Insecticides and Wildlife[1]

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Chemical insecticides (often called 'pesticides') are viewed by some as a boon (a blessing) and others as a bane (a problem or fatal poison), and both views are correct. Most people don't like insects and other invertebrate animals, so they would like all invertebrate animals to die or at least disappear from view. However, when we use chemical insecticides to eliminate insects, we discover that the effects of insecticides are not always limited to these 6-legged targets. Vertebrate animals also can be affected by insecticides, and this includes wildlife, pets, and humans. So, one of the great, inadequately met challenges confronting humans is to develop ways to manage insects without affecting non-target organisms, especially vertebrate animals such as wildlife and humans. The purpose of this article is to provide information on pesticides, how they work, and the negative impacts they can have on wildlife. It is intended for anyone who uses or is interested in pesticides.

At this point, 'wildlife' needs to be defined. The American perspective on 'wildlife' tends to be limited mostly to vertebrate animals, namely amphibians, reptiles, birds, mammals, and perhaps fish. In contrast, most of the rest of the world recognizes invertebrates (insects, worms, snails, etc.) to be components of 'wildlife'. For the purpose of this discussion, however, we will consider wildlife to consist of wild vertebrate animals rather than invertebrates.

Why Pesticides Are Used

When confronted by a pest situation, the use of pesticides to solve the 'problem' is often the easy answer. Although most people will readily embrace the idea of using non-pesticidal means of pest control, in the final analysis pesticides tend to be chosen. Why are pesticides used so often? The decision is due to several factors, including:

- the lack of alternatives to pesticides;
- use of pesticides may be the least expensive or most convenient technique;
- pesticides may be the most reliable technique, serving to minimize risk and pest-related problems; and
- due to failure to act in a timely manner to prevent a problem from developing, certain alternatives to pesticides cannot be used.

Pesticides often are the most convenient, economic, effective, and reliable technique for pest suppression once the pest situation is out-of-hand (i.e., pests or their damage are so abundant as to cause nuisance or economic loss). However, pesticides sometimes cause serious problems with wildlife conservation, so it may be best to minimize pesticide use or use them carefully or selectively. Thus, it is important to know the characteristics and limits of pesticides so they can be used most appropriately and most safely.

What is a pesticide?

Toxicants that are designed to kill pests are called **pesticides**, though pesticides also can affect wildlife, which we do not generally consider to be pests. Pesticides are used to suppress pests such as insects, plant diseases, weeds, or vertebrates that cause damage or are a health risk or nuisance.

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Among the many types of pesticides are **herbicides** (used to kill weeds), **insecticides** (for insects), **fungicides** (for fungi), **rodenticides** (for rats and mice), **acaricides** (for mites and ticks), **bactericides** (for bacteria), **nematicides** (for nematodes), **molluscicides** (for snails and slugs), **avicides** (for pest birds), **piscicides** (for pest fish), and **algaecides** (for algae). Sometimes pesticides are also used to kill vertebrate animals such as coyotes, *Canis latrans*, or prairie dogs, *Cynomys* spp. Most often, the pesticide of concern to vertebrates is an insecticide, although sometimes herbicides and fungicides can cause problems for wildlife. Less often, molluscicides and rodenticides are encountered by wildlife. In any event, the term 'pesticide' actually suggests control of more than just insect pests, despite our tendency to equate 'pests' with 'insects'.

Characteristics of Pesticides

The ability of a pesticide to cause injury or death is called **toxicity**. The active ingredient causing death is called the **toxicant**, and the toxicant typically comprises only a small proportion of the formulation that is purchased and applied. The toxicant usually is applied after mixing with water, which is an inexpensive and convenient method of diluting the toxicant to the desired concentration.

Toxicity is dose dependent. Low levels of pesticide may be eliminated (metabolized or excreted) by an animal from its body without suffering harm, or they may have no measurable effect on the animal. High levels of toxin exposure, of course, are more likely to cause injury. Interestingly, most people fail to appreciate this dose phenomenon, instead either treating all pesticides callously, as if they were not a risk, or overreacting to a perceived risk, as if all pesticides and all exposure rates were extremely hazardous. The best approach is to treat pesticides as if they were prescription drugs: relatively safe if used according to directions, and hazardous if misapplied. As with prescription drugs, there is some variation among individuals or among species in terms of susceptibility or adverse reaction.

Another term often used in association with pesticides is hazard. **Hazard** is the likelihood that a toxicant will cause injury to a non-target organism. Hazard is a function not only of toxicity, but also dose of the toxicant, length of exposure, and method of application. Thus, very toxic products can be applied safely if efforts are made to minimize hazard. Likewise, products with relatively low toxicity can be hazardous if used inappropriately. For example, if an insecticide that is toxic to pollinators is applied to a flowering crop during the day, high mortality to insect pollinators is probable. However, if the same insecticide is applied at dusk when pollinators are inactive, much less mortality to pollinating insects is likely because the product would be partly degraded by light and foliar pH before pollinators became fully active on the following day.

Figure 1. A bird killed following application of a highly toxic insecticide. Animal cadavers are a prized resource for carnivores and scavengers, so do not persist for very long in nature. Thus, the impact of highly toxic pesticides can be underestimated because there is little evidence of animal death.

How do we measure toxicity?

