

# Pesticides and Birds: From DDT to Today's Poisons

By Caroline Cox

During the three decades that DDT, the organochlorine insecticide that has been called "the most widespread and pernicious of global pollutants,"<sup>1</sup> was used in the United States, its effects on birds were both devastating and notorious. However, the pesticides used in the last twenty years, since DDT's registrations were cancelled in 1972,<sup>5</sup> continue to impact birds. Some are acutely toxic enough that small doses kill birds; others cause a variety of less lethal, but still damaging, effects. Pesticides injure birds both directly and indirectly, and birds are often affected by a combination of different kinds of effects. For the birds' own sake, and because, like the miner's canary, they can warn us when our own health or the health of our ecosystem is threatened, these effects are worth our attention and action.

Concerns about DDT's effects on nontarget organisms, including birds, were first raised shortly after the end of World War II.<sup>2</sup> By the 1960s there was good evidence that living things concentrated DDT and that it was extremely persistent. For example, scientists measured concentrations in gulls, mergansers, and cormorants that were as much as a thousand times higher than the already high concentrations in the mud of the Long Island, New York marsh where the birds fed. The marsh had been sprayed for twenty years as part of a mosquito control program.<sup>3</sup>

These DDT residues caused both acute and chronic health problems. DDT caused direct mortality of some birds by poisoning their nervous system even in birds like robins that feed relatively low on the food chain.<sup>1</sup> (See article beginning on p.) DDT and its metabolites cause eggs to have thin shells and reduce levels of a hormone necessary for female birds to lay eggs.<sup>4</sup> Population declines and local disap-

pearance of peregrine falcons (see article beginning on p.), bald and golden eagles, ospreys, kestrels, and other predatory birds were recorded.

DDT's history certainly paints a frightening picture. But what about the many other pesticides in use today? How do they impact birds? This article is a brief overview of this com-

PELLING SUBJECT.

## **What Types of Pesticides Kill Birds?**

DDT's chemical relatives, the organochlorine insecticides, are still killing birds. In addition, although organophosphate and carbamate insecticides are not as persistent as organochlo-

## **Why Do Pesticide-poisoned Birds Die Out of Sight?**

The number of documented pesticide poisonings of birds is likely to represent only the tip of the iceberg of actual bird kills. The reasons for this are diverse, and worth remembering when documented numbers are small.

There are only a "handful of scientists"<sup>1</sup> documenting pesticide-linked bird kills in the United States. Even those few don't have enough funding to analyze all the birds they receive. It is easy to misdiagnose a bird's cause of death.<sup>1</sup> The most common analysis, inhibition of the enzyme cholinesterase by organophosphate and carbamate insecticides, can be difficult to interpret because normal levels of the activity of this enzyme is not known for many species of birds.<sup>2</sup>

Many bird kills go unnoticed. Even if an attempt is made to find dead birds, many are missed. They can be overlooked in the treated area or may have flown varying distances from the poisoning site before they die. Dying birds may hide, and carcasses can be removed by scavengers or crushed by motor vehicles before they are found. Bacterial decay and insect attack occur rapidly.<sup>1</sup> Birds that are sick, but not dead, may be less likely to be caught for analysis because they are less active or hiding.<sup>3</sup>

Isolated reports of bird kills are seldom sufficient to initiate any

changes in pest management practices; scientific documentation is required. Documentation of pesticides' effects on birds requires carefully designed field studies, which are expensive and time-consuming to conduct. These studies need to be large enough so that there are enough birds to show statistically significant results.<sup>4</sup>

Since a direct census of birds is difficult, singing bird counts are commonly used in these studies as an index of bird abundance. However, birds often decrease their singing as nesting advances, so that if a pesticide causes nest failure, the number of singing birds may actually increase. In addition, some studies of sublethal effects (for example, pesticides causing an increase in the distance that birds had to fly to find food for their nestlings, and increased vulnerability to predators) have shown that singing does not decrease as these sublethal effects increase.<sup>4</sup> Thus, more extensive studies will be required to document non-lethal effects.

