolites inhibit acetylcholinesterase by blocking the active center to which the neurotransmitter acetylcholine normally binds; the enzyme thereby becomes phosphorylated and inactive. Excess acetylcholine then builds up at nerve synapses causing symptoms which mimic the muscarinic, nicotinic, and central nervous system actions of acetylcholine, thus impairing normal nervous function. Ultimate cause of death resulting from lethal levels of OP exposure is by asphyxia due to respiratory failure. Recovery of enzyme inhibition usually involves spontaneous reversal, or hydrolysis, of the phosphorylated enzyme. This process may be accelerated by the use of certain oxime derivatives such as 2-pyridine aldoxime methiodide (2-PAM). However, with some OPs, new enzyme must be synthesized (Murphy 1986).

Measurements of enzyme levels in brain samples of kestrels are therefore of diagnostic value in determining exposure to and death caused by fenthion poisoning. Ludke et al. (1975) proposed that inhibition of brain cholinesterase (ChE) levels of at least 20% are indicative of exposure to a ChE-inhibiting compound while inhibition of 50% or more is reason to attribute death to exposure. In a suspected OP field poisoning case, residue levels are required in addition to depressed brain ChE activity for a positive diagnosis.

Determination of plasma ChE activity has the advantage that values can be obtained from repeated, non-destructive sampling techniques. Plasma enzyme analysis may also reveal levels of exposure

to fenthion not high enough to depress brain ChE (Smith et al. 1986). Secondary goals of the study, therefore, were to identify ChE levels in brain and plasma samples of study birds and to locate and quantify fenthion residues associated with both predator and prey.

METHODS[2]

House sparrows were captured by mist netting on the Macdonald College campus of McGill University in Ste. Anne de Bellevue, Quebec from April to October 1988. Fledglings were first recorded in the nets 26 May and were retained along with adult birds. Each sparrow was weighed on a Sartorius PT600 digital pan balance and banded with size no. 2 numbered plastic leg bands (National Band and Tag Co., Newport, KY) for later identification. Sparrows were housed in an indoor flight pen (7 x 7 x 2.4 m, L x W x H) with gravel substrate and equipped with 2 screened windows measuring 0.4 x 1.2 m each. Birds were exposed to natural photoperiod and ambient temperature throughout the duration of the study. Natural vegetation along the perimeter of the pen provided perches and shelter. Commercial wild bird seed and water were provided ad libitum in the center of the pen at ground level. General condition and behaviors of captive sparrows were monitored several times daily.

PRELIMINARY RANGE-FINDING CURVE

Sparrows were allowed to acclimate to captivity for a period of at least one week before testing began. Adult sparrows of unknown age and in apparently good health were randomly chosen for use in fenthion trials and reweighed. To determine the effects of Rid-A-Bird perch

solution on adult house sparrows, the birds were put individually in small holding boxes and dermally exposed via a single fenthion-treated perch (Besser and DeCino 1963). Commercial strength Rid-A-Bird solution (Rid-A-Bird 1100®) and all-weather perches, model no. 61-2 were purchased from a local distributor. Perch solution was analyzed prior to testing in order to verify the manufacturer's listed fenthion concentration.

Holding boxes were constructed of Coroplast (Coroplast, Ltd., Granby, QC) stapled onto a wood frame and measured 0.4 x 0.4 x 0.5 m (L x W x H). A sliding front door covered with screen allowed for observation of the bird and provided adequate ventilation. A single wicked-perch, 0.5 m in length, was filled with Rid-A-Bird solution and inserted through either side of the holding box as the only comfortable perching option available to the sparrow. To ensure this, the bottom of the exposure box was modified with a single piece of Coroplast bent in the shape of a 'V' to produce a 65° angle.

The exposure box was placed in a flight pen as described previously and sparrow perching activity was observed through a one-way glass window. Elapsed time on the perch was clocked to the nearest second using a stopwatch. Upon completion of exposure, the sparrow was released into the flight pen. Sparrows were observed closely to record behavior, as well as onset and progression of any symptoms of fenthion exposure and length of survival. Once paralysis set in, time of death was determined and birds were stored frozen for possible future analysis of ChE activity and residue quantification. To determine the range of perch exposure to fenthion which acutely

affected house sparrows, a preliminary range finding trial was performed as described by Nichols and Crabb (1979) using both male and female birds.

FENTHION EXPOSURE-EFFECT CURVE

Based upon preliminary findings, the lower range of lethal dermal exposure was identified for further study. A new group of sparrows were exposed at 6 of the lowest levels of lethal exposure in order to generate a curve from which to choose the level of exposure to be used in the remainder of the study. To eliminate any possible variability in results due to sex, only male sparrows were used in these trials. The average weight of house sparrows used for these trials was 28.1 $\pm$ 1.1 g (n=36). Six sparrows were randomly assigned to identical interior observation pens (2.4 x 1.2 x 2.4 m, L x W x H) and exposed to perches for times ranging from 1 to 6 min at whole minute intervals. Trials were run simultaneously to prevent any possible bias in results due to fluctuation in daily temperature. Pens were well ventilated from both the floor and ceiling. Data from 6 replicates were used to generate the curve (n=36). Those birds which likely contained higher amounts of fenthion residues, due to longer perch exposure times, and which survived the longest period of time in an impaired state were considered to represent the greatest hazard to avian predators.

SECONDARY TOXICITY TRIALS

Adult male kestrels randomly chosen from the Macdonald Raptor Research Centre (MRRC) colony were tested for their natural instinct to kill live prey. The normal diet of kestrels in the colony is day-

old cockerels. Kestrels were housed individually in small holding boxes, given 1 freshly killed, uncontaminated sparrow, and then fasted an additional day. Two live, uncontaminated juvenile sparrows were placed into a flight pen with a kestrel early in the morning of the following day. Useful birds were defined as those kestrels which killed at least one of the house sparrows within the same day. Twenty such birds were marked with colored plastic leg bands for later identification and returned to the colony.

One ml of blood was drawn from each kestrel for analysis of normal plasma ChE levels. Blood samples were collected into heparinized syringes via brachial venipuncture. Whole blood was refrigerated until centrifuged (2000 g x 15 minutes at 4° C). Plasma was drawn off and frozen until analysis.

Kestrels known to kill live prey were randomly assigned to 20 trials (14 exposure trials and 6 controls). As hunger has been shown to influence the predatory behavior of kestrels in the laboratory (Mueller 1973), all kestrels were fasted prior to a trial to increase their motivation to hunt. The average weight of the fasted kestrels was 91.7 $\pm$ 6.5 g (n=20).