Toxicants vary greatly in their ability to injure wildlife (and humans), and it is always a good idea to be familiar with the toxicity level of any pesticide that you handle or apply. Toxicants are sometimes grouped into categories (see Table 1), with category I pesticides being the most toxic and category IV being the least toxic. Category I chemicals are quite hazardous, and you should avoid them unless you have training on how to handle these materials. To purchase category I chemicals, you must have a restricted use pesticide applicator license. With most category II products, in contrast, farmers and homeowners and others without special knowledge or a license can purchase these from agricultural supply centers, garden centers, and hardware or discount stores without restriction. Category II chemicals, though readily accessible, should be treated with respect because they are capable of harming humans and wildlife. Category III and IV pesticides are less hazardous than the preceding categories, and of course they are readily available, but they also may cause injury and death if misused or misapplied. If use of a pesticide is necessary, it is advisable to use category III and IV pesticides, if they will be effective, based on safety considerations.

Another way to assess toxicity is to obtain the LD_{50} value. The **LD_{50} value** is the dose of active ingredient, expressed in milligrams (mg) of toxicant per kilogram (kg) of test animal, that will kill 50% of the test subjects. Products with a low LD_{50} value are more toxic than those with a high

value. Expressing toxicity in this manner adjusts for different sizes (weights) of subjects. This is necessary because a small organism will be more easily killed by a certain dose of toxicant than a large organism that is exposed to the same dose. The route of exposure is also important, with oral (ingestion) being a much more hazardous route of exposure than dermal (skin) exposure. For pesticides that might be inhaled, a slightly different measure of toxicity is used, the **LC_{50} value**; this represents the concentration of toxicant (in milligrams per liter) that induces mortality in 50% of the test subjects.

It may be difficult to obtain detailed toxicity data for some pesticides. One good source of toxicity, handling, and disposal information is the **MSDS (Material Safety Data Sheet)**. The MSDS accompanies all extremely hazardous (restricted use) materials, but also is generally accessible from internet (WWW) databases for all toxicants and hazardous materials. Also, toxicity levels for most products are indicated by signal words found on the label. The **signal word** indicates the degree of hazard, though unfortunately it does not correspond directly to the aforementioned toxicity categories. The signal words are DANGER POISON for toxicity category I, WARNING for toxicity category II, and CAUTION for both categories III and IV.

Pesticides vary in their ability to affect wildlife. The short-term risk of poisoning, or **acute toxicity**, can be quite different than the risk resulting from prolonged exposure, or **chronic toxicity**. Acute toxicity is mortality resulting from dermal contact, inhalation, or ingestion, and typically occurs soon after exposure to recently treated yards, crops, or forests. Chronic toxicity often is a more subtle disruption resulting from alteration of physiological processes and is manifested by changes in hormone levels, immune responses, reproduction, and behavior. Chronic toxicity typically occurs only after prolonged exposure to low levels of toxicant.

In general, herbicides and fungicides are considerably less toxic to wildlife than insecticides (Table 2) and rodenticides. However, some herbicides and fungicides, while not displaying high levels of acute toxicity, are suspected of being toxic when they move into the water supply or food chain and are ingested over long periods of time. Sometimes there are interactions resulting in greater toxicity of insecticides when other pesticides, including herbicides, are present in the environment. In other words, the presence of one toxin exacerbates (synergizes) the actions of another. Because most wildlife poisonings involve insecticides, we will focus mostly on toxicants directed to insects. Toxicants occur naturally in the environment of people and wildlife, and it is easy to become unduly alarmed or overly cautious with respect to toxicants. Many of the items that we are exposed to on a daily basis are toxic too, and in some cases they are more toxic than pesticides. For perspective, consider that the oral LD_{50} value for caffeine is 192 mg/kg, for acetaminophen (Tylenol) it is 338, for ammonia it is 350, for bleach it is 800, and for table salt it is 3000.

How Insecticides Work

Pesticides have various ways of killing pests. The method of killing, or **mode of action**, depends on the nature of the pest organism and the physiological system within the pest that has been targeted for interference or destruction. Some pesticides are quite selective; some insect growth regulators, for example, affect insects as they molt from stage to stage by interfering with their developmental processes (usually hormones or molting). Vertebrate animals and plants lack many of the developmental systems or physiological processes found in insects, so they are essentially immune to such growth regulator-based insecticides. Thus, some insecticides are considered to be quite selective, and not too hazardous to the environment. They are not perfectly benign, however, because some close relatives of insects such as crustaceans (e.g., shrimp, crabs) can be affected and because beneficial, non-target insects may also be present.

The mode of action of most insecticides can be described as **nerve poisons** because they interfere with normal functioning of the nervous system. The specific mode of nerve inhibition varies among the groups of insecticides, but among the organophosphates and carbamates the enzyme acetylcholinesterase is inhibited, resulting in excessive neuroexcitation (often expressed as twitching and lack of coordination) in insects. Another common form of toxicity is caused by disruption of nerve axon function due to influx of sodium or chloride ions. As with acetylcholinesterase inhibitors, most insecticides causing disruption of nerve axon function also cause overexcitation. The nerves of both vertebrate and invertebrate animals function similarly, though not identically, so insecticides can pose a risk to wildlife if they are applied in an environment where wildlife occur. If vertebrates are poisoned by these products, we typically see excessive salivation and tearing, tremors, convulsions, difficulty in breathing, vomiting, diarrhea, and paralysis in the affected animal. Some of the newer classes of nerve toxins take advantage of the differences that exist between the nerves of vertebrates and insects, providing an important measure of safety.