1. Stone, W.B. 1987. In the matter of Ciba-Geigy Corp. et al. Unpublished testimony. Delmar, NY: Department of Environmental Conservation, Wildlife Resources Center.
2. Hill, E.F. 1988. Brain cholinesterase activity of apparently normal wild birds. *J. Wildl. Dis.* 24(1):51-61.
3. Busby, D.G., L.M. White, and P.A. Pearce. 1991. Brain acetylcholinesterase activity in forest songbirds exposed to a new method of UULV fenitrothion spraying. *Arch. Environ. Contam. Toxicol.* 20:25-31.
4. Peakall, D.B. and J.R. Bart. 1983. Impacts of aerial application of insecticides on forest birds. *CRC Critical Reviews in Environ-*

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rines because they are more rapidly degraded by light and microbes, their high acute toxicity and widespread use make them a significant problem to birds. Several acutely toxic herbicides also kill birds. A brief survey of bird mortality associated with each of these classes of pesticides follows.

**Organochlorines:** The organochlorine threat to birds did not go away when DDT's registered uses were cancelled. Some organochlorine pesticides (for example, the miticide dicofol which also causes eggshell thinning in birds<sup>6</sup>) are still used in the United States, and uses of others (for example, chlordane) were restricted or cancelled much later than DDT.<sup>5</sup>

In addition, DDT and related compounds are still used in many other countries. Birds that migrate to other countries can bring residues stored in their tissues back to the U.S. For example, analysis of great horned owls for organochlorine residues at the Virginia Wildlife Center found high levels of the DDT metabolite DDE,<sup>7</sup> and smaller amounts of dicofol and methoxychlor<sup>8</sup> (another organochlorine insecticide still registered for use in the U.S.). Although the owls do not migrate, the organochlorine residues could have come from the birds' migratory prey, persistent residues from previous years carried by the long-lived birds, or from current uses of dicofol and methoxychlor. It is not possible to determine the source.<sup>8</sup>

**Organophosphates:** Organophosphate insecticides inhibit an enzyme (acetylcholinesterase) essential for proper functioning of the nervous system. Because we all have similar mechanisms of nerve transmission, this mode of action is similar in target insects, birds, and mammals. Many organophosphates are acutely toxic to birds at very low doses. A recent compilation of acute lethal doses (LD<sub>50</sub>S\*) for the mallard duck showed that 16 of 20 organophosphates were acutely toxic at doses less than 20 milligrams

per kilogram (mg/kg) of body weight and the most toxic had an LD<sub>50</sub> over twenty times smaller.<sup>7</sup>

Well documented bird kills have been caused by the organophosphates diazinon, isofenphos, and chlorpyrifos with one kill involving thirty to forty thousand birds.<sup>9,10,11</sup> A review of aerial forestry applications showed that all four organophosphates reviewed, phosphamidon, fenitrothion, acephate, and trichlorofon, caused reductions in the abundance of singing males, the number of birds present, or the number of species present.<sup>12</sup>

**Carbamates:** Carbamate insecti-

study of effects on bird populations following a forestry application found that both the abundance of birds and the number of bird species present was reduced by aerial spraying of carbaryl, and these effects persisted until the summer following the spray. Although other studies of forestry carbaryl applications found no significant effects on birds, reviewers noted that this study lasted longer and used larger spray blocks than the studies that found no effects.<sup>12</sup>

**Herbicides:** While it might be expected that herbicides in general are less acutely toxic to birds (and other



Laughing gull.

cides have a mode of action similar to the organophosphates and, like the organophosphates, some kill birds at low doses. Carbofuran, which has been estimated to kill one to two million birds annually in the U.S.,<sup>13</sup> is probably the best known example. (See article beginning on p. .)

