POTENTIAL PRIMARY AND SECONDARY HAZARDS OF AVICIDES

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ABSTRACT: There are six chemicals or groups of chemicals that are currently registered as avicides that can be used in some or all of the U.S. Most of these chemicals, because of their diverse chemical composition and innate toxicological effects, present somewhat different primary and secondary hazards to avian and mammalian predators and scavengers. Of the chemicals reviewed, all appear to present some degree of primary hazard to non-target birds and mammals; however, PA-14, the Starlicide family of chemicals and fenthion appear to be the least hazardous when used according to use directions. 4-Aminopyridine, endrin and strychnine, because of their high acute toxicity and lack of selectivity, must be considered potentially more hazardous. With respect to secondary hazards, the ranking of chemicals changes considerably and only PA-14 appears to present a negligible hazard. The Starlicide family of chemicals presents negligible hazards to most animal species under currently registered uses, but may be potentially hazardous to cats and owls under specific use conditions. Two chemicals, 4-aminopyridine and strychnine, are potentially more hazardous to predatory and scavenger animals due to their highly toxic nature and rapid lethal effects in target species, leaving unassimilated chemical in the gastrointestinal tract. The remaining chemicals, endrin and fenthion, have been shown to possess the potential for more significant secondary poisoning; however, because of restrictive uses, most of the potential hazards have been avoided in operational use.

INTRODUCTION

Lethal bird control chemicals, or avicides, represent many diverse basic chemical structures with different physical/chemical properties. Therefore, almost every chemical may present unique and specific hazards to some target and non-target organisms. For the purposes of this paper I have chosen the narrowest definition of avicide, that is, those materials that act through, or result in, direct mortality of the target species. To further restrict this topic, I am going to limit my discussion to chemicals that are currently registered in the U.S. (by State or Federal regulations) (Schafer 1979, Matheny 1980).

Before I go any further, I feel that it is necessary for me to clarify my interpretation of what primary and secondary hazards are. Technically, primary hazards refer to the potential life-threatening results to target and non-target organisms that result from the direct consumption of, or exposure to, chemicals in their originally applied form. Secondary hazards refer to the potential life-threatening results to non-target predatory or scavenger organisms, resulting from exposure to, or consumption of, prey tissue containing a chemical in its original or in an altered form. I realize that, although these definitions may be technically correct, they may not be acceptable to a large number of individuals due to the great difficulty in separating primary and secondary hazards in predator-prey relationships. Therefore, throughout this paper I am going to use a much broader definition of secondary hazards that also includes some modes of primary exposure. This definition includes not only tissue residues but also all chemical contamination contained in, or on, the target species regardless of location or form at the time they are consumed.

For the purposes of this paper, the commonly used and registered avicides have been classified by the following chemical or activity groups:

1. Alkaloids
2. Anilines
3. Chlorinated hydrocarbons
4. Detergents
5. Organophosphates
6. Pyridines

Under each category, the following items accompanied by the individual avicide name will be briefly discussed:

1. Current registered uses and restrictions.
2. Brief toxicology summary.
3. Primary hazard potential of current uses.
4. Secondary hazard potential of current uses.

ALKALOIDS

Strychnine Alkaloid and Strychnine Sulfate

Strychnine alkaloid (strychnidin-10-one) and strychnine sulfate are currently registered in California to control blackbirds, cowbirds, crowned sparrows, horned larks, house finches and meadow larks in orchards and vineyards, and horned larks in agricultural crops. State registrations for magpie control also exist in Nevada and Wyoming (Anonymous 1983). Federal registrations for the use of strychnine are currently in effect for the control of pigeons and sparrows on nonagricultural sites. All the avicide registrations, except those for magpies, use strychnine-treated grain baits (0.25 to 1.0%) exposed in troughs or in furrows between crop plantings to limit the exposure to non-target species. Magpie baits are made of suet. Because of the recent conclusion of the RPAR (Rebuttable Presumption Against Registration) action brought against strychnine by the EPA (U.S. Environmental Protection Agency), restrictions with respect to bait application and bird carcass disposal have been

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tightened for all uses (Anonymous 1983). Restrictions on the use of strychnine for pigeon control have also been tightened to preclude its use in Puerto Rico, within five miles of Peregrine falcon habitats, and at locations where migratory falcons are present. These restrictions are designed to minimize primary and secondary hazards to non-target species, and to supplement existing time-of-year (generally fall-winter) use restrictions.