Within the many classes of insecticides (Table 3), several aspects of nerve transmission are affected, and most classes

of insecticides target the nervous system. However, despite their apparent similarities, insecticides differ greatly in persistence, with newer products usually being less persistent and much less likely to accumulate in animals and ecosystems. Also, sometimes the individual products are more or less hazardous to categories of wildlife such as fish or birds, so it is worth investigating the environmental hazards in advance of applying insecticides. Many of the newer products are fairly selective, presenting less hazard to wildlife, though fish often are especially susceptible.

The persistent organochlorines, which were responsible for some serious wildlife problems in the 1950s and 1960s, have been largely eliminated from use. The organophosphates and carbamates, which lack the persistence of the organochlorines but often cause acute poisoning in wildlife, are also becoming less common. The pyrethroid insecticides are much less hazardous to most wildlife, though fish are quite susceptible. Also, although pyrethroids cause little direct toxicity to vertebrate animals, they can be so effective at killing insects as to potentially induce mortality by depriving wildlife of food; this is called **indirect mortality**. With the newest classes of insecticides, such as nicotinoids and spinosyns, the direct hazards to wildlife are minimized, though indirect effects such as food deprivation remain an issue.

The toxicity of some insecticides is shown in Table 4. Note that the toxicity varies considerably among classes of insecticides. The organophosphates and carbamates contain some very toxic materials when they are compared to the pyrethroids. However, even the pyrethroids seem toxic when compared to the insect growth regulators and some of the so-called 'natural products' which are mostly botanical insecticides but also include a toxin-containing microorganism (*Bacillus thuringiensis*) and a chemical product of soil-dwelling microbes (spinosyns). Also, though not apparent from these data, many pyrethroids are particularly harmful to fish. The toxicity of products within a class of insecticide can also vary. Comparing aldicarb to carbaryl, for example, shows that there is greater than a 500-fold difference in toxicity even within a single class of chemicals. Thus, it is important to know the toxicity of individual products, and to make no assumptions about insecticide safety based on the chemical class. It is also important to note that it is not safe to assume that natural products are inherently safer. *Bacillus thuringiensis* and petroleum oils are practically non-toxic to test mammals, whereas some plant-derived compounds such as nicotine rank as fairly toxic compounds.

Generally, insecticides and fungicides do not affect plants. Nor do herbicides generally have strong effects on insects and plant disease, or fungicides affect insects (see Table 2). There are exceptions, of course, as there are some plants or even varieties of plants that are sensitive to certain chemicals, including the adjuvants mixed with the toxicants, making them unsafe to use in certain situations. A few products are **biocides**, chemicals designed to kill all life. Why would anyone want to kill all life? Well, biocides can be useful when you are trying to be absolutely sure that products such as furniture or grain that are being imported are free of exotic or dangerous organisms. Most biocides are fumigants that are applied to structures to eliminate termite infestations, or to structures containing stored grain. The principal out-of-doors application of biocides is soil fumigation, wherein fumigant is injected into the soil to kill insects, nematodes, plant pathogens, and weed seeds that threaten a crop. So even though the fumigation occurs out-of-doors, the toxicant is largely retained in the soil, and the toxicity is localized. Needless to say, biocides can be a risk to wildlife if animals are found in these situations, though they typically are not, and the toxicity of the fumigant dissipates quickly. Thus, the hazard to wildlife comes principally from insecticides, or other chemicals directed at animals (nematodes, molluscs, rodenticides), but not often from pesticides directed at plants or plant disease.

How Wildlife Come into Contact with Insecticides

Wildlife can come into contact with insecticides in many ways, including:

- when they walk on plants or soil that contains pesticides;
- when aquatic organisms are immersed in water that contains a high concentration of pesticide;
- when they consume plants, water, or insects contaminated with pesticides;
- when they inadvertently consume pesticide-treated insect baits or granular insecticides; and
- when they are sprayed directly by aqueous formulations of pesticide.

Insecticides that cause toxicity only after they have been eaten are called **stomach poisons**. Those that can affect the insect by external exposure (as by walking on the insecticide residue) are called **contact poisons**. Most modern insecticides are both contact and stomach poisons. Some water-soluble products can be absorbed into the host plant (or animal) and be moved around, contacting the insect wherever it feeds. When applied to soil or plants, these

systemic pesticides normally are taken up into the plant through the roots or foliar tissue and move mostly upwards (to the actively growing regions). Systemic pesticides can be translocated in the water-conducting (xylem) and food-conducting (phloem) systems of a plant, but especially the xylem. Insects that feed on vascular tissues such as xylem or phloem (insects with piercing-sucking mouthparts) tend to concentrate the insecticide and are especially likely to be poisoned. However, some pesticides move only short distances, as from one side of a leaf to another; these are said to be **translaminar**. Inhalation of toxicants through the insect's ventilatory system is also possible, though not especially common, and such products are called **fumigants**.

Depending on the toxicity of the product and the level or duration of contact, vertebrate animals may be unaffected, sickened, or killed following such exposure. Other variables affecting susceptibility to being poisoned include the size, age, and health of the animal. Generally, it is the small, young, very old, or physiologically stressed individuals that are most likely to be affected.

We tend to think of pesticide applications as involving application of liquid formulations, commonly called 'sprays'. Indeed, this is the most common means of pesticide application. However, some products are not applied in a water-based spray, especially the pesticides aimed at vertebrate pests. Often these are applied as baits, which usually are a solid material, but even these usually have been pretreated with a liquid formulation of pesticide during the preparation of the bait. Bait formulations are also directed at some arthropods and mollusks, especially ants, cockroaches, grasshoppers, crickets, mole crickets, earwigs, silverfish, sowbugs, and terrestrial slugs and snails. The other common non-liquid formulations are granules and dusts. Granules are small pesticide-treated pellets of inert clay or fertilizer particles that are applied on or in the soil (occasionally to plants, such as the whorls of corn, which are rolled, unfolding leaves that can hold the granules). Dusts are similar, but much smaller in size, and will adhere to vegetation much more readily. Each product type, like the various liquid formulations, has advantages and disadvantages. For example, coverage and penetration of dense vegetation are superior with dusts, but granules are less likely to suffer from drift problems, and liquids are easy to apply and relatively inexpensive.

