

The Impact of Fenthion on Birds of Prey

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Fenthion is an organophosphate insecticide (OP) registered worldwide. It is widely used in mosquito control (Grue 1982) and is frequently applied to livestock (Smith 1987), domestic pets and various crops. It is also used as a contact avicide to control birds (Thomsett 1987; Bruggers *et al.* 1989; Wiemeyer 1991). Fenthion has been frequently involved in wildlife mortality including several thousand passerines (Seabloom *et al.* 1973), Black-billed Magpies, *Pica pica*, (Hanson & Howell 1981), and raptors (Henny *et al.* 1987; Wiemeyer 1991). Several cases of secondary poisoning of raptors by contaminated target bird species have been reported with fenthion used as an avicide and as an insecticide (Seabloom *et al.* 1973; Hanson & Howell 1981; Zinkl *et al.* 1981; DeWeese *et al.* 1983; Wenneborg 1986; Thomsett 1987; Bruggers *et al.* 1989; Dominick *et al.* 1989).

The toxicity of fenthion results from its oxidation in the body to several potent secondary metabolites (Francis & Barnes 1963). Fenthion inhibits acetylcholinesterase (AChE), the enzyme that breaks down the neurotransmitter acetylcholine, causing its accumulation at nerve synapses and consequent disruption of nerve function (O'Brien 1967:55).

In American Kestrels, *Falco sparverius* and fenthion-exposed House Sparrows, *Passer domesticus*, fenthion intoxication was characterized by coughing and sneezing, body tremors, overall weakness, ataxia, loss of balance, partial paralysis of the wings, excessive salivation, and finally complete paralysis of the legs (Hunt *et al.* 1991; Hunt *et al.* in press). These observations are in agreement with fenthion intoxication symptoms reported elsewhere for both orally dosed and dermally exposed birds (Besser & DeCino 1963; Hudson *et al.* 1984).

Birds are highly sensitive to fenthion as indicated by low oral LD₅₀ values ranging from 2.5 to 25.9 mg/kg (Tucker & Crabtree 1970; Schafer *et al.* 1983; Smith 1987) and low LC₅₀ values ranging from 30 to 231 ppm in the diet (Hill *et al.* 1975). Dietary toxicity of fenthion appears to vary between families and species of birds (Hill 1972; Hill *et al.* 1975). Among species studied, raptors are more sensitive to the toxic effects of this OP than some of the potential target species. For example, the acute oral LD₅₀ values of fenthion were 1.0 mg/kg of body weight for the American Kestrel and 5.6 mg/kg of body weight for the House Sparrow, a target bird species (Schafer *et al.* 1964; Schafer *et al.* 1969). American Kestrels were also found to be susceptible to other OPs and carbamates relative to other avian species. Similarly, Eastern Screech-owls, *Otus asio*, were found to be sensitive to fenthion (LD₅₀ value=4.6 mg/kg) and other ChE inhibitors (Wiemeyer & Sparling 1991).

Secondary poisoning has not generally been considered to be an important route of exposure to OPs. However, the ingestion of OP poisoned vertebrates (birds and small mammals) has resulted in mortality in raptors under field conditions (Mendelsshon & Paz 1977; Wilson *et al.* 1991).

Traditionally, an inhibition of total brain ChE level of 20% has been determined to be diagnostic of exposure. Inhibition of 50% is associated with mortality as well as with a number of severe sublethal effects (Grue *et al.* 1991). Prior to our work (Hunt *et al.* 1991; Hunt *et al.* in press), no one had systematically investigated the secondary poisoning hazard of fenthion to birds of prey under realistic conditions when used as an avicide dispensed through a toxic perch system.

Toxic hazard of fenthion to raptors in field situations has been demonstrated by observations of sick or dead birds in association with fenthion treatments (Beard 1969; Elder & Henderson 1969; Seabloom *et al.* 1973; Stickel 1973; Zinkl *et al.* 1981). External contamination on target birds is a contributing factor in the secondary poisoning of raptors (Schafer *et al.* 1964; Hunt *et al.* 1991; Hunt *et al.* in press). Our studies have shown that American Kestrels have no aversion to eating, on a daily basis, House Sparrows that had come into contact with the fenthion perches (Hunt *et al.* 1991; Hunt *et al.* in press). Moreover, kestrels captured fenthion-exposed sparrows 12 times more often than normal, unexposed sparrows in the same flocks. Fenthion-exposed sparrows displayed aberrant behavior at the time of capture (Hunt *et al.* in press). Similarly, following spraying of the avicide Queletox® (60% fenthion) in Kenya, the flapping moribund Red-billed Quelea, *Quelea quelea*, were very conspicuous to passing raptors and were quickly eaten (Thomsett 1987).