Strychnine alkaloid and strychnine sulfate are potent convulsants and rapidly absorbed from the gastrointestinal (GI) tract. Convulsive seizures commonly appear within 5 to 30 minutes after ingestion, and the usual cause of death is respiratory failure (Savarie 1981). Strychnine is extremely toxic to most bird species with acute oral LD50s ranging from 1.0 to 20 mg/kg (Schafer 1981); however, gallinaceous birds appear to be somewhat more resistant than other birds to acute intoxication (Ochs 1976, Rudd and Genelly 1956). Strychnine is rapidly detoxified and excreted. However, because of its rapid action, large amounts of unabsorbed strychnine may remain in the GI tract of target species at death and therefore may pose some hazard to scavenger and predatory animals (Savarie 1981).

Primary hazards to non-target birds and mammals are obviously present with the use of strychnine because of its high acute toxicity. These hazards are limited in nonagricultural areas to other bird and mammal species present in and around structures and in agricultural crops to trough-feeding and ground-feeding species. Doves and small rodents are probably the most likely animals to be killed in agricultural areas (Rudd and Genelly 1956, Palmer 1970), while pheasants and quail are the least likely, although there is a lack of reported kills of any species following the use of strychnine as an avicide in the literature (Koehler 1962, Lyndall 1962, Palmer 1970, Clark 1976). In nonagricultural areas, house sparrows or other graniverous birds and domestic animals associated with human habitation may also be poisoned, but again there is a lack of any reported kills in the literature (Watkins 1976). Because of limitations in the time of the year that strychnine products can be used under many labels, it is likely that non-migratory or local birds will be impacted more than migrating birds.

Significant secondary hazards of strychnine to scavenger and predatory animals have been assumed and inferred, but little data are available to substantiate or repudiate these assumptions (Rudd and Genelly 1956, Schitoskey 1975). The secondary hazards that have occasionally been reported following the ingestion of strychnine poisoned animals are most likely the result of the consumption of GI contents containing a large amount of unassimilated strychnine (Ochs 1976). Similar hazards can also be expected from any rapid-acting acute toxicant that is not rapidly hydrolyzed or metabolized.

ANILINES

Starlicide, CAT and CPT

Starlicide (DRC-1339, 3-chloro-4-methylbenzenamine HCl) and its closely related family members CPT (3-chloro-4-methylbenzenamine) and CAT (N-(3-chloro-4-methylphenyl)acetamide) were specifically developed by the U.S. Fish and Wildlife Service (FWS) for use as avicides. It is currently Federally registered in the U.S. for controlling starling populations at animal feedlots, gulls on breeding islands used by terns or other threatened bird species, and for experimental use on other species of pest birds. Starlicide is also State registered for use in controlling starlings at mink farms in Wisconsin and for controlling blackbirds, crows and pigeons in California. CAT and CPT have been investigated as potential starling/blackbird roost toxicants in the U.S. (Peoples et al. 1976, Lefebvre et al. 1981) and CPT is being used to experimentally control starling roosts in France. Starlicide formulations are directly available to farmers with minimal use restrictions but the remaining registered products are tightly controlled to prevent misuse. In the U.S., CPT and CAT are not available for operational bird control use.

Starlicide and related compounds are unique because of their high toxicity to most species of pest birds (oral LD50s of 1.0 to 10 mg/kg; sensitive), and low-to-moderate toxicity to most mammals and predatory birds (oral LD50s of 250 to 1000 mg/kg; non-sensitive) (Lefebvre et al. 1981, Schafer 1981). Although the mode of action of these compounds is still not well understood, they apparently cause death in sensitive species by nephrotoxicity (DeCino et al. 1966, Palmore 1978). In non-sensitive species, central nervous system (CNS) depression and the attendant cardiac or respiratory arrest is the cause of death (Felsenstein et al. 1974). Attempts have been made to relate the selectivity of these compounds to specific enzyme systems, but, only insufficient and conflicting data are presently available to verify the relationships (Apostolou 1969, Mull and Giri 1972).