Granular formulations, in which the pesticide is incorporated with a carrier into small particles slightly larger than those found in granulated sugar,<sup>14</sup> have been particularly hazardous to birds. Over half of house sparrows fed granular formulations of three carbamates (aldicarb, carbofuran, and bendiocarb) were killed by ingestion of just one granule and only five to ten granules killed red-winged blackbirds.<sup>15</sup>

Another carbamate, carbaryl (Sevin), has an acute toxicity to birds several orders of magnitude lower (about 2000 mg/kg).<sup>16</sup> However, a

animals) than insecticides, some herbicides are lethal to birds in small doses. Dinoseb, a dinitrophenol herbicide that interferes with the basic energy metabolism in both plant and animal cells,<sup>17</sup> kills wild birds at doses of 7 mg/kg<sup>18</sup> and is as acutely toxic to birds as some of the most toxic insecticides.

Paraquat, another herbicide that is highly toxic to humans and animals,<sup>19</sup> kills adult birds at several hundred milligrams per kilogram,<sup>18</sup> but can kill nestling kestrels at doses almost as low as dinoseb's lethal doses. Some nestlings died after being fed paraquat at 10 mg/kg, which is one-third of the LD<sub>50</sub> for humans.<sup>20</sup> At least one documented bird kill has occurred following agricultural use of paraquat.<sup>21</sup>

The herbicide DNOC (dinitro-oresol), also used as an insecticide, fungicide, and defoliant,<sup>14</sup> is moderately toxic to birds. Pheasants, a jack-

\* The LD<sub>50</sub> is the dose of a chemical that kills 50 percent of a population of test animals.

daw, a skylark, and wood pigeons were found dead in Great Britain following use of DNOC on wheat and corn fields.<sup>22</sup>

### **Indirect Effects: Secondary Poisoning of Predatory Birds**

Stories of birds killed by DDT not directly, but indirectly by consuming prey that contained high residues of the insecticide, were common when DDT was in frequent use. However, similar situations also exist with other insecticides.

Some of the best information about this kind of poisoning comes from studies of pour-on organophosphate insecticides (famphur, for example) used to kill warbles, flies that live just under the skin of cattle. Famphur poured onto cattle was shown to cause subsequent poisoning of magpies, birds that feed on cattle hair for part of their diet. In addition, researchers found three red-tailed hawks (two dead) that had been poisoned by the famphur after eating poisoned magpies.<sup>23</sup> A decline in magpie populations throughout the western United States was correlated in time with the widespread use of famphur. Similar secondary poisonings have also been reported for barn owls, great horned owls, and bald eagles.<sup>24-26</sup>

Another organophosphate insecticide, parathion, can kill American kestrels that feed on frogs raised in water containing the insecticide.<sup>27</sup> Similar problems may occur with other organophosphates.

### **Indirect Effects: Starvation**

Broad spectrum pesticides can also kill or injure birds by depriving them of their usual source of food. For example, synthetic pyrethroids (which, as a group have a relatively low acute toxicity to birds) can destroy birds' food supplies. Waterfowl that feed on aquatic insects, small insectivorous birds, and nestlings fed on insects are especially vulnerable.<sup>28</sup>

Forestry application of fenitrothion, an organophosphate insecticide, caused a decrease of almost one-third in the weight of insects available for birds to eat.<sup>29</sup>

In addition, organophosphate insecticides are known to cause anorexia (loss of appetite) in birds. The resulting starvation can be an important cause of death.<sup>30</sup> Grackles fed lethal doses of any of four different organo-

phosphates (dicrotophos, fenitrothion, fenthion, and methyl parathion) lost over 25 percent of their body weight before death in a laboratory study. The researchers noted that several large bird kills observed in migrating birds are probably associated with this anorexia occurring at a time when most of the birds' fat reserves have been used.

Herbicides can indirectly cause birds' starvation or force them to leave treated areas because the herbicides destroy the habitat used by the birds' prey. In Maine forests, a study of bird and insect populations following application of the herbicide glyphosate to clearcuts showed that both birds

“A decline in magpie populations throughout the western United States was correlated in time with the widespread use of famphur.”

and insects were less abundant in treated areas. The abundance of insects remained low for three years after the herbicide treatment.<sup>31,32</sup>

### **Indirect Effects: Predation**

In a laboratory study using a house cat as a predator, bobwhite quail dosed with the organophosphate insecticide methyl parathion were more susceptible to predation than were untreated quail. Treated birds spent more time standing still than untreated birds.<sup>33</sup> In addition, quail dosed with sublethal doses of methyl parathion had lower survival under field conditions due to an increase in predation.<sup>34</sup>

### **Sublethal Effects**

In doses that do not kill, pesticides cause a myriad of adverse effects on the health of birds. These can include a reduction in the amount of food consumed, loss of weight, changes in physical activity, and a decrease in the production, fertility, or hatchability of eggs. A few examples follow.