Some products are subject to bioaccumulation. **Bioaccumulation** results when, during the course of an animal's life, the rate of intake of a chemical exceeds the rate of elimination. With persistent pesticides, a long-lived animal can repeatedly acquire small, non-toxic doses, eventually resulting in the accumulation of high levels of toxicant. Bioaccumulation may result from biomagnification or bioconcentration. **Biomagnification** is an increase in the concentration of a chemical at each trophic level of a food chain. For example, persistent chemicals in water may accumulate in algae, then accumulate in algae-eating insects, then accumulate in insect-eating fish, and finally accumulate in fish-eating vertebrates. The net result of the chemical being passed along the food chain can be a very high concentration in the animal at the top of the food pyramid. **Bioconcentration** is also an increase in chemical levels but occurs independent of trophic levels. Thus, long-lived wildlife can accumulate high levels of pesticides simply by consuming low levels over a long period of time if they are persistent and are not excreted.

Figure 2. Carolina anole, *Anolis caroliniensis*, consuming a moth. Small reptiles such as small lizards and snakes often are dependent on insects for food.
Credits: Lary Reeves, University of Florida

Figure 3. Southern leopard frog, *Lithobates sphenocephalus*, is typical of amphibians in being unusually dependent on insects as a food source during the adult stage.
Credits: Lary Reeves, University of Florida

Acute Effects of Insecticides

All wildlife can be poisoned by excessive exposure to certain insecticides, but the problem is particularly pronounced with birds. A simple example is the occurrence of seed-feeding flies, *Delia* spp. (Diptera: Anthomyiidae), which damage crop seeds planted in the spring. To prevent damage by such insects while the seed is germinating, coatings containing insecticides are commonly applied to seeds before they are planted. Seed treatment with insecticides (and often fungicides) not only protects the germinating seed, but if the insecticide acts systemically it may also impart protection to young plants, particularly from piercing-sucking insects such as aphids (Hemiptera: Aphididae). Unfortunately, granivorous (seed-eating) birds often will feed on seeds that have been recently planted, and thereby ingest a lethal dose of insecticide. Also, because sand is sometimes consumed by birds to aid in grinding up seeds, birds sometimes feed on granular insecticide, again resulting in bird mortality. Finally, application of liquid insecticides sometimes results in a lethal dose of insecticide being applied directly to wildlife. Though this seems unlikely, when aircraft are used to apply insecticides an extensive land area is treated quickly, and wildlife may not have adequate time to escape. Not only are crop fields treated, but often adjacent border areas (hedge rows, fence rows, irrigation ditches, road margins) are treated deliberately or inadvertently. In the case of nestling birds, there is no opportunity to avoid exposure, even from tractor-mounted sprayers.

There can also be a problem with birds flying into fields that were recently treated, perhaps to feast on dying insecticide-containing insects, and thereby ingesting a lethal dose of insecticide. For example, studies of horned lark, *Eremophila alpestris*, and McCowan's longspur, *Calcarius mccownii*, in relation to chlorpyrifos-treated wheat fields in Montana, US, showed that these birds fed on recently killed cutworms. The percent of their stomachs filled with cutworms was 100% and 95.2% for longspurs and larks, respectively, three days after insecticide treatment. When examined 9 days after treatment, after most of the susceptible insects would have been killed and scavenged, the cutworm contents had diminished to 71.4% and 70.9%, respectively. Birds collected from untreated areas, in contrast, had only 27.2% and 7.7% cutworms in their stomachs, respectively. Unfortunately, birds scavenging on insecticide-killed cutworms acquired more than an easy meal, as brain cholinesterase activity was inhibited by up to 50%. When brain cholinesterase activity is suppressed to this level there are behavioral and physiological consequences, and bird survival diminishes.

Ingestion of insecticide granules is also a particular problem for birds. Insecticide granules are any large carrier particle to which a toxicant will adhere, and from which the toxicant will eventually be washed away by rain or soil water and be made available in the soil to kill pests. Unfortunately, birds habitually consume small, hard particles (usually silica or 'sand') that lodge in their gizzards, helping to grind up food and enhance digestion. Not only do foraging adults mistake insecticide granules for sand and other forms of grit and eat them, but birds often feed this poisonous 'grit' to nestlings. Grit preferences of birds affect their risk of eating pesticide granules, and of being poisoned by granular insecticides. When size and shape of preferred grit overlap with pesticide granules, they are at greater risk of being poisoned. Not surprisingly, a study of granular insecticide use on the Canadian prairie showed that there was a negative correlation of granular insecticide use and the abundance of several birds, including American robin, *Turdus migratorius*; horned lark, *Eremophila alpestris*; house sparrow, *Passer domesticus*; mourning dove, *Zenaida macroura*; western meadowlark, *Sturnella neglecta*; black-billed magpie, *Pica pica*; European starling, *Sturnus vulgaris*; and killdeer, *Charadrius vociferous*. Thus, application of granules can have serious environmental consequences.