Two identical flight pens with vegetative cover were used, one for exposure trials and the other for controls, to prevent possible fenthion contamination. For each trial, 2 male house sparrows were exposed simultaneously and for approximately equal lengths of time to fenthion-treated perches in 2 identical exposure boxes. Average weight of house sparrows used in these trials was 27.6 $\pm$ 1.6 g (n=24). One member of the pair was randomly selected to be placed in a holding

box while the other was placed in a flight pen with a kestrel in an attempt to simulate natural prey capture. Kestrels were provided with a centrally located 1.5 m high T-perch. The birds were observed until either the kestrel made a kill or the house sparrow died. Kestrels were allowed to eat as much of the sparrow as they wanted without interruption. The sparrow in the holding box was sacrificed by cervical dislocation at approximately the same time the kestrel captured the sparrow in the flight pen and frozen as a substitute sample for later analysis of fenthion residues. Sparrow carcasses were weighed upon completion of each trial and specific body parts consumed were noted.

Kestrels were blood sampled (0.5 ml) approximately 1.5 h after they began to consume contaminated prey and then released back into the flight pen for continued observation of possible symptoms of fenthion intoxication and mortality. Whenever possible, an additional blood sample (0.5 ml) was collected from kestrels later in the trial (prior to sacrificing birds in the case of controls). When exact time to death was not known (as in cases where kestrels died overnight) an estimated time was calculated by averaging the last time when the kestrel was observed alive and the time at which it was discovered dead (McIlroy 1984). If kestrels did not die within 24 h after consuming a contaminated sparrow, they were offered 1 fenthion-exposed house sparrow per day until death (Schafer et al. 1964). Kestrel carcasses and plasma samples were stored frozen for later analysis.

Sample analyses included brain and plasma ChE activity of American kestrels, and residue analysis on external carcass and whole

internal carcass samples of house sparrows, on gastro-intestinal (GI) tracts of kestrels and on feet samples of sparrows and kestrels. Due to their small size, sparrow feet were pooled into 4 groups for analysis.

CHEMICAL ANALYSIS

Whole heads, bodies and feet of all birds in the study were stored separately in aluminum foil when frozen to accommodate independent thawing of samples for ChE and residue analyses. All samples were frozen at -20° C. Total cholinesterase activity was determined colorimetrically by the method of Ellman et al. (1961) as modified by Hill and Fleming (1982) and the Toxicology Research Division (Macaulay and Trudeau 1988) of the Canadian Wildlife Service, Hull, Quebec. Acetylthiocholine iodide (ASChI) was used as the substrate and dithiodinitrobenzoic acid (DTNB) as the chromogen (Sigma Chemical Co., St.Louis, MO). Brain samples were frozen for 12 weeks and plasma samples for 13 weeks prior to ChE analysis. Kestrel brain samples were randomized and analyzed the same day, while plasma samples were randomized and run over a 2 day period.

Heads were partially thawed to facilitate removal of the brain, one longitudinal half of which was used for the assay while the other half was refrozen and saved. Brain tissue for the assay was weighed and then homogenized in cold 0.05M phosphate buffer, pH 7.9, at a ratio of 100mg/ml. Plasma samples were thawed and an aliquot taken directly for analysis. All samples were run in duplicate (or triplicate if reproducibility did not fall within 10%). Average values were used in all calculations. Brain and plasma ChE activity are

reported in International Enzyme Units, which is the activity of enzyme required to convert one μmole of substrate per min per g of brain tissue (wet weight) or per L of plasma.

Spectrophotometric measurements of enzymatic reaction rates were performed with a Hewlett-Packard Diode Array Spectrophotometer model 8452A, controlled by an IBM-XT model 286 computer equipped with a Hewlett-Packard Thinkjet printer model 2225A. The cuvette compartment was maintained at 30° C with a Lauda Circulator bath model K-2/R. Rate of color production was measured at 406nm. Precinorm E control sera (Boehringer Mannheim Canada, Dorval, QC) were assayed on a daily basis as a standard reference to verify accuracy and precision between series of assays.

Residue analyses were performed by Novalab, Ltd. (Lachine, QC) according to their Method Development for the Analysis of Fenthion and Other Organophosphorus Pesticides in Avian Tissue contracted in March 1989 by the National Wildlife Research Centre of the Canadian Wildlife Service (unpublished data). Fenthion chemical reference standard was purchased from Supelco Canada, Ltd. (Oakville, ON). Spiking levels of 0.15, 0.25, and 0.56 parts per million (ppm) on 4 replicates of each sample type yielded 94.3% recovery. The lower limit of detection was 0.05 ppm. Samples were frozen for a maximum of 44 weeks before residue quantification. Samples were coded and randomly assayed according to tissue type. Tissue was homogenized and extracted twice using a polytron homogenizer in the presence of anhydrous sodium sulfate and dichloromethane. Combined extracts were filtered, concentrated on a rotary evaporator, taken up in hexane, and

reconcentrated under nitrogen to a final volume of 5ml. The sample extract was further purified by column chromatography using a 15 x 470 mm glass column packed with 20% deactivated Florisil (15cm) and anhydrous sodium sulfate (top 1-2cm). Final eluant was concentrated as described above and brought to the appropriate volume for analysis. Samples were analyzed on a Hewlett-Packard 5890 gas chromatograph equipped with a nitrogen-phosphorus detector. Oven temperature was increased from 180° C to 270° C over the course of the run. Detector and injector temperatures were 300° C and 250° C respectively. Duplicate analyses were performed on 10% of all samples. A general organophosphate and organochlorine pesticide screen was also run on several randomly chosen samples following the Environment Quebec B.E.S.T. Method. This assessed any previous exposure or body burdens of pesticides in the resident test population which may have exerted possible potentiating effects in combination with fenthion.

STATISTICAL ANALYSIS

Linear regression was performed on all combinations of independent and dependent variables in secondary poisoning trials using the regression procedure of SAS (SAS Institute Inc. 1985). All tests were performed at the 0.05 level of significance. A few noticeably different values of plasma ChE activity were identified as extreme values using a test for detection of outliers (Sokal and Rohlf 1981) and were not included in the calculation of the means. Reported values are mean $\pm$ 1 SD.

RESULTS

PRELIMINARY RANGE-FINDING CURVE

Trials consisted of exposure times from 30 sec to 16.5 min with 1 or 2 sparrows exposed at each time period (Fig. 1). In the 30 sec trials 1 sparrow was partially paralyzed in 8.5 h and died approximately 10.5 h following exposure. The other sparrow at this exposure began to show signs of paralysis the second day after exposure, but ultimately recovered. In the 16.5 min trial the partially paralyzed sparrow fell off the perch and died 30 min later. All other sparrows tested in these trials died, presumably of fenthion toxicosis. For the purpose of this study, a lethal exposure was defined as an exposure which caused mortality due to direct effects within a 24 hour time period. A 1 min exposure therefore, was considered the lowest lethal exposure to the test population (see Fig. 1).

FENTHION EXPOSURE-EFFECT CURVE

Average times to onset of paralysis and death of house sparrows are shown in Fig. 2. Onset of paralysis was defined by initial loss of balance followed by an unwillingness to fly or unsteady locomotion on the ground. Differences in perching behavior of sparrows ranged from remaining still on the perch for the duration of the exposure period, to moving back and forth on the perch constantly, wiping their bills on the perch, leaning forward and jumping onto the screened window several times, and hopping from the perch to the bottom of the exposure box and back. Only two or three birds of the 36 total

appeared to exhibit some aversion to contact with charged perches.