The Starlicide family of compounds are also potent chronic toxicants in sensitive species but not in non-sensitive species (Schafer et al. 1977), even though they are apparently rapidly metabolized and excreted in both groups of animals and are not accumulated in the body (Cunningham et al. 1981). The toxicity of this family of compounds appears to be directly related to irreversible necrosis of the kidney in sensitive species, and death occurs after almost all of the assimilated chemical has left the body (Cunningham et al. 1981). Although reproductive effects are not well documented in sensitive species, they appear to be temporary and due to impaired kidney function (Schafer et al. 1977).

A number of studies have been conducted relating to the primary hazards of Starlicide use in animal feedlots, indicating that the only major birds at risk are blackbirds when use directions for this product are followed (Besser et al. 1967, Ford 1967, Royall et al. 1967). This selectivity has been accomplished by the selection of chemical, of baits (pellets) that are well accepted by starlings and rejected by other species, and the dilution of treated baits with untreated to minimize overtreatment. Numerous instances of gallinaceous birds, sparrows and other non-target species feeding on Starlicide baits have been recorded with no reported mortalities (Ford 1967, Royall et al. 1967).

Similarly, none of the remaining registered uses have resulted in significant primary hazards to non-target species because of selectivity in bait choices and placement (Simpson 1972, Kreps 1974). Use of CAT/CPT in experimental roost sprays in the U.S. and France have similarly demonstrated the selectivity of this family of compounds to birds in general, and to pest birds specifically.

The secondary hazards of starlicide products have been assessed numerous times, both in the laboratory and in the field. The only instances of documented secondary poisoning have occurred when crows have scavenged on the gut contents of pigeons killed with Starlicide baits (Kreps 1974). Instances of dogs, cats, hogs, owls, fox and hawks preying or scavenging on starlicide-killed birds have never resulted in documented secondary poisoning in the field (Besser et al. 1967, Ford 1967, Royall et al. 1967). Laboratory studies have verified the lack of secondary hazards (DeCino et al. 1966, Lefebvre et al. 1981). It is important to note that applications of this family of compounds involving routes other than oral may result in secondary hazards to some selected species. For example, lethal dermal applications of CPT or CAT can result in massive amounts of external body contamination (5 to 10 mg/bird). This contamination could easily prove fatal to the few predatory or scavenger species such as cats and owls that are sensitive to these compounds. Tests conducted at the DWRC have verified the susceptibility of these animals, but, in general, it appears that Starlicide/CAT/CPT are almost universally low in secondary hazard potential.

CHLORINATED HYDROCARBONS

Endrin

Endrin (1a alpha, 2 beta,2a beta,3 alpha,6 alpha,6a beta,7 beta, 7a alpha)-3,4,5,6,9,9,-hexachloro-1a,2,2a,3,6,6a,7,7a-octahydro-2,7:3,6-dimethanonaphth(2,3-b)oxirene) is currently Federally registered for use in wicked perches for the control of starlings, sparrows and pigeons in or adjacent to structures, and its use is limited to pest control operators (PCOs). As a result of the EPA RPAR deliberations, endrin cannot now be used within one mile of Peregrine falcon roosting sites nor two miles of nesting sites (Anonymous 1979).

Endrin is an insecticide and directly affects the CNS through the stimulation of vagal centers and simulates the effects of strychnine (Deichman and Gerard 1969). It is also highly soluble in body fats, is accumulated, and can be released in lethal amounts during periods of stress (Schafer 1981). Endrin is broadly and highly toxic to all species of birds and mammals with acute oral and dermal LD50s generally less than 20 mg/kg. Mortalities from orally or dermally poisoned birds can occur within hours of exposure but may occur many days later, depending upon the amount of endrin encountered. Because it is accumulated and is quite resistant to degredation/metabolism, endrin is even more toxic by chronic administration and has been implicated in reproductive failure in birds (DeWitt 1956).