The threat of poisoning extends beyond the birds that feed directly on the granules to also include their predators. In Canada, potato fields treated with fonofos for suppression of wireworm were entered by ducks and other waterfowl, and the birds often perished following ingestion of the insecticide granules. In turn, the dying and dead birds were fed upon by bald eagles, *Haliaeetus leucocephalus*, which also perished. Not surprisingly, the cadavers of the waterfowl displayed signs of organophosphate poisoning, including an average level of brain cholinesterase inhibition of 74%.

Other vertebrates are not immune to such poisoning, but it is most pronounced in birds and fish. With fish, the toxicant is contacted primarily via runoff of water from treated fields. Most insecticide labels prohibit treatment of water bodies, and often require a significant barrier zone or untreated border in an effort to limit the drift of liquid pesticide into water. Nevertheless, contamination of water is not unusual.

How severe is the pesticide poisoning problem? The United States Fish and Wildlife Service estimates that over 670 million birds are exposed to pesticide on farmlands in the United States, and that about 10% die immediately as a result. This does not include those that are sickened and

die later, eggs left unhatched, or nestlings left to starve. Organophosphate and carbamate insecticides are most commonly implicated. Farms are not the only source of pesticide poisoning, of course, as pesticides are a common element of suburban landscape maintenance, too. Poisoned animals often are found in relatively good condition, or at least they lack wounds and are intact. Such animals should be considered possible poisonings and submitted to laboratories accustomed to dealing with wildlife samples. Diagnosis of poisoning is not a simple task, and standard veterinary laboratories are not often equipped or experienced with wildlife poisoning.

Figure 4. Passerine birds such as this boat-tailed grackle, *Quiscalus major*, are often considered to be omnivorous, but many are very dependent on insects for feeding their young. Rapidly growing bird chicks need high levels of protein and fat in their diet, and insects provide these nutrients.
Credits: Lary Reeves, University of Florida

Sublethal Effects of Insecticides

Various behavioral and physiological processes can be affected by pesticides, resulting in disruptive, though sublethal, effects on wildlife. Though not causing direct mortality, these effects nevertheless may indirectly cause increased mortality among wildlife (e.g., greater susceptibility to predation) or reduced reproduction (e.g., hormonal imbalance or eggshell thinning). Disruption of nervous system function, alteration of hormone levels, and inducement of oxidative stress via free radical generation are among the sublethal effects induced by insecticides.

Acetylcholinesterase inhibition in birds is much studied, especially in relation to organophosphate insecticide exposure. In general, when cholinesterase activity drops to 50% of normal or less, behavioral and physiological irregularities are apparent, with death following 80% inhibition or greater. At sublethal levels, affected birds may display impairment of memory and learning and inability to thermoregulate properly, make greater use of cover, exhibit reduction of feeding and flying, and show changes in resting posture. Birds may recover within a few hours, however. Similar responses occur in mammals, fish, reptiles, and amphibians. A study of the western fence lizard, *Sceloporus occidentalis*, showed that high levels of carbaryl exposure resulted in reduction in arboreal sprint speed and endurance. Such sublethal factors could affect fitness by reducing the ability of these lizards to escape predation. Similarly, studies have documented reduced swimming speed and distance in tadpoles of the leopard frog, *Rana blairi*.

Not all insecticides are equally disruptive, of course. For example, a study of northern bobwhite (*Colinus virginianus*) foraging in soybean fields of North Carolina found that broods of young quail were present in the soybeans at the time of year when insecticides were applied. Acetylcholinesterase levels and body size were reduced when chicks were exposed to methyl parathion, but not when exposed to methomyl or thiocarb. Methyl parathion is an older product, and its use is now generally banned in the US.

Probably the best-documented example of sublethal effects is eggshell thinning among predatory birds that was caused by organochlorines. The negative correlation between organochlorine residues in birds and eggshell thickness has been observed for many species, in many areas of the world, causing the eggs to be crushed by the nesting birds. For example, organochlorine residues were negatively correlated with reproductive success in bald eagle, *Haliaeetus leucocephalus*; osprey, *Pandion haliaetus*; peregrine falcon, *Falco peregrinus*; Eurasian sparrow hawk, *Accipiter nisus*; American kestrel, *Falco sparverius*; herring gull, *Larus argentatus*; brown pelican, *Pelecanus occidentalis*; and others. This problem is now known to be due to endocrine (hormone) disruption.

Another interesting and well-documented sublethal effect of pesticides is endocrine disruption in American alligator, *Alligator mississippiensis*. Male alligators living in Lake Apopka, Florida, have low testosterone levels. Formerly, the area around Lake Apopka was intensively farmed. Lake Apopka also was the site of a DDT and dicofol (which is closely related to DDT) spill, and the insecticides had estrogen-like effects, resulting in feminization of the males. The penis size of male alligators was reduced by 25%, bone density was affected in females, and egg hatching was reduced. Alligator numbers were reduced by 90% in the years after the pesticide spill. Several organochlorine insecticides, in addition to DDT and its closely related compounds, including endosulfan, toxaphene, dieldrin, and BHC, have been shown to have the potential to disrupt the physiological processes regulated by hormones. DDT is not

acutely toxic to most birds and mammals, but long-term exposure is damaging.

Figure 5. Cattle egrets, *Bubulcus ibis*, are often seen aggregated in pastures and roadsides, where they feast on insects stirred up by grazing animals or mowers.
Credits: Lary Reeves, University of Florida

DDT and other organochlorine insecticides were widely used before their adverse effects were fully appreciated, and though their use is prohibited in many areas of the world, they remain in use elsewhere due to their effectiveness, persistence, and low cost. Birds that migrate long distances may move into and out of countries where DDT is used, so it remains a continuing threat even where it is not currently used. DDT and DDE (a degradation product of DDT that is not insecticidal) affect enzymes controlling calcium deposition in bird eggshells, so eggshell thinning occurs, disrupting normal egg development. Similarly, long-lived fish and marine mammals continue to be exposed to these pesticides because they wash from the land into the oceans, where they are ingested.