Responses of sparrows to contact with fenthion appeared to be a function of time spent on the perch. The variability of the curve (Fig. 2) may be reflective of environmental factors, the small sample size, the inherent genetic diversity in the test population, the difficulty in identifying the exact onset of paralysis, or the wide differences in perching behavior of the sparrows.

Observed symptoms of fenthion intoxication generally progressed from a coughing or sneezing reaction to body tremors, overall weakness, ataxia, loss of balance, partial paralysis (wings), excessive salivation, and, finally, complete paralysis of the legs. Occurrence and severity of symptoms varied between individuals. These observations are in agreement with fenthion intoxication symptoms reported elsewhere for both orally dosed and dermally exposed birds (Besser and DeCino 1963; Hudson et al. 1984).

Any one of these 6 exposure times could be considered environmentally realistic. In the wild, time spent on the perch would depend on whether birds were staging (seconds), resting (minutes), or roosting (hours) (Besser and DeCino 1963). Five min was selected as the length of exposure for sparrows for all subsequent trials as a compromise between extent of exposure and ensuing survival. A greater survival time would allow for target birds to disperse from the area of treatment in a field situation, thereby enlarging the area of risk, while increasing time spent on the treated perch should result in more residues per bird.

SECONDARY TOXICITY TRIALS

Twenty-one of the first 24 kestrels randomly chosen from the colony successfully captured and killed live prey. Of the 14 kestrels used for secondary poisoning trials, 11 died within one day after killing and at least partially consuming 1 sparrow. Time until death for kestrels ranged from 1 to 15.5 h (Table 1A). Brain ChE activity for these birds averaged 81.3% inhibition (Table 2) of control activity. Plasma ChE activity averaged 96.6% inhibition (Table 3) as compared to control activity, giving an indication of how rapidly kestrel enzyme levels were affected. Comparable values of kestrel weights, time to prey capture, amount of prey consumed, and temperature in control trials are shown in Table 1B.

Two kestrels died on day 2 of the trial, between 1 and 14.2 h after consumption of their second fenthion-exposed sparrow (Table 1A). Cholinesterase inhibition determined for samples in these 2 trials averaged 77.9% for brain and 96.6% for plasma (Tables 2 and 3). Finally, 1 kestrel died within 1 hour after killing and consuming only part of a third sparrow on the third day of testing. Brain ChE was inhibited 92% (Table 2), while plasma ChE was inhibited 54% the first day and 96% the second day as compared with controls. Of the 3 kestrels which were serially blood sampled, the above was the only example in which successive samples revealed such a dramatic decrease in plasma enzyme activity. Control values for brain ChE activity of colony kestrels were in close agreement with control values for wild kestrels published by Hill (1988).

Atypical behavior noted in exposed sparrows included jerky and

irregular preening movements, picking at one or both feet, difficulty in perching, indifference to external stimuli, and an unwillingness to fly. Tail flicking and bill wiping behaviors, which were often observed in sparrows in exposure boxes and upon release into the flight pen, became less apparent in exposed birds as trials progressed. Summers-Smith (1963) described these behaviors as indications of fear or nervousness.

The degree of activity of exposed house sparrows varied between individuals. Some were much more active and ventured out to feed, while others remained under the cover of vegetation the majority of the time. At first, most birds flew rapidly about the pen, and next perched high and then low in the brush. As onset of paralysis began birds hopped about on the ground, finally sitting stationary under medium to heavy cover.

The behavior of the kestrels prior to catching prey in all trials was also variable. Some falcons were extremely alert, bobbed their head and tail when perched, and actively searched for prey on the ground and in the vegetation. Others appeared quite passive and indifferent, and seemed content to search for insects near the ground or quietly perch in the brush.

No house sparrow feet were consumed in any of the exposure or control trials, even though in the pretesting stage some kestrels ate one or both feet of their prey. In addition, none of the kestels consumed an entire sparrow. Average amount of tissue consumed in all trials, based on the weight of sparrow carcasses after predation, was 11.7 $\pm$ 5.6 g. Specific body parts consumed included the head

(excluding the beak), the neck, upper abdomen, and the muscles in the wings. Minimal plucking of prey was observed, and what little there was mostly occurred in the area of the neck and under the wings. Caching behavior was commonly observed in kestrels, and one sparrow carcass from exposure trials was not recovered. In trials which lasted 2 days or more, kestrels continued to consume at least some contaminated prey. Both the elapsed time to prey capture and the amount of prey consumed tended to decrease over the trial period (Table 1A).

The amount of prey consumed was positively correlated with time to kestrel death (Table 4). Conversely, kestrel brain ChE activity was negatively correlated with the amount of prey consumed and the elapsed time until prey was killed. Kestrel weight was negatively correlated with both the elapsed time to kestrel death and kestrel brain ChE activity. None of the variables significantly affected kestrel plasma ChE activity.

Kestrels exhibited fenthion intoxication symptoms similar to those observed for house sparrows. Kestrels tended to seek out dense vegetation as paralysis progressed; however several were found lying dead in the open area of the flight pen.

Most fenthion contamination detected in house sparrows was on the feet, with lower concentrations on the plumage and within the carcass (Table 5). Except for one case, residue analysis on kestrel samples detected more fenthion on feet than in GI tracts (Table 6). Detectable amounts of fenthion were found on all kestrels analyzed from secondary poisoning trials. The higher amount of fenthion

residues reported for the GI tract of kestrel 1685 as compared to all other kestrel GI tracts examined may reflect the larger amount of contaminated prey this bird ate in the 3 days of the trial. In addition, one control sample of house sparrow plumage and one control sample of kestrel feet, as well as the pooled control sample of sparrow feet, all revealed traces of fenthion.

The general organochlorine pesticide screen revealed trace concentrations of polychlorinated biphenyls (PCBs) and p,p'-DDE in house sparrow samples well below 1 ppm. Low levels of PCB residues were also detected in one kestrel GI sample. This value may have been due to PCBs present in the sparrow it consumed. The OP screen showed no detectable levels of diazinon, fenitrothion, malathion, or parathion in any of the samples.

DISCUSSION

Schafer et al. (1964) first suggested that external contamination on perch-killed target birds might be a contributing factor in the secondary poisoning of raptors. High amounts of fenthion detected on the plumage and feet of house sparrows in this study lend support to that idea. Thus, kestrels may have ingested unaltered fenthion while plucking the sparrow or may have ingested secondary metabolites during consumption of internal tissue of prey. Residues identified on kestrel feet indicate that dermal absorption of fenthion by the feet of kestrels during capture and consumption of prey via contaminated plumage or foliage may also have contributed to observed toxicity. Contaminated foliage from insecticidal spraying with fenitrothion has

been suggested as an important route of exposure to songbirds (Mineau and Peakall 1987). Finally, a few kestrels were observed standing in and drinking from the water source provided for sparrows and it is possible they may have ingested some fenthion by this route.

