# Evaluation of Black-billed Magpies as Indicators of Environmental Contamination at the Rocky Mountain Arsenal

Work Plan - 1995

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by

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# **INTRODUCTION**

The Rocky Mountain Arsenal (RMA) is located in southwest Adams County, Colorado, adjacent to metropolitan Denver. Established in 1942, the U. S. Army utilized the Arsenal for the production, storage, and dismantling of conventional and chemical munitions. Later, Arsenal facilities were leased to private companies for the production of chemical pesticides. During these times, significant areas on the Arsenal were contaminated by waste storage and disposal practices. In 1982 the Arsenal was added to the National Priorities List and managed as a Superfund site. Since 1989, the Army and U. S. Fish and Wildlife Service have managed the Arsenal as a Wildlife Area. The Rocky Mountain Arsenal National Wildlife Refuge Act was passed in 1992.

Numerous programs on the Arsenal have monitored contaminant locations and concentrations. Of 666 chemicals attributable to past activities on the Arsenal (Ebasco 1988), 22 were selected for continued monitoring because they were detected in the Arsenal biota (Stollar et al. 1990). These compounds were ranked based on carcinogenic and teratogenic properties, solubility in water, soil leaching, and solubility in lipids. Ten compounds currently of special concern are aldrin, dieldrin, endrin, DDT, DDE, arsenic, mercury, isodrin, *gamma*-chlordane, and *alpha*-chlordane. Of these, the organochlorine dieldrin is the major analyte of concern for wildlife risk. It was selected because of its distribution on the Arsenal (ESE 1987), its toxicity and persistence, and its potential to bioaccumulate (Stickel 1970). Dieldrin was detected most often in the Arsenal biota, being found in 60% of sedentary biota samples and at all trophic levels (Stollar et al. 1992).

# Dieldrin

Dieldrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octrahydro-*endo*-1,4*exo*-5,8-dimethanonaphthalene) is a chlorinated cyclodiene. It was primarily used as an agricultural pesticide applied directly to the soil. Dieldrin is stable in the environment over a broad range of pH and soil conditions (ESE 1989) and has a high potential to bioaccumulate. Earthworms concentrated dieldrin 7.1 times soil levels and insects 11.9-58.4 times soil levels in a simulated terrestrial ecosystem (Gile and Gillett 1979). In kestrels fed DDT and dieldrin and barn owls fed dieldrin, residues in carcasses showed geometric mean accumulations of 5.0-5.1 and 15.9-16.6, respectively (Mendenhall et al. 1983, Wiemeyer et al. 1986).

Dieldrin toxicity has been assessed in the laboratory and on wildlife (Hudson et al. 1984) with most work focusing on mammals. Exposure to dieldrin has been associated with alterations in carbohydrate metabolism (Reymann et al. 1983, Mahmood et al. 1981), hepatic effects (Smith 1991), responses in the central nervous system (Matsumura and Ghiadsuddin 1983, Eldefrawi et al. 1985, Cole and Cassida 1986), and immune responses (Krzystyniak et al. 1985). Reproductive effects in mammals include decreased litter size, toxicity to young (Treon and Cleveland 1955), and behavioral neglect by parents (Virgo and Bellward 1973).

Information on avian response to dieldrin is more limited, but birds do tend to show a range of sensitivities. Tucker and Haegele (1971) studied the oral toxicity of dieldrin in six avian species and found  $LC_{50}$  ranging from 23.4 mg/kg for chukar (<u>Alectoris chukar</u>) to 79.0 mg/kg for ring-necked pheasants (<u>Phasianus colchicus</u>). An  $LC_{50}$  of 9 mg/kg has been found for California quail (<u>Callipepla californica</u>) and grey partridge (<u>Perdix perdix</u>), while mallards (<u>Anas platyrhynchos</u>) had an  $LC_{50}$  of 381 mg/kg (Hudson et al. 1984). Ohlendorf et al. (1981) determined that brain concentrations of 5 ppm dieldrin are hazardous to some avian

species and 9 ppm is diagnostic of dieldrin poisoning. Weimeyer and Cromartie (1981) stated that dieldrin levels in the brain of 3.2 ppm were hazardous and 4.0 ppm lethal. Cowbirds may be adversely affected at 1 ppm (Heinz and Johnson 1981). Brains residues of dieldrin in dead birds were not correlated with treatment level, sex or reproductive status (Fergin and Schafer 1977). Dosage levels can affect the timing of death, but not the resulting brain residues. Death occurs when brain levels reach a certain lethal threshold regardless of treatment level. Environmental factors can be important in determining toxic levels of dieldrin (DeWitt 1956, Davison et al. 1971, Fergin and Schafer 1977).