Organochlorines are no longer used widely. In Europe and North America (generally the only comprehensive sources of information on this subject) it is the organophosphate and carbamate acetylcholinesterase inhibitors that now cause most of the problems in wildlife. Evidence that the organochlorine situation has improved, and that these pesticides are less of a threat, is apparent in the wildlife literature from many areas of the world. For example, when osprey, *Pandion haliaetus*, populations were monitored recently along the Columbia River system in Oregon, USA, these predators were found to display increased abundance, higher reproductive rates, and significantly lower egg concentrations of most organochlorine insecticides. As recently as the 1980s and 1990s, organochlorine concentrations were high in the fish preyed upon by ospreys in this area, but the situation has improved markedly. This clearly demonstrates that legislation limiting organochlorine pesticide use has benefited wildlife. In the USA, new restrictions have been placed on the use of acetylcholinesterase inhibitors (mostly organophosphates and carbamates), which replaced organochlorines in most agricultural ecosystems. The EPA Conventional Reduced Risk Pesticide Program, initiated in the 1990s, expedites the review and registration process of conventional pesticides that pose less risk to human health and the environment, including non-target organisms, than existing conventional alternatives. These measures further reduce the risk of wildlife poisoning by insecticides in agricultural areas. However, the newer classes of insecticides are not completely without risk, if only because they deplete insect food resources. Also, use of acetylcholinesterase-inhibiting insecticides remains widespread in some areas of the world.

Indirect Effects of Insecticides on Wildlife

In addition to the direct toxicity caused by insecticides, wildlife may be adversely affected indirectly through deprivation of their primary food source, and these indirect effects may be more important than the direct exposure of wildlife to insecticides. One important indirect effect is the depletion of insect populations caused by insecticide use. Application of broad-spectrum insecticides can cause treated fields to become almost sterile, and if the products are persistent the fields may remain depleted of insect life for weeks. Birds will attempt to compensate for loss of insect food by foraging elsewhere, but there are limits as to how far they can fly and then return regularly to a nest with food for nestlings. If the distance of travel is too great, the nest will be abandoned. Due to the high cost of insecticide development and registration, agrochemical companies favor development of broad-spectrum products because, once registered, they can be used extensively on a large number of crops for numerous pest problems and generate large profits before the patent on the pesticide expires. The nonselective nature of such broad-spectrum products is particularly damaging to bird populations; if only the pests were affected, some insect fauna would remain to support bird life, but usually other herbivorous insects, predators, parasitoids, pollinators, and scavengers are also affected, leaving no insects to sustain bird life.

Other pesticides (mostly herbicides) also affect wildlife indirectly, mostly through change in floral diversity (loss of edible weeds, weed seeds, or fungi, and also depletion of habitat or cover) caused by herbicide (and to a lesser

degree by fungicide) application. Grass and weed seed can be an important food resource, and clean culture of crops—though beneficial in terms of plant growth efficiency, energy efficiency and water conservation—can greatly reduce food abundance for bird life. The intensification and specialization of agriculture is manifested in the ever-increasing scale (field size) in agriculture, which usually results from merging smaller fields, reducing crop heterogeneity, and in destroying hedge-row and other border area habitat. These practices have negative effects on wildlife because they may have no place to nest, no place to nest that isn't treated with pesticides, or no source of shelter when crops are harvested, etc. Research in Montana, US, wheat fields showed that herbicide use not only reduced the abundance of broad-leaf weeds, but also the abundance and biomass of insects important to game bird chicks. Weeds also favored the occurrence of ground beetles (Carabidae), important insect predators in this cropping system. The researchers suggested leaving the edges of fields free of herbicide and insecticide treatments, thereby favoring the survival of both beneficial insects and game birds.

Figure 6. Predatory birds such as this burrowing owl, *Athene cunicularia*, as well as other small owls, hawks, and kites, are often thought of as feeding on mammals and birds, but insects are an important part in their diet.
Credits: Lary Reeves, University of Florida

Figure 7. Some birds are nearly wholly dependent on insect life for food. Note the unusual 'whiskers' near the beak of this Chuck-will's-widow, *Caprimulgus carolinensis*. These structures cause insects to be directed to the mouth while the birds sweep through the air in pursuit of 'dinner'.
Credits: Lary Reeves, University of Florida

Risks of Insecticides to Wildlife

Given the long history of negative impacts of pesticides on wildlife, it is tempting to indict all pesticides as hazardous, and suspect all wildlife problems as being caused by pesticides. However, the issue of pesticide toxicity is complicated, and the results of a study investigating the application of malathion (an insecticide) and glyphosate (an herbicide) on tadpoles and predaceous diving beetle larvae (Dytiscidae) serve well to illustrate the issue. The malathion did not greatly affect the tadpoles directly, but glyphosate did reduce survival. The results are complicated by the presence of predators, especially predatory beetles, because they were more susceptible to the insecticide than were the tadpoles. Thus, the addition of insecticide increased tadpole survival by killing their predators! Also, it seems that the glyphosate, which might not be considered to be toxic to animals because it is registered to kill plants, killed tadpoles because the formulation contained a surfactant that was toxic.