The contaminated feet of prey would undoubtedly have been the most obvious route of exposure to fenthion. However, none of the 20 kestrels in the study ingested any house sparrow feet. Kestrels within the MRRC colony often consume the feet of their normal diet of day-old cockerels, and other sources report similar observations for other birds of prey (Schafer and Cunningham 1965; Holler and Schafer 1982). Gizzard contents of birds from suspected poisoning cases in the field offer additional evidence that raptors on occasion may consume entire prey (U.S. Fish and Wildlife Service 1986; Illinois Department of Agriculture 1988), thus exposing themselves to potentially greater amounts of fenthion.

Traditionally, the definition of secondary poisoning dictates exposure to or ingestion of a compound or its metabolites in the postabsorptive tissues of a prey species by a non-target predatory species (Schafer 1984). Primary poisoning, by contrast, refers to exposure to or ingestion of the original formulation of the chemical to target or non-target species (Schafer 1984). Unlike the study of Hill and Mendenhall (1980) using barn owls (Tyto alba) and famphur-dosed Japanese quail (Coturnix coturnix), the distinction was not made here between true primary and secondary poisoning of American kestrels by fenthion-contaminated house sparrows. Fenthion residues obtained in this study on the plumage and feet of the prey species are

evidence that considerable amounts of the original formulation may be present on target birds following perch contact. Since the experimental protocol is designed to represent a realistic predator-prey interaction, it is necessary to consider an even broader definition of secondary poisoning, as did Schafer (1984), which includes dermal contact with or ingestion of parent fenthion by the predator as a result of external contamination of prey.

This study focused on the lower range of lethal fenthion exposure in house sparrows and the secondary effect of these contaminated target birds on American kestrels. It is quite possible, in view of the extremely high reported toxicity of fenthion to kestrels (Schafer et al. 1969), that sublethal levels of fenthion exposure in house sparrows could also lead to mortality in kestrels. Kestrels in this study showed at best, only partial aversion to exposed prey as a continued food source, agreeing with observations by Schafer et al. (1964). Rapid consumption of several sublethally poisoned target birds by a hungry, opportunistic avian predator would therefore be a distinct possibility in the wild (Larsen 1964; Wenneborg 1986). Migrating or breeding wild raptors in poorer physiological condition might be expected to succumb more rapidly after consuming contaminated prey (Grue et al. 1983). If weight is considered a measure of physiological state, the observation that kestrel weight was negatively correlated with the elapsed time to death is in contrast with Grue et al. (1983). The reason for my results is unclear, but it is likely that additional factors in combination with weight may collectively modify the toxic response.

Finally, kestrels were observed to progress through typical symptoms of OP exposure, including paralysis. These results are in contrast with Schafer et al. (1964), who detected no paralysis in kestrels fed orally dosed (fenthion) house sparrows. Since kestrel samples contained up to 19.2 ppm of fenthion (excluding possible external plumage contamination), they could conceivably become prey for another species of avian predator, thus raising the possibility of tertiary poisoning (Henny et al. 1987).

While this study focused on only one model, it is a realistic one in terms of both predator and prey species, use of live prey as opposed to dead or tethered prey, and a simulated natural environment. All three of the species targeted by this product, i.e. the house sparrow, European starling, and rock dove, are common prey of both urban and rural raptors (Palmer 1988). This may have important implications in terms of the peregrine falcon (F. peregrinus) recovery program currently underway in North America (Canadian Wildlife Service 1988) since falcons are released in urban areas where avian pests are plentiful and Rid-A-Bird perches are often installed. However, even in the absence of published data on the sensitivity of other species of raptors to fenthion, it should be recognized that the American kestrel cannot be viewed as "the" representative species for all birds of prey, not even for the endangered congeneric peregrine falcon (Wiemeyer and Lincer 1987). Tucker and Leitzke (1979) sternly caution that phylogenetic similarity does not necessarily imply similar toxicologic response.

This study identified fenthion as a secondary poisoning hazard to

American kestrels following ingestion of perch-exposed house sparrows. An environmentally realistic predation situation was successfully simulated in the laboratory by first identifying the range of exposure to fenthion in house sparrows where it was felt the greatest risk of secondary poisoning to kestrels existed. If selective predation of fenthion-exposed prey is occurring, this risk may be further escalated (Chapter 2). Reported secondary poisoning incidents in the field for several species of raptors and prey serve to reinforce the validity of our findings in the laboratory. In addition, fenthion residue obtained for samples in this study were consistent with values reported in actual field poisoning cases (Dominick et al. in prep.). This strengthens the fact that my predator-prey model can potentially predict realistic field exposure conditions. It is hoped that the results of this model can be used to help assess the risk to other avian predators and scavengers and to prevent the occurrence of further incidents of secondary fenthion poisoning in the field.

FOOTNOTES

1) New address Wilton, IA 52778 U.S.A.

2) Experimental protocol was reviewed by the McGill University Animal Care Committee and complies with the requirements of the Canadian Council on Animal Care for the ethical use of experimental animals.

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Table 1A. Summary of results of secondary poisoning trials of American kestrels with fenthion.

	Time of Death						
	First Day (N=14)			Second Day (N=3)			Third Day (N=1)
	Mean	SD	Range	Mean	SD	Range	
Kestrel Weight[a] (g)	90.2	5.8	82.0-100.9	89.4	9.7	82.9-100.5	93.3
Time to capture (min)	188	166	1-630	128	82	60-220	90
Prey consumed (g)	12.6[b]	5.0	4.8-20.6	6.8	3.1	4.8-10.4	4.3
Low/High temp (°C)	18/24		10.5-34.0	16/21		12.2-25.5	20/22
Time to death (min)	372[c]	300	60-930	468[d]	570	60-855	40

[a]) pre-trial weight
[b]) N=13, one sparrow carcass not recovered
[c]) N=11
[d]) N=2

Table 1B. Summary of variables in control kestrels (N=6) used in secondary fenthion poisoning trials.

	Mean	SD	Range
Kestrel Weight (g)	95.3	7.2	83.9-103.5
Time to capture (min)	154	114	10-300
Prey consumed (g)	15.3[a]	6.7	8.7-22.2
Low/High temp (°C)	16.8/20.3		11.7-23.9

[a]) N=3

Table 2. Brain cholinesterase (ChE) activity (in μmol/min/g) in American kestrels exposed to fenthion-contaminated house sparrows.

Sample	N	Mean	SD	Range	% Inhibition[a]
Controls	6	27.27	4.95	20.80-34.81	
Trials	14	5.02	1.76	2.08-7.57	81.5
1 day	11	5.10	1.73	2.08-7.57	81.3
2 day	2	6.01	0.67	5.53-6.48	77.9
3 day	1	2.18			92.0

[a]) as compared to controls

Table 3. Plasma cholinesterase (ChE) activity (in µmol/min/L) in American kestrels exposed to fenthion-contaminated house sparrows.