Effects of dieldrin may be sublethal. Increased dieldrin levels in mallard diets led to decreased biogenic amines serotonin, norepinephrine, and dopamine and increased micorsomal enzymes and liver proteins, DNA, and RNA (Sharma et al. 1976). Dieldrin may also deplete neurotransmitters (Heinz et al. 1980) or alter whole brain serotonin (Willhite and Sharma 1978). Behavioral changes resulting in reduced survival chances may also occur. In mallards, decreased pecking and increased avoidance actions were observed with increasing dieldrin levels (Sharma et al. 1976). At 2 ppm dieldrin, Busbee (1977) observed changes in the ontogeny of mouse killing in loggerhead shrikes (Lanius Judovicianus). Quail chicks suppressed the group avoidance response to a moving silhouette at 5 ppm dietary level (Kreitzer and Heinz 1974). In herring gulls (Larus argentatus), nest defense and attentiveness decreased significantly in contaminated colonies (Fox et al. 1978). Dieldrin accumulates in egg yolks but does not affect hatchability. It may poison chicks (St. Omen 1970). In Barn owls (Tyto albo), dieldrin produced slight eggshell thinning but did not reduce overall

breeding success (Mendenhall et al. 1983). The estimated critical level of dieldrin in eggs is greater than 1 ppm (Blus 1982).

## **Black-billed Magpies**

Black-billed magpies (<u>Pica pica</u>) occur throughout the Northern hemisphere, including western portions of North America. They are abundant, year long residents at the Arsenal. In Montana, adults average 180.0 g (Todd 1968). Females are slightly smaller (10%) than males but morphological measurements generally overlap between the sexes. First year birds are similar in size to adults, but can distinguished by plumage characteristics (Birkhead 1991).

North American magpies build large, domed nests. Nests are aggregated in suitable habitat and may not be evenly distributed (Reese and Kadlec 1985, Buitron 1988, Birkhead 1991). While magpies do not protect exclusive territories, a small area (30 m radius) around the nest is defended during the breeding season (Buitron 1988). Generally, magpies feed outside of the area at a group feeding ground. Territorial interactions between neighboring magpies are infrequent (Reese and Kadlec 1985, Buitron 1988). Magpies are generally monogamous and may stay paired for several seasons. Breeding birds are sedentary and often use the same territory year after year. In South Dakota, magpies movements averaged only 280 m between breeding seasons (Buitron 1988). Up to 40 % of a population may not breed in a given year (Birkhead 1991). In North America, these non-breeding birds roam widely and are not easily studied. In Europe, 81% and 17% of non-breeders are first year birds and second year birds, respectively (Birkhead 1991).

Nests are initiated in late April, after a courtship and nest building (or renovation) period. Magpies are determinate layers (Davis 1995) with average clutch sizes of just over 6 eggs (Erpino 1969, Reese and Kadlec 1985, Buitron 1988). The female begins incubation part way through laying. The incubation period lasts from 18 - 24 days and eggs hatch asynchronously over 2-3 days (Birkhead 1991). The male feeds the female, the exclusive incubator, at a mean rate of 1.6 feeds/hr during this period (Buitron 1988). Following failed clutches, renesting rates are 10-16% (Brown 1957, Reese and Kadlec 1985). Both parents feed the young (male 3.1 feeds/hr, female 1.4 feeds/hr). The female broods nestlings 80% of the time at day 1 and less than 10% at day 15 (Buitron 1988). Young mappies grow rapidly from 7 g at hatching to 180 g after day 18. They fledge at approximately 27 days at 75-100% of adult weight, but may remain dependant on adults up to 70 days (See Birkhead 1991). In North America, 60-73.5% of nests produce at least one fledgling. Number of chicks produced per pair ranges from 2.2 -2.5, or 3.5-4.4 chicks per successful pair (Birkhead 1991). Most nestling mortality is the result of starvation. Little is known about fledgling dispersal and survival in North America. Low returns from banding studies (Buitron 1988) suggest that mortality may be high and dispersal distances may be large. No information on adult survival is available for North American magpies.