Under aviary conditions where kestrels were allowed to hunt, most (11/14) died within one day after killing and at least partially consuming 1 House Sparrow exposed to fenthion. The inhibition of ChE activity in the brain of these birds was 81% and 97% in the plasma as compared to controls (Hunt *et al.* 1991). Another study has shown that the average depression of brain ChE activity for birds that died 8 hours after dosing with fenthion was 88.0% for the American Kestrels and 92.0% for Eastern Screech-owls (Wiemeyer & Sparling 1991). The capture behavior of American Kestrels treated with 50 mg/kg of acephate (corresponding inhibition of brain ChE activity = 25% and ChE plasma activity = 40%) was not affected when using an artificial moving prey model (Rudolph *et al.* 1984). In our laboratory, we are conducting a series of experiments on American Kestrels treated with another ChE inhibitor, Physostigmine. Doses of this carbamate ChE inhibitor were selected to span 25-50% inhibition of brain ChE activity and kestrels had to catch a living wild mouse, *Mus musculus*. These results will increase our knowledge on the general effect of sublethal doses of ChE inhibitors on the hunting behavior of the kestrel, a behavior that is necessary for survival of adults and their offspring.

The significance of OP-related mortality to wildlife populations is not known. However, the mortality of about 400 raptors (species with reproductive potentials lower than those of most birds) following the misuse of the OP monocrotophos on a 8 km² area (Mendelsshon & Paz 1977) indicates that the potential for population effects exists (Grue *et al.* 1983). Moreover, sublethal exposures to fenthion and other ChE inhibitors are suspected to have detrimental effects on survival and reproduction (Grue *et al.* 1991; Patnode & White 1991).

Sublethal exposure of birds of prey to OPs may result in loss of body weight due to pesticide-induced anorexia as observed in other bird species (Costa & Murphy 1982; Grue 1982). Lowered food consumption may reduce egg production (Stromborg 1977; 1981) in raptors and this effect on reproduction may be more severe with the alteration of hormone concentrations due to OP exposure (Rattner *et al.* 1982a,b). Growth of nestlings and particularly growth of altricial nestlings may be depressed following OP exposure resulting in lower weights at fledging (Pearce & Busby 1980; Powell & Gray 1980). Lower body weight at fledging is associated with decrease in post-fledging survival in non-treated birds (Perrins 1965; Smith 1967; Loman 1977). Also survival of adult raptors and that of their offspring depends on their ability to hunt. Since OPs can reduce visual acuity (Plestina & Pivkovic-Plestina 1978), vigilance (Levin & Rodnitzky 1976), and food-seeking behavior (Adams 1977), raptors may become less efficient in providing food to their young, their mate, or themselves.

Where fenthion is used as a contact avicide, secondary poisoning hazard is expected to be greater for raptors feeding on one of the nuisance species targeted by fenthion (i.e., Rock Dove (*Columba livia*), European Starling, *Sturnus vulgaris*, House Sparrow, and Red-billed Quelea), and on other nuisance species as the Red-winged Blackbird, *Agelaius phoeniceus*, the Brown-headed Cowbird, *Molothrus ater*, and the Common Grackle, *Quiscalus quiscula*, which may have access to fenthion perches in agricultural areas in North America. In urban areas, non-target species such as the Chimney Swift, *Chaetura pelagica*, and the Cliff Swallow, *Hirundo pyrrhonota*, may also be contaminated with fenthion since they are nesting on structures where fenthion perches are

used (chimneys and bridges respectively). Raptors susceptible to fenthion poisoning include the opportunistic American Kestrel (Bildstein & Collopy 1987; Mueller 1987; Hunt *et al.* in press), and several Buteo and Accipiter species (Storer 1966; Palmer 1988). Moreover, several species of falcons (American Kestrel, Peregrine Falcon, *F. peregrinus*, and Merlin, *F. columbarius*, are becoming increasingly common in urban areas. Fenthion has been suspected as a cause of mortality in Saskatoon Merlins (L. Oliphant, pers. comm.) and approximately 90% of the prey species of urban Merlins in Saskatoon consisted of House Sparrows (Oliphant 1974). Rock Doves are captured by Merlins only on few occasions (Warkentin & Oliphant 1988). These species along with the European Starling are prey taken by Peregrine Falcons (Can. Wildl. Serv. 1988; Palmer 1988). Consumption of fenthion-contaminated birds could have severe implications for recovery programs (Cade *et al.* 1988). Falcons are released in urban areas where fenthion is used as a perch avicide and the risk of secondary poisoning extends to rural areas where they are also used (Weber 1979; Wenneborg 1986).

Fenthion used as an insecticide in terrestrial and aquatic environment is relatively non persistent but it can be acutely toxic to birds (U.S. Environmental Protection Agency 1988). When used as an avicide, fenthion is a clear hazard to nontargeted birds of prey.

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