Primary hazards to non-target birds and mammals from wicked perch applications of endrin are limited by the placement of the perch and the small numbers of non-target birds that frequent sites where such perches are applied (Jackson 1978). This is particularly true in urban areas where almost all of the birds roosting inside or on structures are pest species. However, because of its non-selectivity, any bird alighting on an endrin-treated perch will probably be killed. Primary hazards to mammals are almost nonexistant due to the placement of perches on structural members well off the ground surface. By exercising care not to overfill perches, providing a secure mounting, and by following use directions, primary mammal poisoning need not be of major concern.

Secondary hazards to non-target birds from field exposure to birds killed or intoxicated by endrin perch treatments are not well documented, but it is an area of endrin toxicity that needs to be addressed. Although endrin has a very high potential for causing secondary hazards, careful use and pick-up of bird carcasses should preclude problems in urban and suburban areas where pigeons and sparrows are the problem species. The potential problem in urban, and particularly suburban areas, where endrin is used to control starlings is much harder to solve. The reason for this concern is that starlings are opportunistic feeders and their feeding range can encompass an area up to 30 miles in diameter around their roosting site. Thus starlings may die in a wide variety of areas where they can become prey for a number of avian and mammalian predators and scavengers, many of which are more sensitive to endrin intoxication than the target species (Rudd and Genelly 1956, Schafer et al. 1969). Raptors are particularly sensitive (Schafer et al. 1969). Thus, although documented kills of predatory and scavenger species following wicked perch use of endrin do not appear in the literature, it is not necessarily because there are no hazards but may be simply because of the difficulty in making objective observations.

DETERGENTS

PA-14

PA-14 is a non-ionic surfactant that lowers the surface tension of water, enhancing its ability to penetrate the oily feathers of birds. When PA-14 is applied to roosting birds and proper temperature and moisture conditions follow, it is a very effective material for inducing mortality (Lefebvre and Seubert 1970). PA-14 is Federally registered for the control of roosting blackbirds and starlings in the U.S. under the guidance of governmental agencies trained in bird control. Aerial application procedures are currently being modified to include application by ground-based (sprinkler-irrigation) systems to reduce reliance on rainfall.

PA-14 is not highly toxic to birds and mammals (LD50s are in the 2.0 to 3.0 gm/kg range), but simply causes death from hypothermia (Lefebvre and Seubert 1970). Mortalities generally occur in the roost proper and within 5 to 10 hours of application, but, depending on weather conditions, birds can die anywhere within their feeding range.

Primary hazards of PA-14 are very limited due to its low acute toxicity and biodegradability. Species at risk include those birds that may coinhabit roost sites with blackbirds, although their number and variety is normally limited. Robins (Turdus migratorius) and Rusty blackbirds (Euphagus carolinus) are probably the largest number of non-target species present in most roosts (Lefebvre and Seubert 1970). Because PA-14 is not selective, effective applications will be similarly non-selective in producing mortality of non-target species. PA-14 is also highly, but reversibly irritating, to mucous membranes, which represents its major hazard to non-target organisms.

No known instances of secondary poisoning have occurred with PA-14. Secondary hazards of PA-14 are, for all practical purposes, negligible because it has little acute toxicity and no chronic toxicity.

ORGANOPHOSPHATES

Fenthion

Fenthion (O-dimethyl O-(3-methyl-4-methylthio)phenyl phosphorothioate) is federally registered in the U.S. for controlling pest birds only when it is used in wicked perches, and its use is restricted to PCOs. Fenthion has been used in the U.S. and other countries as an aerially applied avicide, but this use is not registered in the U.S. because of the high application rates required and the potential of this compound--and most organophosphates--for producing secondary hazards. Other than label directions, no specific restrictions exist for the use of fenthion in wicked perches.

Fenthion, like most organophosphates, is an irreversible inhibitor of acetylcholinesterase (Schafer 1971). This, and the knowledge that one or more fenthion metabolites are more toxic than the parent chemical, indicates that fenthion will display a high degree of chronicity in sensitive animal species (Deichman and Gerarde 1969). Mortalities of birds from dermally applied fenthion generally occur from 3 to 12 hours post-exposure, but can take place over a period of days, and are characterized by generalized convulsions and respiratory arrest.