Magpies are generalist ground feeders. Preferred feeding areas are short grasslands and wet meadows. Adults show a seasonal variation in food habits, feeding on plant material in winter and animal material, mostly invertebrates, in summer (Birkhead 1991). Other food items include carrion, small mammals, fruit, small birds, and reptiles (Kalmbach 1927). Analysis of magpie pellets by Reebs and Boag (1987) suggested that small mammals may be

important to wintering magpies in Alberta, Canada. Nestlings are fed mostly invertebrates, including beetles, flies, caterpillars, and grasshoppers (Kalmbach 1927). Magpies are short term hoarders and cache surplus food, especially in the autumn (Birkhead 1991).

#### **Black-billed magpies as Biomonitors**

Although black-billed magpies have not been utilized in a formal biomonitoring program, they have been shown to be sensitive to organophosphate insecticides. Henny et al. (1985) found 38 dead magpies in three months following pour-on application of famphur to control cattle warbles (<u>Hypoderma</u> sp.). Starting on treatment day, magpie deaths peaked between day 5 and day 13. Residue concentrations in the gizzards of dead birds were estimated at 5.2-6.1 mg/kg. Brain cholinesterase was depressed 70-92% in all dead birds. A red-tail hawk (<u>Buteo jamaicensis</u>) was found dead after eating a famphur contaminated magpie. Magpies have also been used to detect the presence of heavy metals. Black-billed magpie feathers were used to detect the presence of lead and cadmium around industrial areas in Poland (Dmowski and Golimowski 1993). These researchers detected lead levels as high as 1500 micrograms/g dry wt. and cadmim levels as high as 40 micrograms/g dry wt. in magpie feathers.

Black-billed magpies may be a suitable biomonitor for environmental contaminants at the Rocky Mountain Arsenal. Magpies are dispersed across the Arsenal in contaminated and uncontaminated areas, and are common at off-site areas for comparisons. They are easily observed, tolerant of human activities, and lend themselves to demographic and reproductive studies. Magpies occupy an intermediate trophic level, are year round residents, and utilize a variety of foods and so may be susceptible to bioaccumulation and bioamplification. Because

of their sedentary habits and long lifespan (up to 8 years), they arealso suitable for long term monitoring.

# **OBJECTIVES**

The purpose of this investigation is to evaluate black-billed magpies as biomonitors of environmental contamination at RMA. Because little is known about magpie responses to contaminants, this study will collect baseline data on exposure pathways, exposure levels, and demographic, reproductive, and behavioral responses of magpies. This data will be used in the design of a long-term biomonitoring program using black-billed magpies. While dieldrin will be the major analyte of concern, I will evaluate exposure and responses to other COC's when possible.

The objectives of this investigation are:

1) quantify exposure of dieldrin and the other COC's to magpie nestlings and adults by analyzing for these contaminants in eggs, nestlings, fledglings, adults, and prey items, and by correlating residues to contaminant levels in home ranges and core use areas,

2) quantify response to exposure by monitoring magpie densities, hatching success, nestling growth and development, nestling survival, fledgling survival, adult survival, and behavioral attentiveness of breeding adult magpies,

3) create a Geographic Information System (GIS) integrating contaminant distributions, magpie use areas, exposure levels, and magpie responses, and

4) collect baseline data and develop tools to be used in the design and implementation of a long term biomonitoring program that can provide information on contaminant bioavailability and remediation efficacy.

STUDY PLAN

This research is scheduled to be conducted for three full field seasons. In 1995, research will focus on collecting baseline demographic, reproductive, and behavioral data and collecting samples for contaminant analysis. In addition, 1995 will be used as a pilot season for the development and evaluation of field techniques and sampling efficiency. Of particular concern will be the usefulness of lethal vs non-lethal samples, evaluation of statistical power and sample sizes, and the necessity of a companion positive dosing study. At this time, a dosing study is not planned. However, if it is deemed necessary for the interpretation of field results, a dosing study will be designed in 1996 and initiated in 1997. Current budgets do not include support for a dosing study.