Sample	N	Mean	SD	Range	% Inhibition[a]
Pretest	21	1556	337	1045-2215	
Controls					
1st sample	4	1478	236	1264-1782	
2nd sample	2	1412	181	1282-1539	
Trials					
1st sample	9	50.10	3.59	45.4-54.5	96.6
2nd sample	3	46.58	5.55	40.2-50.3	96.7

a) as compared with either 1st or 2nd sample controls

Table 4. Linear regression analysis on variables in secondary poisoning trials of American kestrels exposed to fenthion-contaminated house sparrows.

Variables	R-square	T for Ho: parameter = 0	Prob > |T|
Dependent variable:			
Elapsed time to kestrel death (min)	0.8944		
Independent variables:			
Kestrel weight (g)		-3.409	0.0271*
Amount of prey consumed (g)		3.314	0.0296*
Dependent variable:			
Kestrel brain ChE activity (IU)	0.9226		
Independent variables:			
Kestrel weight (g)		-2.863	0.0458*
Elapsed time to prey capture (min)		-3.219	0.0323*
Amount of prey consumed (g)		-3.225	0.0321*

* significant at the 0.05 level

Table 5. Concentration of fenthion residues (in ppm) in house sparrow samples.

Sample	Plumage and Skin	Internal Carcass	Feet[a]
240*	ND	ND	
245*	ND	ND	
232*	ND	ND	
229*	0.06	ND	
321	488	4.5	
192	315	6.1	
253	88.8	1.1	
308	631	4.0	
Pool 1*			0.55
Pool 2			1152
Pool 3			1057
Pool 4			908

[a]) Calculated from a pool of 6 sparrows
* Denotes a control
ND Not Detected

Table 6. Concentration of fenthion residues (in ppm) in American kestrel samples.

Sample	Gastrointestinal tract	Feet
1222*	ND	0.46
1748*	ND	ND
1727*	ND	ND
1221*	ND	ND
1685[a]	14.3	12.6
1663	1.4	2.4
1603	1.7	19.2
1117	1.6	6.9

[a]) kestrel from the 3 day trial
* Denotes a control
ND Not Detected

Fig. 1. Preliminary range-finding curve for onset of paralysis in house sparrows following exposure to fenthion-treated perches. White squares indicate 1 sparrow, black squares represent the average time to onset of paralysis in 2 sparrows (N=18, 11 males, 7 females).

PRELIMINARY RANGE-FINDING CURVE

FENTHION EXPOSURE IN HOUSE SPARROWS

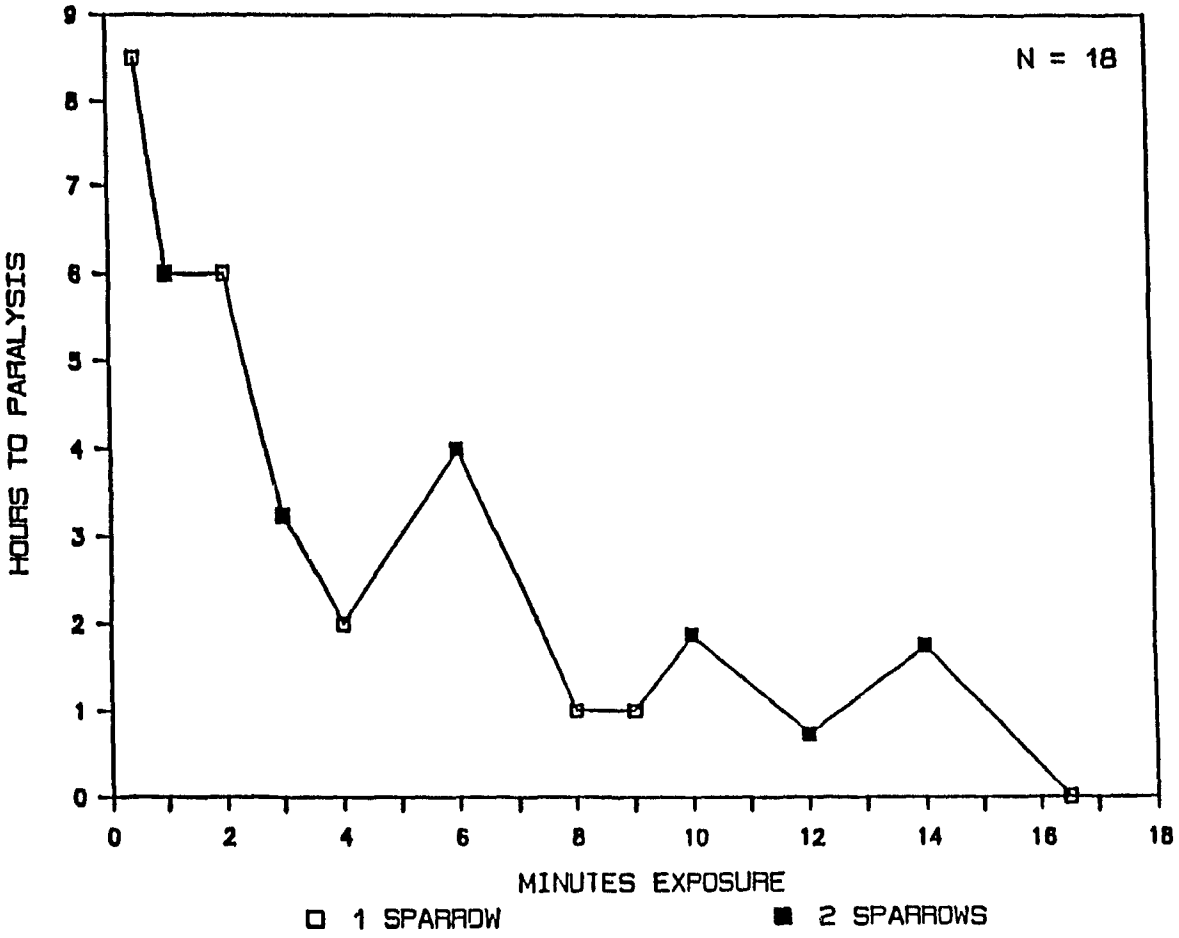
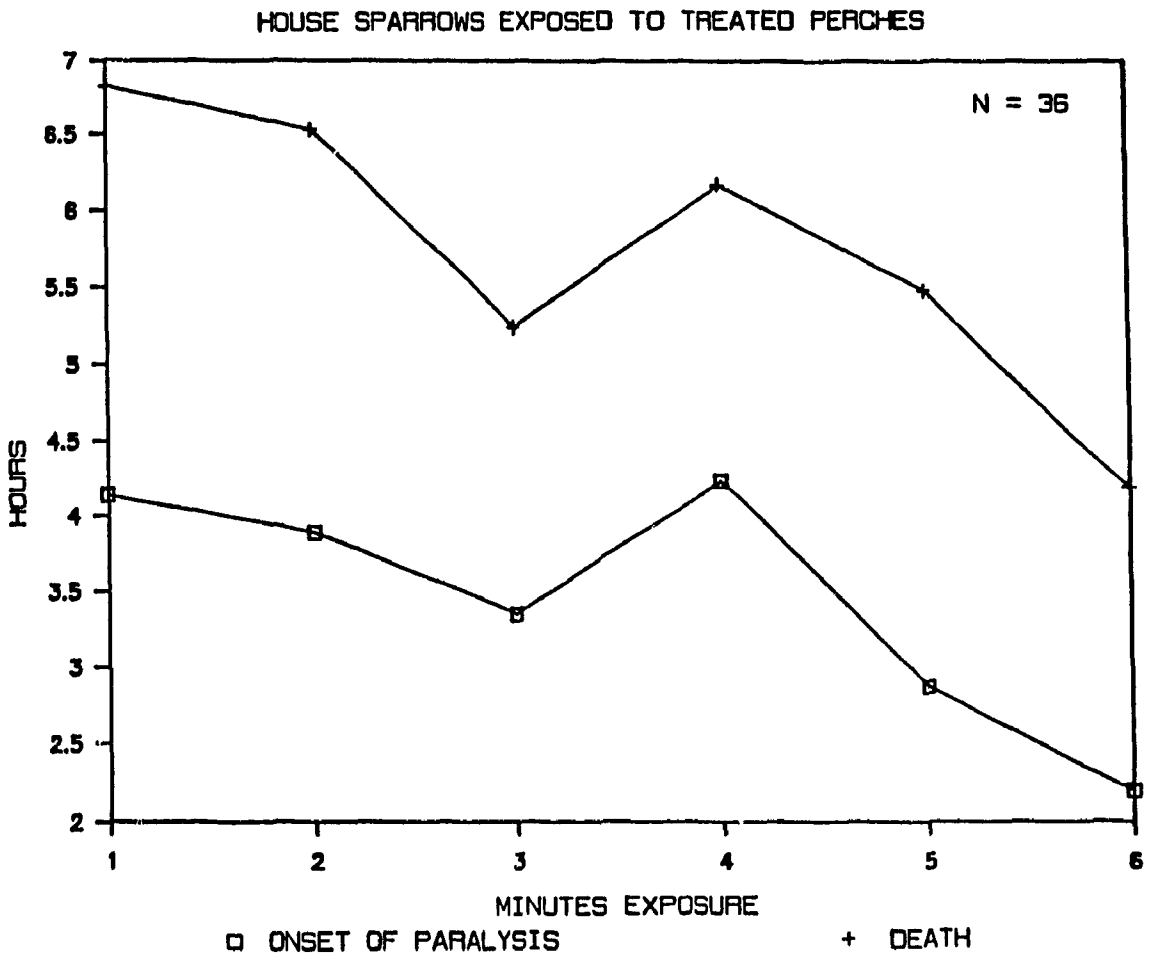