Primary hazards from wicked perch applications of fenthion are similar to those previously discussed with endrin, except that hazards to most mammals should be considerably reduced due to the lower degree of acute toxicity displayed by fenthion. Acute oral LD50s for most bird species range from 1.0 to 20 mg/kg while most mammals are susceptible in the 100-500 mg/kg range (Schafer 1981). The acute dermal toxicity of fenthion to bird and mammal species is generally 2 to 5 times the acute oral dose.

Secondary hazards of fenthion are also similar to those discussed with endrin except that mammalian predatory or scavenger species are much less likely to be impacted due to the lesser degree of toxicity to mammalian species (Schafer 1981). Fenthion is also less persistant in the environment than endrin and thus hazards from repeated exposure are likely to be much reduced. However, it should be emphasized that fenthion, even though it is a much safer compound to use in wicked perches from the standpoint of the applicator and mammalian predatory and scavenger species, still presents a potential risk to avian predators and scavengers that is very similar to endrin (Schafer et al. 1969).

PYRIDINES

4-Aminopyridine

4-Aminopyridine (4AP, Avitrol) is probably the avicide in greatest use in the U.S. today. It is Federally registered for use on a number of species including blackbirds, sparrows and pigeons in and around structures; blackbirds, crows, sparrows and starlings in and around roosting and feeding sites; starlings in feedlots; blackbirds and starlings in ripening corn and sunflower; and gulls at breeding sites. In addition, it is state-registered in California for control of finches in grapes and sparrows in sprouting agricultural crops. The use of 4AP is limited to PCOs and Certified Applicators. Although 4AP is often considered an area repellent or frightening agent, it is essentially a toxicant.

4AP is highly toxic to all vertebrates. Acute oral LD50s range from 1.0 to 20 mg/kg for almost all avian and mammalian species. In birds and mammals it produces symptoms typical of CNS stimulants with the initial symptoms occurring from 10 to 30 minutes after ingestion and death occurring up to 4 hours later (Schafer et al. 1973). Occasionally the convulsive stages are accompanied by audible vocalizations due to the involuntary contraction of the diaphragm. In most gregarious species of birds the vocalizations are pronounced and as a result non-affected birds are frightened from the immediate area (Schafer 1981). Sparrows, pigeons and doves do not produce loud or effective vocalizations; however, their eratic behavior has a similar effect on nearby birds. 4AP has repeatedly been shown to be non-cumulative in birds and mammals and is either rapidly metabolized or excreted by intoxicated animals (Schafer 1981). Reproductive effects of 4AP appear to be related to the direct toxic effects of 4AP (Schafer et al. 1975).

Primary hazards from the use of 4AP in bird control depend upon the method of exposure to target species, since 4AP is simply just another highly toxic chemical with little built-in differential selectivity between target and non-target species (Schafer 1981). In urban areas, short-term exposure

of toxic baits to prebaited birds and the use of dilutions with non-treated baits can effectively reduce overtreatment and at least indicate which non-target species are present (Mampe 1976). In agricultural areas the use of dilution factors and bait placement becomes more important in order to take advantage of any possible bias in the feeding habits of non-target species (Besser 1976). Small rodents may be impacted, but no reports of dead or dying rodents have been reported in conjunction with avicidal uses of 4AP. Hazards to domestic animals and children can be minimized through the proper placement of bait material and cleanup of any unconsumed baits.

4AP has repeatedly been shown to present minimum secondary-hazard potential to predatory and scavenger animals under a variety of laboratory test conditions (Schafer et al. 1974). In field use, only scavenger species such as magpies and crows appear to have been impacted. However, when some species of birds are allowed to eat 4AP-treated baits with little or no diluent present, 4AP can potentially result in secondary hazards to cats, dogs and raptors that consume unassimilated 4AP from the GI tract of prey species (Holler and Schafer 1982).