### METHODS

# **Experimental Design**

This study has been designed around three treatment levels, core, non-core, and offsite. Birds, nests, samples, demographic, and reproductive measurements will be designated to a treatment level based on geographic location. Core will include the interior six sections on RMA (Sections 1, 2, 25, 26, 35, 36), non-core will encompass the remaining areas of RMA, and off-site will include one or more off-post areas. I will collect samples randomly within each area. At a finer scale, I will use overlap between known contaminant distributions and magpie home range estimates as a continuous measure of exposure to provide greater resolution in evaluating correlations between exposure and response variables.

#### **Capture and Marking**

I will attempt to capture and mark all adult and juvenile magpies in each of the treatment areas. I will utilize a variety of traps to capture adults, including circular walk-in traps (Alsager et al. 1972), bal chatri (Berger and Mueller 1959), bow nets (Tordoff 1954), Tomahawk live-traps, and noose carpets (Collister 1967, Anderson and Hamerstrom 1967, Kahn and Millsap 1978). I will use carrion, small mammals, grains, and if necessary, a captive female magpie as bait. Nestlings will be taken from the nest for marking several days prior to fledging.

Upon capture, I will place a hood over the bird's head, weigh it using an Acculab electronic scale, and record its general condition. I will determine its age from plumage characteristics and measure wing length, tail length, bill length, bill depth, head length, and tarsal width. These measurements will be used in discriminant function analysis to aid the determination of sex (Birkhead 1991). Each bird will be fitted with a colored visual identification leg band and a standard USFWS band.

# **Exposure Assessment**

<u>Telemetry</u>--Upon capture, 10 adult and 10 fledgling magpies in each treatment area will be fitted with a radio transmitter. I will attach transmitters to tail retrices (Fitzner and Fitzner 1977) or with a leg harness (Rappole and Tipton 1991). I will use telemetry to visually locate each instrumented bird four times each week. Sampling times will be randomized each day. In addition to the instrumented animals, I will collect visual locations on 20 non-transmittered birds (10 adult, 10 fledglings) in each treatment area. For each bird with at least 30 independent locations, I will estimate 95% (home range) and 50% (core use) harmonic mean

contours (Dixon and Chapman 1980). I will integrate this data into a GIS and calculate overlap between magpie use areas and known areas of contamination.

As North American magpies are social feeders and generally use group feeding areas, I will investigate the mechanisms of these feeding behaviors. This information will be used in conjunction with home range data to estimate exposure. I will place a carrion food source (roadkills) at random locations on RMA 30 min before sunrise. I will observe the food from a distance for four hours and record the time and direction from which each visiting magpie arrives. Following the initial four hour observation period, I will revisit the food source every two hours and record which birds are present. When used with home range data, this information will be used to determine distances that individual magpies will travel to a food source and indicate potential exposure to birds on the periphery of contaminated areas.

I will use instrumented adults and juveniles to determine dispersal distances and directions. This data will allow the determination of movement patterns (sources and sinks) on and around RMA and provide a more detailed evaluation of exposure and movements that could potentially confound a long term monitoring study.

<u>Samples</u>--I will collect lethal and non-lethal samples for contaminant analysis. From each captured bird I will collect 2 ml of blood from the jugular or brachial vein. I will swab the area with 70% ETOH and use a 3 cc syringe with a 0.5 inch, 26 g needle rinsed with sodium heparin. Blood serum will be separated and divided for metal and organochlorine analyses. Blood will be stored in RMA freezer facilities until being sent to the contract laboratory.

I will collect 2 eggs from 10 randomly selected nests in each treatment area. I will collect eggs during the first week of incubation to coincide with planned nest monitoring.

Eggs will be kept on ice until being taken to RMA freezer facilities and forwarded to the contract laboratory.

I will collect 2 nestlings from 10 random nests (excluding nests in which egg samples have been taken) in each treatment area. I will remove nestlings during planned banding activities several days prior to fledging. I will collect 10 adults from each of the treatment areas following breeding activities. All specimens will be stored in RMA freezer facilities until being sent to the contract laboratory. Blood, liver, and carcasses will be analyzed for organochlorines, arsenic, and mercury. Brain tissue will be analyzed for organochlorine residues.