- DDT, in addition to its other ef-

fects, has been shown to feminize the development of embryos in contaminated gull eggs. The feminized gulls may alter reproductive behavior and explain the skewed sex ratios and reduced numbers of breeding males found in a California gull population.<sup>35</sup>

- Parathion stops egg production in quail, and methyl parathion affects the duration of ovulatory cycles.<sup>36</sup> Both of these insecticides have also been shown to lower birds' tolerance to cold.<sup>37,38</sup>

- Dicrotophos has been shown to disrupt parental care of starling nestlings. A single oral dose resulted in females making fewer trips to feed their young and increased the amount of time that the mothers spent away from their nests. As a result, nestlings lost weight following the dicrotophos treatment.<sup>39</sup>

- Carbaryl causes changes in the stride of chicks at levels below which any reduction of their brain cholinesterase levels can be measured. Effects on the chicks' strides could be measured up to 40 days after the last dose of carbaryl was given. The chicks also suffered from enlarged livers.<sup>40</sup>

### **Combined Effects**

When birds in the wild are harmed by pesticides, acute mortality, indirect impacts, and sublethal effects occur together in a multi-faceted combination. The resulting stories are certainly not pretty, but offer a fascinating glimpse in the complex and pervasive ways that pesticides can effect the ecosystem.

**Laughing Gulls and Parathion:** In one well-documented incident, a breeding colony of laughing gulls near Corpus Christi, Texas, was poisoned by a parathion application made to kill bollworms on a cotton field about three miles from the gull colony. Over a hundred dead adult birds were found on the islands where the gulls nest, and about the same number on the banks of a pond adjacent to the treated cotton field. An estimated 25 percent of the colony's chicks also died. The adults were killed by ingestion of poisoned insects from the cotton field; insect parts in the intestinal tracts of the birds contained poisoned residues and brain acetylcholinesterase activity was less than half of healthy birds. The chicks appeared to die of a combination of causes, however. About half of a sample of dead

chicks also contained poisoned insects (fed to them by their parents); the remainder showed no signs of parathion poisoning, but appeared to have died from starvation or neglect following their parents' death.<sup>41</sup>

Subsequent research showed that sublethal exposure to parathion reduced the amount of time that parent gulls spent on their nests. The researchers concluded that chicks with less attentive parents could be more susceptible to predation or egg failure.<sup>42</sup> This means that even nonlethal doses of parathion could result in the death of chicks.

### Golden-cheeked Warblers and



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**Malathion:** The U.S. Animal and Plant Health Inspection Service (APHIS) is currently conducting a program in Guatemala to eradicate the Mediterranean fruitfly (medfly). The medfly was first introduced into Central America in 1955 and has been present in Guatemala since 1975. It has been the subject of proposed and operational eradication programs in the U.S. (including Hawaii) and Mexico since 1979.<sup>43</sup>

The current program in Guatemala<sup>43</sup> involves both aerial and ground spraying of baited malathion (an organophosphate insecticide) in areas that include the winter habitat of a bird that was listed as an endangered species in the United States in June 1990, the golden-cheeked warbler.<sup>44</sup> Only several hundred breeding pairs of the warbler survive, and their summer breeding range is in Texas.<sup>45</sup>