SUMMARY

The phenomenon of primary and secondary hazards, both potential and observed, is a complex interaction of many factors that can and has often been modified to mitigate hazards. These include, but are not limited to, relative susceptibility to intoxication, relative body size, feeding habits, protective responses, bait types, chemical concentration, application rates, types of exposure, time to intoxication, when and where mortalities occur, route of exposure, sublethal aversions, relative acceptability, timing of treatment, habitats treated, etc. It is important to recognize that secondary hazards may also result from consumption/exposure of unassimilated toxicant which is more a form of "primary" than "secondary" exposure. The former is exemplified by consumption of prey gut contents as opposed to consumption of organ/muscle tissues.

Toxicity data alone do not constitute the only basis for estimating the risks of chemicals to non-target species, but is only one of the factors to be considered. Risks can only be accurately assessed when all the chemicals, toxicological and behavioral parameters can be evaluated as a package. In this paper I have attempted to provide the reader with the current state-of-the-art hazard information with respect to six avicides. Application of this information in accurately assessing risks for specific situations must take into consideration all known mitigating circumstances.

LITERATURE CITED

ANONYMOUS. 1979. Endrin; Intent to cancel registrations and denial of applications for registration of pesticide products containing endrin and statement of reasons. Fed. Reg. 44(144):43632-43657.

ANONYMOUS. 1983. Intent to cancel registrations of pesticide products containing strychnine; Denial of applications for registration of pesticide products containing strychnine; Determination concluding the rebuttable presumption against registration; Availability of position document. Fed. Reg. 48(203):48522-48530.

APOSTOLOU, A. 1969. Comparative toxicology of the avicides 3-chloro-p-toluidine and 2-chloro-4-acetotoluidine in birds and mammals. Ph.D. Thesis, Univ. of Calif., Davis, California. 85pp.

BESSER, J. F. 1976. 4-Aminopyridine for protecting crops from birds-A current review. Proc. Vertebr. Pest Conf. 8:51-53.

BESSER, J. F., W. C. ROYALL, JR., and J. W. DEGRAZIO. 1967. Baiting starlings with DRC-1339 at a cattle feedlot. J. Wildl. Manage. 31(1):48-51.

CLARK, D. O. 1976. An overview of depredating bird damage control in California. Proc. Bird Control Seminar 7:21-27.

CUNNINGHAM, D. J., E. W. SCHAFER, JR., and L. K. MCCONNELL. 1981. DRC-1339 and DRC-2698 residues in starlings: preliminary evaluation of their effects on secondary hazard potential. Proc. Bird Control Seminar 8:65-70. Bowling Green, Ohio.

DECINO, T. J., D. J. CUNNINGHAM, and E. W. SCHAFER, JR. 1966. Toxicity of DRC-1339 to starlings. J. Wildl. Manage. 30(2):249-253.

DEICHMANN, W. B., and H. W. GERARDE. 1969. Toxicology of drugs and chemicals. Academic Press, New York. pp238-239.

DEWITT, J. B. 1956. Chronic toxicity to quail and pheasants of some chlorinated insecticides. J. Agric. Food Chem. 4:863.

FELSENSTEIN, W. C., R. P. SMITH, and R. E. GOSSELIN. 1974. Toxicological studies on the avicide 3-chloro-4-methylaniline. Toxicol. Appl. Pharmacol. 28(1):110-125.

FORD, H. S. 1967. Winter starling control in Idaho, Nevada and Oregon. Proc. Vertebr. Pest Conf. 3: 104-110.

HOLLER, N. R., and E. W. SCHAFER, JR. 1982. Potential secondary hazards of Avitrol baits to Sharp-shinned hawks and American kestrels. J. Wildl. Manage. 46(2):457-462.

KOEHLER, J. W. 1962. Linnets, Horned larks, Crowned sparrows and woodpeckers. Proc. Vertebr. Pest Control Conf. 1:174-185.

KREPS, L. B. 1974. Feral pigeon control. Proc. Vertebr. Pest Conf. 6:257-262.

JACKSON, W. B. 1978. Rid-A-Bird perches to control bird damage. Proc. Vertebr. Pest Conf. 8:47-50.

LEFEBVRE, P. W., and J. L. SEUBERT. 1970. Surfactants as blackbird stressing agents. Proc. Vertebr. Pest Conf. 4:156-161.