<u>Prey Contamination</u>--I will determine contaminant exposure in nestling prey using esophageal constrictions (Hoff 1992, Mellot and Woods 1993) on 2 chicks from 10 different nests in each of the treatment areas. I will sample nestlings for one hour on the mornings of day 6, 15, and 21. I will use small metal wing tags to identify individual chicks. Collected food items will be identified, placed in glass containers, and stored in RMA freezer facilities until being sent to the contract laboratory.

I will attempt to find and locate adult food caches when the activity is observed. I will collect pellets from nesting and roosting areas to determine prey usage. If necessary, I will sample prey within known magpie feeding areas. Samples will be collected and handled the same as nestling prey samples.

# **Exposure Response**

<u>Density</u>--I will use capture data as a complete census to calculate magpie densities in each treatment area. If all birds are not marked, I will drive transects through each area to determine the proportion of unmarked birds to estimate population size and density. <u>Hatching rate</u>--I will use foot and vehicle surveys to locate all potential magpie nests and monitor activity. I will check active nests 3-4 days after incubation begins to determine maximum clutch size and collect eggs for contaminant analysis. I will recheck nests several days prior to hatching and again in 5-6 days to determine hatching rate (# hatched / # of eggs).

<u>Nestling growth</u>--In each treatment area, I will use the 10 nests chosen for esophageal constrictions to monitor nestling growth. I will weigh each chick and measure total length, wing length, and bill length following esophageal sampling at days 6, 15, and 21. I will also check for any physical abnormalities or growth deformities. If growth is similar between chicks used for prey sampling and those not, I will pool the data. Otherwise, only data from non-sampled chicks will be used.

<u>Nestling Survival and Nest success</u>--For each nest, I will determine the number of chicks that survive to fledging age (27 days) to estimate nestling survival. I will calculate nest success for each treatment area as # nest fledging at least one chick / total # of nests with at least one egg.

<u>Survival</u>--I will use instrumented birds to calculate adult and juvenile survival. Birds will be relocated visually four times each week. I will use Kaplan Meier survival estimates (Kaplan and Meier 1958, Pollack et al. 1989) to estimate monthly survival rates.

#### **Behavior**

Because both parent's contributions are essential to nest success in magpies, (Birkhead 1991), lack of nest attentiveness or neglect by either parent will result in breeding failure. Therefore, I will sample parent attentiveness at nests in each treatment area during the incubation and nestling stages.

I will randomly select 5 pairs in each treatment area to be observed. Each pair will be observed 6 times in the incubation stage and 6 times during the nestling stage. Sampling periods will last 2 hr. and be stratified into 3 daily periods. During each nesting stage, I will sample every pair twice in each daily period. I will record the amount of time the female spends on the nest and frequency of feeding bouts by the male. In addition, I will record aggressiveness of adults during planned nest visits, including the distance each birds remains from the observer, calling, and attempted strikes.

# Analysis

Exposure and Response--I will use Analysis of Variance (ANOVA) to test for differences in residues in eggs, fledglings, adults, and blood residues among the3 treatment levels. I will divide prey items into categories (invertebrate, animal, vegetable, other) and use ANOVA to test for differences in contaminant levels of each prey category between treatment areas. For each ANOVA, I will test assumptions of normality of errors with Shapiro-Wilkes test and assumptions of homogeneous variances of errors with Hartley's test. For birds with home range estimations (>30 locations), I will use regression analysis to test for relationships between home range and core use area overlap with known contaminant distribution (independent) and residues in egg, fledglings, adults, blood, and prey items (dependant). I will test the assumption that errors are normally distributed using Shapiro-Wilkes tests.