The Environmental Defense Fund

(EDF) has notified USDA that they believe the agency is in violation of the Endangered Species Act because the environmental analysis written by APHIS for the eradication project did not adequately consider the impacts on the warbler. EDF noted that a variety of impacts on the warbler were possible. First, APHIS had assumed in their calculations of potential impacts on birds that the birds' only exposure to malathion came from eating poisoned insects. APHIS did not include exposure through preening and direct contact with the birds' feet. In addition, APHIS did not consider the cumulative impact of the eradication

program's pesticides and other pesticide use in Guatemala. Malathion is used agriculturally (on coffee plantations) in the area, as well as are other potentially harmful pesticides including aldicarb and paraquat. APHIS considered only the potential for direct mortality and did not consider sublethal effects that might make it difficult for the bird to survive its return migration to Texas. For example, insect abundance is reduced by the malathion. Although APHIS believes that this reduction is unlikely to be long-lasting, recovery of insect abundance will not help the warblers if they have already left on their northward migration.<sup>44</sup>

The benefit of medfly eradication cited by APHIS is that the acreage of cropland in Guatemala will be expanded. This too could have an indirect adverse impact on the warbler,

## Regulatory Issues

There are several steps that can be taken to reduce pesticides' impacts on birds while alternative pest management are being implemented. These include the following:

- The current regulatory process uses several species of birds (mallard ducks and quail) as test species for determining the acute toxicity of pesticides. These birds are not as susceptible to many pesticides as other species.<sup>1</sup> In addition, there is very little testing of chronic, indirect, and sublethal toxicity. All of this information is vital if we are to accurately assess what the impact of a pesticide on birds will be.

- Most directions for applying pesticides specify the rate at which the pesticide is to be used without regard to whether the chemical might be applied in combination with other pesticides. When two or more pesticides are applied together that have similar effects on birds, birds can receive a multiple dose. For example, 160 Canada geese died in the Snake River (Oregon) after three cholinesterase-inhibiting insecticides were applied to a field of alfalfa. Although application rates for each chemical separately were below recommended rates, in combination they had a devastating effect on geese.<sup>2</sup> Such problems will continue until more consideration is given to cumulative and synergistic effects.

- Effects on birds need to assume greater importance in the regulatory process as a whole. When EPA cancelled registrations of the organophosphate insecticide diazinon on golf courses and turf farms in 1990, it was "the first EPA action to cancel a pesticide registration based solely on risk to birds."<sup>3</sup> This is a dismal record indeed.

1. Porter, Stuart, toxicologist, Wildlife Center of Virginia. Personal communication. October 9, 1991.
2. Blus, L.J. 1991. Canada goose die-off related to simultaneous application of three anticholinesterase insecticides. *Northwestern Naturalist* 72:29-33.
3. Wolfson, Steve, attorney, U.S. EPA Office of General Counsel. Letter to Mary O'Brien, NCAP staff scientist. July 19, 1990.

as its primary habitat is forest. For example, APHIS believes that dates could be grown as a export crop in Guatemala if the medfly is eradicated. This would probably result in a loss of warbler habitat.<sup>44</sup>

Interestingly, the extinction of an endemic honeycreeper on the Hawaiian island of Lanai occurred at the time of a baited malathion spray during the 1970s.<sup>46,47</sup> Although evidence from Hawaii is mostly anecdotal, it would be very difficult to document this kind of effect.

### **Pesticides: Not for the Birds!**

Pesticides will continue to kill birds, reduce their food resources, and disrupt their normal behaviors as long as pesticides continue to be used. The only way to eliminate the effects that pesticides have on birds is to use nonchemical resource management techniques. On farms, in forests, on lawns, and elsewhere that pesticides are used, managers are finding that these techniques work well and make economic sense. Our job is to see that they are implemented more widely. This is not a simple task, but one that is essential if we are to seriously heed the message of our miners' canaries. ■