Fig. 2. Fenthion exposure-effect curve for house sparrows following exposure to fenthion-treated perches (N=6 for each exposure).

FENTHION EXPOSURE-EFFECT CURVE



CONNECTING STATEMENT

Chapter 1 identified fenthion as a secondary poisoning hazard to American kestrels following consumption of perch-exposed house sparrows. House sparrows exhibited aberrant behavior as well as typical symptoms of OP intoxication following contact with fenthion-treated perches.

Selective predation of fenthion-exposed prey may amplify the risk of secondary poisoning to avian predators in a field situation. Chapter 2 describes prey selection trials which were conducted to determine the response of kestrels when offered a choice between fenthion-exposed and unexposed house sparrows in a simulated natural setting.

CHAPTER 2

SELECTIVE PREDATION OF FENTHION-EXPOSED PREY BY AMERICAN KESTRELS

SUMMARY

(1) House sparrows (Passer domesticus) were dermally exposed to Rid-A-Bird® perches and solution, 11% fenthion active ingredient, under simulated natural conditions.

(2) Prey choice by American kestrels (Falco sparverius) was identified in the laboratory by offering kestrels a group of live house sparrows, 1 fenthion-exposed and 3 unexposed birds.

(3) 30 predation trials were conducted, 15 in which kestrels captured a sparrow and were considered successful.

(4) Kestrels captured fenthion-exposed prey significantly more often than normal, unexposed prey (Binomial distribution, $P < 0.001$).

(5) Kestrels captured 4 of 5 sparrows obviously affected by fenthion and which were displaying aberrant behaviour at the time of capture.

(6) Selective predation of fenthion-exposed prey by raptors may amplify any existing risk of secondary poisoning in a field situation.

INTRODUCTION

It is a common belief that predators do not remove individuals from prey populations at random (Errington 1963; Slobodkin 1968) but instead exert high selectivity for what Temple (1987) described as "substandard" individuals. Field studies suggest that avian predators capture diseased, injured, or abnormal prey more often than healthy prey (Rudebeck 1950-51; Kenward 1978).

Prey suffering from exposure to organophosphate (OP) pesticides could experience increased susceptibility to predation (Busby et al. 1981; Grue et al. 1983; Galindo et al. 1985). For example, Stehn et al. (1976) described increased consumption of pesticide-killed arthropods by small mammalian predators. Furthermore, in laboratory trials Cooke (1971) demonstrated selective predation of DDT-treated tadpoles by newts.

Avian predators appear to be attracted to distressed and dying insects and invertebrates as a result of insecticide spraying programs (Zinkl et al. 1981). Mendelssohn & Paz (1977) reported an incident of secondary poisoning with the OP insecticide Azodrin® in which approximately 400 birds of prey died as a result of feeding on chemically intoxicated avian and mammalian prey. Other reports also suggest that predatory birds are attracted to contaminated avian prey species in areas treated with OP pesticides (Zinkl et al. 1979).

The effect of OP pesticides on predator-prey relationships in raptors has not been widely studied (Galindo et al. 1985). This study focussed on the predation of fenthion-exposed house sparrows (Passer domesticus) by American kestrels (Falco sparverius). Fenthion is the

active ingredient in Rid-A-Bird 1100® perch solution (Rid-A-Bird, Inc., Muscatine, IA) which is used as a contact avicide to eliminate nuisance birds (Weber 1979). Fenthion has previously been identified as a secondary poisoning hazard to American kestrels (Chapter 1). The goal of this research was to identify prey selection by American kestrels when offered a choice between fenthion-exposed and unexposed house sparrows in a simulated natural setting. Symptoms of fenthion-intoxication and aberrant behaviour in sparrows were also identified in an attempt to determine whether these may influence predator choice. If selective predation on chemically intoxicated prey is occurring, the risk of secondary fenthion poisoning may endanger avian predator or scavenger populations in a field situation.

METHODS

Sparrows were captured, cared for, and dermally exposed to fenthion as described in Chapter 1. The average weight of house sparrows used in these trials was 29.5 $\pm$ 1.8 g (n=76) for males and 28.7 $\pm$ 1.9 g (n=45) for females. For prey selection trials, adult male American kestrels were chosen at random from the Macdonald Raptor Research Centre (MRRC) colony. As hunger has been shown to influence the predatory behaviour of kestrels in the laboratory (Mueller 1973), all kestrels were fasted prior to a trial to increase their motivation to hunt. The average weight of kestrels used in these trials was 93.6 $\pm$ 7.1 g (n=30). Kestrels were not pre-conditioned to capture live prey, nor did they have any prior exposure to either house sparrows or live avian prey. Predation trials were conducted during mornings from

September to November 1988. In each trial, the kestrel was presented with a flock of live house sparrows comprised of 1 fenthion-exposed and 3 unexposed birds. Sex ratios of flocks varied between trials (see Table 1) depending on the availability of the supply of house sparrows captured in mist nets. A new kestrel and a different group of sparrows were used for each trial to prevent the kestrel from forming a specific search image for any particular prey.