I will use multiple linear regression and logistic regression to evaluate relationships between exposure levels and measured response variables. Analysis will be geared toward determining exposure pathways, (e.g. Are nestling contaminant levels correlated to levels in adults or prey items?) and associated ecological responses (e.g. nestling growth and survival). <u>Geographic Analysis</u>--To determine the usefulness of black-billed magpie populations in monitoring the geographic range of contaminants, I will conduct geographic analysis using GS+ (Gamma Designs Software, Plainwell, MI, 1993) and GIS software. For each continuous variable of interest, including residue samples and reproductive parameters, I will use semivariance analysis to design an isotropic variogram. I will test the appropriateness of the model using cross-validation (jackknifing). I will use block kriging to interpolate sample data and calculate interpolation errors. Resulting map files will be output to GIS and integrated with contaminant distribution maps. By determining the correlation between each sample map and contaminant distribution maps, I will evaluate each variable to determine the best "predictor" of contaminant distribution.

# SUMMARY

I will evaluate black-billed magpies as biomonitors of contamination at RMA by investigating exposure levels, pathways, and magpie responses. Methods will include an intensive investigation of magpie ecology, including demographics, productivity, and behavior, combined with lethal and non-lethal measurements of exposure. An accompanying geographical analysis will investigate the potential of magpie monitoring to indicate the geographical distribution of contaminants. This study will provide baseline data on magpie

exposure and "health" and an evaluation of methodology for the design of a sustained monitoring program.

#### LITERATURE CITED

- Alsager, D. E., J. B. Stenrue, and R. L. Boyles. 1972. Capturing black-billed magpies with circular live traps. J. Wildl. Manage. 36:981-983.
- Anderson, K. A., and F. Hamerstrom. 1967. Hen decoys aid in trapping cock prairie chickens with bow nets and noose carpets. J. Wildl. Manage. 31:829-832.
- Berger, D. D., and H. C. Mueller. 1959. The bal chatri: a trap for the birds of prey. Birdbanding. 30:18-26.
- Birkhead, T. R. 1991. The Magpies: the ecology and behavior of black-billed and yellowbilled magpies. T and A D Poyser, London, England. 270pp.
- Blus, L. L. 1982. Further interpretation of the relation of organochlorine residues in brown pelican eggs to reproductive success. Environ. Pollut. 28:15-33.
- Brown, R. L. 1957. The population ecology of the magpie in western Montana. M.S. Thesis. Montana State Univ. Bozeman, Montana.
- Buitron, D. 1988. Female and male specialization in parental care and its consequences in black-billed magpies. Condor 90:29-39.
- Busbee, E. L. 1977. The effects of dieldrin on the behavior of young loggerhead shrikes. Auk 94:28-35.
- Cole, L. M., and Cassida. 1986. Polychlorocycloalkane insecticide induced convulsions in mice in relation to disruption of the GABA-regulated chloride ionophore. Life Sci. 39:1855-1862.
- Collister, A. 1967. Simple noose trap. West. Bird Bander 42:4.
- Davis, D. E. 1955. Determinate laying in barn swallow and black-billed magpies. Condor 57:81-87.
- Davison, K. L., J. L. Sell, and R. J. Rose. 1971. Dieldrin poisoning of chickens during severe dietary restriction. Bull. Environ. Contam. Toxicol. 7:245-255.
- DeWitt, J. B. 1956. Chronic toxicity to quail and pheasants of some chlorinated insecticides. J. Agri. Food Chem. 4:863-866.
- Dixon, K. R., and J. A. Chapman. 1980. Harmonic mean measure of animal activity centers. Ecology 61:1040-1044.

Dmowski. K, and J. Golimowski. 1993. Feathers of the magpie (Pica pica) as a bioindicator material for heavy metal pollution assessment. Sci. Total Environ. 139-140:251-8.

Ebasco. 1988. RMA Chemical Index, Volumes I, II, III.