LEFEBVRE, P. W., N. R. HOLLER, R. E. MATTESON, E. W. SCHAFER, JR., and D. J. CUNNINGHAM. 1981. Developmental status of N-(3-cholro-4-methylphenyl)-acetamide as a candidate blackbird/starling roost toxicant. Proc. Bird Control Seminar 8:65-70.

LYNDALL, R. 1962. Controlling Yellow-billed magpies. Proc. Vertebr. Pest Control Conf. 1:186-189.

MAMPE, C. D. 1976. Current status report-pigeon control. Proc. Bird Control Seminar 7:95-101.

MATHENY, R. W. 1980. Federally registered pesticides for vertebrate pest control. Proc. Vertebr. Pest Conf. 9:63-73.
MULL, R. L., and S. N. GIRI. 1972. The role of renal aromatic N-deacetylase in selective toxicity of avicide 3-chloro-p-toluidine in birds. Biochem. Biophys. Acta 273:222-228.
OCHS, P. 1976. Strychnine. Proc. Bird Control Seminar 7:108-110.
PALMER, T. K. 1970. House finch control in California. Proc. Vertebr. Pest Conf. 4:173-178.
PALMORE, W. P. 1978. Diagnosis of toxic acute renal failure in cats. Florida Vet. J. 14-15:36-37.
PEOPLES, S. A., A. BARGER, A. C. CRABB, and R. G. SCHWAB. 1976. A progress report on a new avicide: 2-chloro-4-acetotoluidide. Proc. Bird Control Seminar 7:245-246.
ROYALL, W. C., JR., T. J. DECINO, and J. F. BESSER. 1967. Reduction of a starling population at a turkey farm. Poult. Sci. 46(6):1494-1495.
RUDD, R. L., and R. E. GENELLY. 1956. Pesticides: Their use and toxicity in relation to wildlife. Calif. Dept. Fish and Game Bull. No. 7, pp. 139-141.
SAVARIE, P. J. 1981. The nature, modes of action, and toxicity of rodenticides. In: Handbook Series in Agriculture. Vol. III, A. A. Hanson, Ed., CRC Press, West Palm Beach, Florida. pp.113-128.
SCHAFER, E. W., JR. 1979. Registered bird damage chemical controls. Pest Control 47(6):36-39.
SCHAFER, E. W., JR. 1981. Bird control chemicals-nature, modes of action, and toxicity. In: Handbook Series in Agriculture. Vol. III, A. A. Hanson, Ed., CRC Press, West Palm Beach, Florida. pp.129-139.
SCHAFER, E. W., JR., R. B. BRUNTON, and D. J. CUNNINGHAM. 1973. A summary of the acute toxicity of 4-aminopyridine to birds and mammals. Toxicol. Appl. Pharmacol. 26:532-538.
SCHAFER, E. W., JR., R. B. BRUNTON, and N. F. LOCKYER. 1974. Hazards to animals feeding on blackbirds killed with 4-aminopyridine baits. J. Wildl. Manage. 38(3):424-426.
SCHAFER, E. W., JR., R. B. BRUNTON, and N. F. LOCKYER. 1975. The effects of subacute and chronic exposure to 4-aminopyridine on reproduction in Coturinx quail. Bull. Environm. Contam. Toxicol. 13(6):758-764.
SCHAFER, E. W., JR., R. R. WEST, and D. J. CUNNINGHAM. 1969. New starling toxicant: DRC-1347. Pest Control 37(9):22,24,30.
SCHAFER, E. W., JR., R. B. BRUNTON, D. J. CUNNINGHAM, and N. F. LOCKYER. 1977. The chronic toxicity of 3-chloro-4-methylbenzamine HCl to birds. Arch. Environm. Contam. Toxicol. 6:241-248.
SCHITOSKEY, F., JR. 1975. Primary and secondary hazards of three rodenticides to Kit fox. J. Wildl. Manage. 39(2):416-418.
SIMPSON, G. 1972. Some approaches to controlling depredations by crows and jays in Tulare County. Proc. Vertebr. Pest Conf. 5:112-117.
WATKINS, J. 1976. Use of strychnine treated grain for industrial pigeon control. Proc. Bird Control Seminar 7:94-100.