Trials were conducted in indoor 7 x 7 x 2.4 m (L x W x H) flight pens with natural vegetation added along the perimeter and a centrally located 1.5 m high T-perch. One sparrow was exposed to a fenthion-treated perch for 5 min, then held 1 h in a holding box on a clean perch to allow the fenthion to begin to take effect (Galindo et al. 1985). The 3 remaining sparrows were similarly exposed on clean perches as controls and held in an identical manner. After 1 h, all 4 sparrows were released into the flight pen followed by the kestrel. Behaviour was observed through one-way glass windows located at both ends of the flight pen. Trials were terminated either when a kestrel captured a sparrow, or at the end of 2 h. Captured prey was immediately taken from the kestrel to prevent further possible fenthion contamination. Kestrels were then placed in a holding box and maintained on their normal diet of day-old cockerels for a 2-day observation period before being released back into the colony.

Data recorded included time taken by the kestrel to capture a sparrow and which sparrow was chosen. Qualitative observations were also made of the behaviour of captured prey, the hunting behaviour of the kestrel, and the flocking activity of house sparrows. The

activity of house sparrows at the time of capture was broken down into 1 of 3 categories following classifications used by Ruggiero & Cheney (1979). House sparrows were recorded as either 1) active: flying around the pen or hopping about in the vegetation, 2) moderately active: staying low in the brush, not moving continuously, or 3) inactive: sitting still in the vegetation or on the ground. Symptoms of fenthion intoxication and any aberrant behaviour in house sparrows were also noted.

The binomial probability distribution was performed according to Steel & Torrie (1980) at the 0.05 level of significance. Reported values are mean $\pm$ 1 SD.

RESULTS

Fifteen of 30 predation trials resulted in a kestrel capturing a sparrow. Kestrels captured the fenthion-exposed sparrow in 12 of these cases, demonstrating a significant differential selection of contaminated sparrows (Binomial distribution, $p < 0.001$).

Kestrels captured 5 active house sparrows, 6 moderately active sparrows, and 4 inactive ones. Only five fenthion-exposed sparrows displayed noticeable symptoms of intoxication; 4 additional sparrows sitting quiet in the vegetation may have experienced different stages of paralysis. The remaining 3 exposed sparrows captured did not exhibit any abnormal behaviour. In only one of the 3 trials where kestrels took an unexposed sparrow did the exposed sparrow in the group exhibit obvious symptoms of fenthion-intoxication. In this case, the bird was found paralyzed under vegetative cover upon

completion of the trial.

In the 15 unsuccessful predation trials, kestrels actively pursued sparrows in 6 trials and showed at least some interest in prey in 4 others. No predatory behaviour was noted for kestrels in the remaining 5 trials. Data from these trials are summarized in Table 1. Fenthion-exposed house sparrows exhibited obvious symptoms of intoxication in only 3 of the unsuccessful predation trials, i.e. within a 2 h period.

Symptoms of fenthion intoxication and aberrant behaviour noted in house sparrows were similar to those observed in secondary poisoning trials in Chapter 1. Behaviour of kestrels was also highly variable. Some kestrels ignored the house sparrows and preened or slept. Other birds oriented toward prey, assumed a forward upright posture, compressed their plumage, and exhibited head bobbing and tail pumping motions, all of which are believed to signal an impending attack on prey (Bildstein & Collopy 1987; Palmer 1988). The kestrels also displayed widely different foraging techniques including perch or still hunting, aerial pursuits, and deliberate searches on the ground in an attempt to flush prey from cover. These behaviours have also been described for wild kestrels (Bildstein & Collopy 1987; Palmer 1988). Although hunting behaviour was often observed immediately after a trial was initiated, kestrels took 1 h on average to successfully capture a sparrow (Table 1).

House sparrows tended to form loose groups. An attack by a kestrel resulted in a temporary scattering of the sparrows. Sparrows were observed to flit around on vegetation, preen, fly between patches

of vegetation, and occasionally venture to the center of the pen to feed. Sparrows apparently recognized kestrels as a threat and often gave repeated warning calls throughout the trial. Flocking house sparrows displayed behaviour much more similar to that observed for birds in an aviary flock than for single sparrows in secondary poisoning trials (Chapter 1).

DISCUSSION

Prey movement is an important stimulus for many predators and increases the probability of initiating an attack (Cushing 1939; Ingles 1940; Ruggiero et al. 1979). Selection of more active prey has also been reported for several raptor species (Kaufman 1974, Snyder 1975, Snyder et al. 1976). Ruggiero & Cheney (1979) suggested that aberrant prey movement in mice might be a more appealing attack stimulus to kestrels than normal movement. Selection of prey based on abnormal behavioural characteristics has been reported by Rudebeck (1950-51). House sparrows which exhibited difficulty in locomotion as a result of fenthion contamination may have presented themselves as conspicuous targets for kestrels in predation trials. In successful predation trials, kestrels captured 4 of 5 sparrows which were obviously affected by fenthion and which were displaying aberrant movement. In the 3 trials where kestrels captured normally behaving, fenthion-exposed sparrows they may have been cueing in on some subtle aspect of prey behaviour which was not apparent to the human observer (Rudebeck 1950-51). In contrast, kestrels captured 4 sparrows which were not moving, suggesting that prey movement was not a necessary

stimulus for an attack under this set of conditions. Galindo et al. (1985) also found that prey movement was not an essential stimulus in releasing the stalking action of cats to methyl parathion-dosed quail.

A second general explanation for predator choice reasons that certain prey are more vulnerable and therefore easier to catch than others (Dekker 1980; Mueller 1987). Anecdotal accounts describe kestrels being attracted to birds caught in traps (Hodgdon 1975; Bird & Greenwood 1981), attacking birds caught in mist nets (Windsor & Emlen 1975), and taking birds directly out of burrow openings or nest boxes (Windsor & Emlen 1975; Steffen 1981). Fenthion-exposed prey may be more susceptible to predation because of reduced stamina. This may then affect avoidance behaviour, i.e. the prey is reluctant to flush and escape is impaired (McEwen & Brown 1966; Tucker & Leitzke 1979). Farr (1977) found that shrimp sublethally exposed to parathion were easy to capture by killifish in part due to decreased physical endurance. In their study of several salmonid species, Post & Leasure (1974) suggested malathion-exposed fish were unable to elude predators in the wild.

As symptoms of fenthion intoxication appeared and intensified in house sparrows, they began to exhibit decreased response to external stimuli. Thus, their antipredator behaviour may have been affected making them easier targets (Cooke 1971). In turn, kestrels may have redirected their efforts towards the fenthion-exposed house sparrows. Since the experimental design did not allow the determination of the kestrel's first choice, but only the first sparrow successfully captured, kestrels could have pursued different sparrows throughout

the trial prior to capturing a bird.