- Eldefrawi, M. E., and S. M. Sherby, I. M. Abalis, and A. T. Eldefrawi. 1985. Interactions of Pyrethroid and cyclodiene insecticides with nicotine acetylcholine and GABA receptors. Neurotoxicology 6:47-62.
- Environmental Science and Engineering, Inc. 1987. Introduction to the Contamination Assessment Reports. Version 2.3. Prepared for Office of the Program Manager, Rocky Mountain Arsenal.
- \_\_\_\_\_. 1989. Environmental Setting, section 2.0. Rocky Mountain Arsenal biota remedial investigation-draft final report, version 2.2.
- Erpino, M. J. 1969. Seasonal cycle of reproductive physiology in black-billed magpie. Condor 71:267-279.
- Fergin, T. J., and E. C. Schafer. 1977. Toxicity of dieldrin to bobwhite quail in relation to sex and reproductive status. Arch. Environ. Contam. and Toxicol. 6:231-219.
- Fitzner, R. E., and J. N. Fitzner. 1977. A hot melt glue technique for attaching radio transmitter tail packages to raptorial birds. N. Amer. Bird Bander . 2:57.
- Fox, G. A., A. P. Gilman, D. B. Peakall, and F. W. Anderka. 1978. Behavioral abnormalities of nesting Lake Ontario Herring Gulls. J. Wildl. Manage. 42:477-483.
- Gile, J. D., and J. W. Gillett. 1979. Fate of <sup>14</sup>C dieldrin in a simulated terrestrial ecosystem. Arch. Environ. Contam. and Toxicol. 8:107-124.
- Heinz, G. H., E. F. Hill, and J. F. Contrera. 1980. Dopamine and norepinephrine depletion in ring doves fed DDE, dieldrin, and Aroclor 1254. Toxicol. Appl. Pharmacol. 53:75-82.
- \_\_\_\_, and R. W. Johnson. 1981. Diagnostic brain residues of dieldrin: some new insights. pp 72-92. <u>in</u> D. W. Lamb and E. E. Kenaga, Eds. Avian and mammalian wildlife toxicology: Second Conference. American Society for Testing Materials Special Technical Publication 757. 164 pp.
- Henny, C. J., L. J. Blus, E. J. Kolbe, and R. E. Fitzner. 1985. Organophosphate insecticide (Famphur) topically applied to cattle kills magpies and hawks. J. Wildl. Manage. 49:648-658.

- Hoff, D. J. 1992. Organophosphorus pesticide exposure to American kestrels (Falco sparverius) in a Southeastern Iowa agroecosystem. M. S. Thesis, Clemson University, 143 pp.
- Hudson, R. H., R. K. Tucker, and M. A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife: Second edition. United States Department of the Interior, Fish and Wildlife Service, Washington, D. C. Resource Publication 153.
- Kahn, R. H., and B. A. Millsap. 1978. An inexpensive method for capturing short-eared owls. N. Amer. Bird Bander. 3:54.
- Kalmbach, E. R. 1927. The magpie in relation to agriculture. U. S. Department of Agriculture Technical Report 24:1-29.
- Kaplan, E. L., and P. Meier. 1958. Nonparametric estimation from incomplete observations. J. Am. Stat. Assoc. 53:457-481.
- Kreitzer, J. F., and G. H. Heinz. 1974. The effect of sublethal dosages of five pesticides and a polychlorinated byphenyl on the avoidance response of coturnix quail chicks. Environ. Pollut. 6:21-29.
- Krzystyniak, K., P. Hugo, D. Flipo, and M. Fournier. 1985. Increases susceptibility to mouse hepatitis virus 3 of peritoneal macrophages exposed to dieldrin. Toxicol. Appl. Pharmacol. 80:397-408.
- Mahmood, A., N. Agarwel, S. Sanyal, P. K. Dudeja, and D. Subrahmanyam. 1981. Acute dieldrin toxicity effect on the uptake of glucose and leucine on brush border enzymes in monkey intestine. Chem.-Biol. Interact. 37:165-170.
- Matsumura, F., and S. M. Ghiadsuddin. 1983. Evidence for similarities between cyclodiene type insecticides and picrotoxin in their action mechanisms. J. Environ. Sci. Health, Part B:1-4.
- Mellot, R. S., and P. E. Woods. 1993. An improved ligature technique for dietary sampling in nestling birds. J. Field Ornithol. 64:205-210.
- Mendenhall, V. M., E. E. Klass, and A. R. McLane. 1983. Breeding success of Barn Owls (Tyto alba) fed low levels of DDE and dieldrin. Arch. Environ. Contam. Toxicol. 12:235-240.
- Ohlendorf, H. M., D. M. Swineford, and L. N. Locke. 1981. Organochlorine residues and mortality of herons. Pesticide Monitoring Journal 14:125-135. <u>in</u> DDE, DDT, and dieldrin: residue in American kestrels and relations to reproduction. Wiemeyer, S.N.,

R. D. Porter, G. L. Hensler, and J. R. Maestrelli. 1986. U. S. Fish and Wildlife Service. Fish and Wildlife Technical Report 6. 33 pp.