### **References**

- Herman, S.G. and J.B. Bulger. 1979. Effects of a forest application of DDT on nontarget organisms. *Wildlife Monographs* No. 69 (October).
- Cottam, C. and E. Higgins. 1946. DDT: Its effect on fish and wildlife. *U.S. Fish Wildl. Serv. Circ.* 11. 14 pp. Cited in reference #1.
- Woodwell, G.M. 1967. Toxic substances and ecological cycles. *Scientific American* 216(3):24-31.
- Peakall, D.B. 1970. Pesticides and the reproduction of birds. *Scientific American* 222(4):73-78.
- U.S. EPA. Office of Pesticides and Toxic Substances. Office of Compliance Monitoring. 1990. *Suspended, cancelled, and restricted pesticides*. 20T-1002. Washington, D.C. (February).
- Bennett, J.K., S.E. Dominguez, and W.L. Griffis. 1990. Effects of dicofol on mallard eggshell quality. *Arch. Environ. Contam. Toxicol.* 19(6):907-912.
- Porter, S.L. 1991. Pesticide poisoning in birds of prey. Unpublished manuscript. Weyer's Cave, VA: Wildlife Center of Virginia.
- Clark, Ed, Wildlife Center of Virginia. Personal communication. September 30, 1991.
- Stone, W.B. 1985. Wildlife mortality related to the use of the pesticide diazinon. 1985 update — by state/province. Unpublished report. Delmar, NY: Department of Environmental Conservation, Wildlife Resources Center
- Stone, W.B. 1987. In the matter of Ciba-Geigy Corp. et al. Unpublished testimony. Delmar, NY: Department of Environmental Conservation, Wildlife Resources Center.
- Stone, W.B. 1989. Wildlife mortality related to the use of diazinon, chlorpyrifos, isofenphos and bendiocarb. 1987-1989. Unpublished report. Delmar, NY: Department of Environmental Conservation, Wildlife Resources Center
- Peakall, D.B. and J.R. Bart. 1983. Impacts of aerial application of insecticides on forest birds. *CRC Critical Reviews in Environmental Control* 13(2):117-165.
- U.S. EPA. Office of Pesticides and Toxic Substances. 1989. Carbofuran: A special review technical support document. Washington, D.C.
- 1991 *Farm Chemicals Handbook*. Willoughby, OH: Meister Publishing Co.
- Balcomb, R. R. Stevens, and C. Bowen. 1984. Toxicity of 16 granular insecticides to wild-caught songbirds. *Bull. Environ. Contam. Toxicol.* 33:302-307.
- U.S. EPA. Office of Pesticides and Toxic Substances. Office of Pesticide Programs. 1984. Pesticide fact sheet (carbaryl). No. 21. Washington, D.C.
- U.S. EPA. Office of Pesticides and Toxic Substances. Office of Pesticide Programs. 1986. Pesticide fact sheet (dinoseb). No. 130. Washington, D.C.
- National Institute for Occupational Safety and Health. 1991. *Registry of toxic effects of chemical substances*. Microfiche edition. D.V. Sweet, ed. Cincinnati, OH. (January).
- U.S. EPA. Office of Pesticides and Toxic Substances. Office of Pesticide Programs. 1987. Pesticide fact sheet (paraquat). No. 131. Washington, D.C.
- Hoffman, D.J., J.C. Franson, O.H. Pattee, and C.M. Bunck. 1985. Survival, growth, and histopathological effects of paraquat ingestion in nestling American kestrels. *Arch. Environ. Contam. Toxicol.* 14: 495-500.
- Rivera, M. 1973. Diagnosis of geese poisoning with gramoxone. *Rev. Cuban Farm.* 7(1): 65-70. Cited in U.S. EPA. Office of Pesticides and Toxic Substances. 1982. Paraquat decision document. Washington, D.C. (July 1).
- Rudd, R.L. and R.E. Genelly. 1956. *Pesticides: Their use and toxicity in relation to wildlife*. Game Bulletin No. 7. Sacramento, CA: Department of Fish and Game. Game Management Branch.
- Henny, C. J. et al. 1985. Organophosphate insecticide (famphur) topically applied to cattle kills magpies and hawks. *J. Wildl. Manage.* 49(3): 648-658.