Kestrels appeared to capture house sparrows sitt ng apart from the rest of the flock. This occurred in 8 of the 12 trials in which kestrels captured the fenthion-exposed sparrow. Mueller (1987) commented on a similar tendency in one of his earlier experiments with kestrels and mice. He also noted that kestrels appeared to select the closest mouse, but such was not the case with house sparrows here. In one instance, the kestrel flew across the flight pen into heavy vegetation and captured the fenthion-exposed sparrow, even though another sparrow was on open ground less than 1 m directly in front of the kestrel. The wide range of foraging techniques used by the kestrels indicates that the laboratory setting did not restrict the natural behaviour of kestrels to a degree totally unrepresentative of an actual field situation.

Finally, it is necessary to address the relevance of results obtained in simulated predation trials in this study to actual field situations by considering the feeding habits and hunting behaviour of avian predators in the wild (Mueller 1987). Secondary poisoning hazards from fenthion would be greatest for raptors which feed preferentially on one of the nuisance species targeted by this type of contact avicide, i.e. rock dove (Columba livia), European starling (Sturnus vulgaris), or house sparrow. These include the versatile and opportunistic American kestrel (Bildstein & Collopy 1987; Mueller 1987) and several Buteo and Accipiter species (Storer 1966; Palmer 1988). Avian scavengers or carrion feeders may also be at risk from pesticide-contaminated bird carcasses (Balcomb 1986).

Several species of falcons such as the kestrel, peregrine falcon (F. peregrinus), and merlin (F. columbarius) are becoming increasingly common in urban areas. Oliphant (1974) reported that approximately 90% of the prey species of urban merlins in Saskatoon consisted of house sparrows. These same falcons also preyed upon rock doves (Warkentin & Oliphant 1988). As all three nuisance bird species are commonly taken by endangered peregrine falcons (Canadian Wildlife Service 1988; Palmer 1988), consumption of fenthion-contaminated birds could have severe implications for peregrine falcon recovery programs (Cade et al. 1988). Falcons are released in urban areas where Rid-A-Bird perches are often installed. The risk of secondary fenthion poisoning extends to raptors in rural environments as well since treated perches are often used on farms and at power plants located in rural areas (Weber 1979; Wenneborg 1986).

Avian predators are commonly attracted to large concentrations of prey, e.g. birds on wintering grounds (Blus et al. 1983) or quelea colonies (Manikowski 1988; Bruggers et al. 1989). In addition, avian predators are known to specialize for extended periods of time on unusual prey densities (Oliphant 1974; Oliphant & McTaggart 1977) or even switch prey to take advantage of a situation of temporary prey abundance (Collopy 1973; Balgooyen 1976; Mueller 1987). Predators in these situations would especially be at risk if they had unlimited access to a contaminated prey base (Wenneborg 1986).

This study identified selective predation of fenthion-exposed house sparrows by American kestrels in a laboratory setting. Several apparent field reports of secondary fenthion poisoning (see Chapter 1)

may be due to selective predation, and many more may go unreported because of the small number of carcasses submitted for laboratory analysis (Woolf & Gremillion-Smith 1987). Consequently, these results suggest a risk for birds of prey in areas where fenthion-treated perches are installed for nuisance bird control. The possibility of selective predation on fenthion exposed prey in the field necessitates a thorough re-evaluation of the further use of fenthion-treated perches if raptor mortalities are to be reduced.

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Table 1. Comparison of variables between successful and unsuccessful predation trials of American kestrels on fenthion-exposed and unexposed house sparrows. Values given are the mean ± 1 SD, with the range given in parentheses.

	A) Successful Trials (n=15)[a]	B) Unsuccessful Trials (n=15)
Time to prey capture (min)	63.3 ± 31.7 (15-120)	
Weight of kestrel (g)	91.0 ± 5.8 (83.3-105.7)	96.1 ± 7.5 (81.8-109)
Weight of sparrow (g)[b]	29.5 ± 1.7 (24.6-34.2)	29.1 ± 2.0 (24.6-32.8)
Temperature (°C)	10.3 ± 3.9 (6.1-17.8)	9.7 ± 5.0 (5.0-22.8)
Ratio of sparrows (M:F) in the flock	4:0 (3)[c] 3:1 (4) 2:2 (7) 1:3 (1)	4:0 (1) 3:1 (6) 2:2 (6) 1:3 (2)

[a]) kestrel captured prey within 2 hours
[b]) n = 60
[c]) number of trials in parentheses

GENERAL CONCLUSIONS

Fenthion was identified as a secondary poisoning hazard to American kestrels following ingestion of perch-exposed house sparrows. Brain ChE activity of kestrels was inhibited to levels diagnostic of poisoning by a ChE-inhibiting compound. Plasma ChE values agreed with results obtained from brain ChE analysis and gave an indication of how rapidly kestrel enzyme levels were affected. Detectable amounts of fenthion were found on all samples analyzed from secondary poisoning trials. Residue analyses identified the majority of fenthion contamination as external for both sparrows and kestrels.

Kestrels exhibited significant differential selection of fenthion-exposed prey when offered the choice between 1 exposed sparrow and 3 normal, unexposed sparrows. Kestrels displayed a similar range of foraging techniques as have been described for wild kestrels, indicating that the artificial setting of the laboratory did not restrict their natural behaviour to a degree where results were unrepresentative of an actual field situation. Aberrant behaviour displayed by fenthion-exposed sparrows may have been more of an appealing attack stimulus to kestrels than the normal behaviour of unexposed sparrows.

Laboratory results obtained here have direct application to potential incidents of secondary fenthion poisoning in the field. The secondary poisoning hazard from fenthion would be greatest for raptor species which feed preferentially on one of the nuisance species

targeted by treated perches. Avian scavengers and carrion feeders may also be at risk in a field situation. In 2 and 3 day secondary poisoning trials, kestrels showed, at best, only partial aversion to fenthion-exposed prey as a continued food source. Rapid consumption of several fenthion-exposed prey by a hungry, opportunistic raptor may therefore be a distinct possibility in a wild situation. No house sparrow feet were consumed in any exposure trials. Contaminated feet would undoubtedly be the most obvious route of fenthion exposure from perch-exposed prey. Gizzard contents of birds from actual field poisoning cases of fenthion offer evidence that raptors may consume entire prey, thus exposing themselves to greater amounts of contamination. Kestrels experienced symptoms of fenthion-intoxication such as instability, indifference to external stimuli, and paralysis thus raising the possibility of tertiary fenthion poisoning in the field.

The results of this avian model for secondary fenthion toxicity suggest that birds of prey are at risk in areas where fenthion-treated perches are installed for nuisance bird control. Additionally, the possibility of selective predation on fenthion-exposed prey in the field should signal the need for a thorough re-evaluation of the use of fenthion-treated perches as a tool for nuisance bird control.