- Pollack, K. H., S. R. Winterstein, C. M. Bunck, and P. D. Curtis. 1989. Survival analysis in telemetry studies: the staggered entry design. J. Wildl. Manage. 53:7-15.
- Reebs, S. G., and D. A. Boag. 1987. Regurgitated pellets and late winter diet of black-billed magpies, Pica pica, in central Alberta. Can. Field Nat. 101:108-110.
- Reese, K.P., and J. A. Kadlec. 1985. Influence of high density and parental age on the habitat selection and reproduction of black-billed magpies. Condor 87:96-105.
- Reymann, A., W. Brown, and J. Drager. 1983. Effects of DDT and dieldrin on intestinal glucose transport and brush border hydrolases. A comparison with phenobartital and methylcholanthrene. Biochem. Pharmacol. 32:1759-1763.
- Rappole, J. H., and A. R. Tipton. 1991. New harness design for attachment of radio transmitters to small passerines. J. Field. Ornithol. 62:335-337.
- Sharma, R. P., D. S. Winn, and J. B. Low. 1976. Toxic, neurochemical, and behavioral effects of dieldrin exposure in mallard ducks. Arch. Environ. Contam. and Toxicol. 5:43-53.
- Smith, A. G. 1991. Chlorinated hydrocarbon insecticides. Pages 731-916 in W. J. Hayes, Jr. and E. R. Laws, Jr. Eds. Handbook of Pesticide Toxicology, Academic Press, Inc. San Diego, CA.
- Stickel, L. F. 1970. Pesticide residues in birds and mammals. pages 254-312 in C. A. Edwards, ed. Environmental pollution by pesticides. Plenum Press, New York, N. Y.
- Stollar, R. L., Harding Lawson Associates, Ebasco Services Inc., Datachem Inc., Enseco-Cal Laboratories, and Midwest Research Institute. 1990. Comprehensive monitoring program final technical plan-biota. Version 3.1. Prepared for Program Manager's Office Rocky Mountain Arsenal Contamination Cleanup.
- \_\_\_\_\_, Harding Lawson Associates, Ebasco Services Inc., Datachem Inc., Enseco Laboratories, and Midwest Research Institute. 1992. Comprehensive monitoring program, biota annual report for 1990 and summary report for 1988 to 1990. Prepared for Program Manager's Office Rocky Mountain Arsenal Contamination Cleanup. Vol I.
- St. Omen, V. V. 1970. Chronic and Acute toxicity of the chlorinated hydrocarbon insecticides in mammals and birds. Can. Vet. J. 11:215-226.

- Todd, K. S. 1968. Weights of black-billed magpies from south-western Montana. Auk 85 508-510.
- Tordoff, H. B. 1954. An automatic live-trap for raptorial birds. J. Wildl. Manage. 18:281-284.
- Treon, J. E., and F. P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals with special reference to aldrin and dieldrin.
- Tucker, R. K., and M.A. Haegele. 1971. Comparative acute oral toxicity of pesticides to six species of birds. Toxicol. Appl. Pharmacol. 20:57-65.
- Virgo, B. B., and Bellward, G. D. 1973. Effect of dieldrin on mouse reproduction. Proc. Can. Fed. Biol. Soc. 16:103.
- Wiemeyer, S. N., R. D. Porter, C. L. Hensler, and J. R. Maestrelli. 1986. DDE, DDT, and Dieldrin: Residues in American Kestrels and relations to reproduction. U. S. Fish and Wildlife Service, Fish and Wildlife Technical Report 6. 33 pp.
- \_\_\_\_\_, and E. Cromartie. 1981. Relationships between brain and carcass organochlorine residues in ospreys. Bull. Environ. Contam. and Toxicol. 27:499-505.
- Willhite C. and R. P. Sharma. 1978. Acute dieldrin exposure in relation to brain monoamine oxidase activity and concentration of brain serotonin and 5-hydroxyindoleacetic acid. Toxicol. Letters 2:71-75.