- Hill, E.F. and V.H. Mendenhall. 1980. Secondary poisoning of barn owls with famphur, an organophosphate insecticide. *J. Wildl. Manage.* 44(3):676-681.
- Franson, J.C., E.J. Kolbe, and J.W. Carpenter. 1985. Famphur toxicosis in a bald eagle. *J. Wildl. Dis.* 21(3):318-320.
- Hill, E.F. research toxicologist, Patuxent Wildlife Center. Letter to Charles Erikson, Center for Veterinary Medicine, Food and Drug Administration. March 14, 1986.
- Fleming, W.J. et al. 1982. Parathion accumulation in cricket frogs and its effect on American kestrels. *J. Toxicol. Environ. Health* 10:921-927.
- Mueller-Beilschmidt, D. 1990. Toxicology and environmental fate of synthetic pyrethroids. *J. Pesticide Reform* 10(3):32-36.
- Millikan, R.L. 1990. Effects of fenitrothion on the arthropod food of tree-foraging forest songbirds. *Can. J. Zool.* 68:2235-2242.
- Grue, C.E. 1982. Response of common grackles to dietary concentration of four organophosphate pesticides. *Arch. Environ. Contam. Toxicol.* 11:617-626.
- Santillo, D., P. Brown, and D. Leslie. 1989. Responses of songbirds to glyphosate-induced habitat changes on clearcuts. *J. Wildl. Manage.* 53(1):64-71.
- Santillo, D., D. Leslie, and P. Brown. 1989. Responses of small mammals and habitat to glyphosate application on clearcuts. *J. Wildl. Manage.* 53(1):164-172.
- Galindo, J.C., R. J. Kendall, C.J. Driver, and T.E. Lacher. 1985. The effect of methyl parathion on susceptibility of bobwhite quail (*Colinus virginianus*) to domestic cat predation. *Behav. Neur. Biol.* 43:21-36.
- Buerger, T.T. 1991. Effects of methyl parathion on northern bobwhite survivability. *Environ. Toxicol. Chem.* 10:527-532.
- Fry, D.M. and Toone, C. K. 1981. DDT-induced feminization of gull embryos. *Science* 213:922-924.
- Rattner, B.A. et al. 1984. Avian endocrine responses to environmental pollutants. *J. Exp. Zool.* 232:683-689.
- Rattner, B.A. and J.C. Franson. 1984. Methyl parathion and fenvalerate toxicity in American kestrels: acute physiological responses and effects of cold. *Can. J. Physiol. Pharmacol.* 62:787-792.
- Rattner, B.A., L. Sileo, and C.G. Scanes. Hormonal responses and tolerance to cold of female quail following parathion ingestion. *Pest. Biochem. Physiol.* 18:132-138.
- Grue, C.E., G.V.N. Powell, and M.J. McChesney. Care of nestlings by wild female starlings exposed to an organophosphate pesticide. *J. Appl. Ecol.* 19:327-335.
- Fargae-Elawar, M. 1989. Enzyme and behavioral changes in young chicks as a result of carbaryl treatment. *J. Toxicol. Environ. Health.* 26:119-131.
- White, D.H., et al. 1979. Parathion causes secondary poisoning in a laughing gull breeding colony. *Bull. Environ. Contam. Toxicol.* 23:281-284.
- White, D.H., C.H. Mitchell, and E.F. Hill. 1983. Parathion alters incubation behavior of laughing gulls. *Bull. Environ. Contam. Toxicol.* 31:93-97.
- Higgins, M.L. et al. 1987. Scope of work for an environmental assessment of the USDA Guatemala medfly eradication program. (February). U.S. Agency for International Development.
- Bean, Michael J., senior attorney, Environmental Defense Fund. Letter to Manuel Lujan, secretary, U.S. Department of the Interior, and Edward Madigan, secretary, U.S. Department of Agriculture. May 21, 1991.
- Bean, Michael J., senior attorney, Environmental Defense Fund. Personal communication. October 4, 1991.
- Dreistadt, S.H. and D.L. Dahlsten. 1986. Lessons from the field: Medfly eradication in California. *Environment* 28(6):18-44.
- Gagné, Wayne. 1984. Testimony on a draft environmental impact statement, Eradication of the tri-fly complex from the state of Hawai'i. Honolulu, HI. December 18.