There are few places where aesthetics are more important than golf courses. Typically, no expense is spared in an effort to have nearly perfect turfgrass. This usually means that pesticides are used liberally, so one might expect that golf courses would be a particularly inhospitable environment for birds. However, assessment of reproductive success in eastern bluebirds, *Sialia sialis*, in Virginia, US, showed that these insectivores can survive quite well, with survival not differing significantly between bluebirds nesting in golf course environments and non-golf course environments. On golf courses, the number of eggs per nest box was higher (28% greater), as was the number of young

birds successfully fledged (17% greater). Overall, pesticide use on the golf course did not impose a significant stress on bluebirds, suggesting that pesticide use and wildlife can be compatible under some circumstances.

Sometimes insecticides can be used to enhance wildlife populations. Often there is a negative relationship between arthropod abundance and some aspect of survival or reproductive performance. For example, several studies of great tit, *Parus major*, in Europe have found influences of hen flea, *Ceratophyllus gallinae* (Siphonaptera), on aspects of behavior or fitness such as roost site selection, nest site selection, timing of breeding, and body mass of nestlings. Reduction in flea abundance by application of an insecticide inside nesting boxes has been shown to improve elements of fitness and could be used for other birds troubled by fleas. Hen fleas are not much of an issue in North America, but nest boxes here may harbor lice, mites, ticks, and certain parasitic flies. Similarly, burrows of prairie dog colonies are sometimes dusted with insecticides to control fleas, the vectors of plague. This serves not only to improve survival of the prairie dogs, but also to protect endangered black-tailed ferret, *Mustela nigripes*, populations.

Animal populations can sometimes be managed for improved health by applying pesticides to them or to food bait to suppress endo- and ectoparasites; this is especially useful where high densities of wildlife are being maintained, such as zoos and game parks. Several methods of self-treatment have been developed for wildlife that facilitate application of insecticide to control ticks that harbor Lyme disease. Elimination of the threat of Lyme disease makes it more tolerable to have wildlife populations in suburban areas.

How do we protect wildlife from insecticides?

The obvious solution for the problem of insecticides causing injury to wildlife is to avoid insecticide use. Although insecticides are convenient, practices that reduce the likelihood of pests attaining damaging levels that warrant insecticide intervention are potentially available. Integrated pest management practices that minimize insect population growth include careful selection of plant cultivars that are less suitable for insects, timing of planting and harvesting to escape infestation by insects, introduction of plant diversity and smaller plantings of the same species, careful use of fertilizer and irrigation to disrupt insect populations, protection of insect natural enemies such as predatory and parasitic insects, and use of nontoxic pest suppression techniques such as pheromone-based trapping or confusion techniques.

Alternatives to insecticides are not always practical, but it is important to apply insecticides only when necessary rather than in a preventative mode, as this reduces exposure of wildlife to insecticides. It is critically important to select insecticides that act specifically on insects, rather than using broad-spectrum nerve-disrupting products, as this may allow wildlife to exist even where insect control is being implemented. Take into consideration the time of day when insecticides will be applied in respect to avoiding direct application to plants while beneficial pollinators and predators are active.

Finally, it is important to recognize that many types of wildlife feed on insects, and insect-based food reservoirs need to be maintained in the vicinity of treated areas. Natural areas that are not treated with insecticides, even relatively small areas like roadsides, fence rows, irrigation ditches, parks, and backyards are important in allowing survival of edible insects and the wildlife that feed upon them.

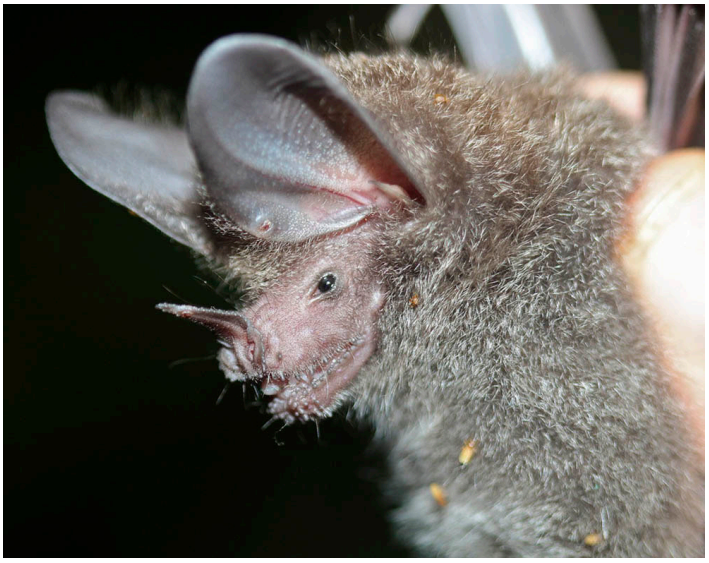

Figure 8. Mammals such as this big-eared bat, *Micronycteris* sp., feast on night-flying insects.
Credits: Lary Reeves, University of Florida

Figure 9. Small mammals, including this cotton mouse, *Peromyscus gossypinus*, often consume large numbers of insects, though most are omnivorous and can feed on many types of food.
Credits: Lary Reeves, University of Florida

Summary

If you are concerned about protecting wildlife from pesticide exposure it is important to know that:

- a number of chemical toxicants are used to manage pests, but perhaps the most important of the pesticides from the perspective of wildlife conservation are the insecticides;
- toxicity is dose dependent, and insecticides are neither entirely safe nor entirely hazardous. Insecticides are used most safely when used according to directions, much like the situation with prescription drugs. However, remember that some forms of wildlife are not much bigger than insects, so they can be quite susceptible to injury;
- information on toxicity and hazard can be found on the container label in the form of a signal word, but more detailed information is found on the Material Safety Data Sheet (MSDS);
- toxicity can occur rapidly (acute toxicity) or following prolonged exposure (chronic toxicity). Lethal effects are difficult to assess, but sublethal effects such as changes in behavior and growth are even more difficult to assess;
- the method of killing by most insecticides, or mode of action, is by disruption of nerve function, though some toxins affect other physiological processes. Because the nerve systems of invertebrates and vertebrates are similar, wildlife can be adversely affected by excessive exposure to insecticides;
- in addition to direct toxicity, insecticides can adversely affect wildlife populations if the insecticides are so effective that they eliminate the food supply for animals that feed on insects;
- wildlife comes into contact with insecticides when they walk on or ingest plants; swim in contaminated water; consume contaminated water or insects; consume treated baits or granular formulations of insecticides; or are sprayed directly. Some insecticides can accumulate in the environment, becoming more hazardous;
- despite the presence of insecticides, wildlife may be unaffected, or may even benefit from insecticides when ectoparasites are controlled;
- rather than depending on insecticides for insect suppression, alternative practices should be considered. Selective insecticides should be favored, and chemicals should be applied only when necessary. It is important to retain populations of some insects so they can serve as a food resource for wildlife.

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Table 1. Acute toxicity categories of pesticides in relation to hazards.

Hazard Indicator	Toxicity Category I	Toxicity Category II	Toxicity Category III	Toxicity Category IV
Signal Word	Danger, or Danger/Poison	Warning	Caution	Caution
Oral LD_{50}	≤ 50 mg/kg	50–500 mg/kg	500–5000 mg/kg	> 5000 mg/kg
Dermal LD_{50}	≤ 200 mg/kg	200–2000 mg/kg	2000–20,000 mg/kg	> 20,000 mg/kg
Inhalation LC_{50} (dust or mist)	≤ 2 mg/l	2–20 mg/l	20–200 mg/l	> 200 mg/l
Eye effects	irreversible corneal opacity at 7 days	corneal opacity reversible within 7 days, or irritation persisting for 7 days	no corneal opacity, or irritation reversible within 7 days	no irritation
Skin irritation	severe irritation or damage at 72 hours	moderate irritation at 72 hours	mild or slight irritation at 72 hours	no irritation

Table 2. Comparative oral toxicity of selected insecticides, fungicides, and herbicides to representative vertebrate animals. Toxicity categories are very highly toxic (*****), highly toxic (****), moderately toxic (***), slightly toxic (**), and practically non-toxic (*).

Pesticide	Vertebrate test animal		
	Mammal	Bird	Fish
Insecticides			
Carbaryl	**	*	****
Methomyl	****	****	***
Malathion	***	***	****
Acephate	**	***	*
Imidacloprid	**	***	***
Thiamethoxam	*	**	*
Permethrin	**	*	*****
Bifenthrin	***	**	****
Cyromazine	**	**	**
Esfenvalerate	***	*	*****
Spinosad	**	**	***
Rynaxypr	**	*	**
Fungicides			
Mancozeb	*	*	***
Maneb	*	**	***
Difenoconazole	**	*	*
Fenbouconazole	**	**	*
Captan	*	**	****
Azoxystrobin	*	*	*
Herbicides			
2,4-D	**	*	*
MCPA	**	***	*
Diquat	**	***	**
Paraquat	***	***	**
Atrazine	**	**	**
Glyphosate	*	*	***

[a] Mammal LD_{50} values are <50 for highly toxic, 51–500 for moderately toxic, 501–5000 for slightly toxic, and >5000 for practically non-toxic. Bird LD_{50} values are <10 for very highly toxic, 10–50 for highly toxic, 51–500 for moderately toxic, 501–2000 for slightly toxic, >2000 for practically non-toxic. Fish LD_{50} values are <0.1 for very highly toxic, 0.1–1 for highly toxic, 1.1–10 for moderately toxic, 10.1–100 for slightly toxic, and >100 for practically non-toxic.

Table 3. Some major groups of insecticides and their characteristics. Note that nearly all groups affect the insect nervous system.

Class	Examples	Mode of Action	Other important characteristics
Insecticides that affect the nervous system			
Inorganic insecticides	boric acid, borates, cryolite, silicates	various, often stomach poisons	persistent, usually low toxicity to mammals
Organochlorines (DDT group)	DDT, dicofol, methoxychlor	prevent nerve repolarization	persistent, metabolically stable, lipophilic, variable toxicity to mammals
Organochlorines (cyclodienes)	dieldrin, chlordane, heptachlor, lindane, endosulfan	GABA receptor antagonist in nerves	persistent, metabolically stable, lipophilic, low toxicity to mammals
Organophosphates	malathion, parathion, diazinon, chlorpyrifos	inhibit acetylcholinesterase in nerve synapses	variable toxicity, water soluble, biodegradable
Carbamates	carbaryl, methomyl, carbofuran, aldicarb	inhibit acetylcholinesterase in nerve synapses	variable toxicity, water soluble, biodegradable
Pyrethroids	permethrin, esfenvalerate, cypermethrin	prevent nerve repolarization	biodegradable, not very toxic to mammals, very toxic to fish
Nicotinoids (= chlornicotinyls and neonicotinoids)	imidacloprid, acetamiprid, thiamethoxam	activation of nicotinic acetylcholine receptors	low mammalian toxicity, but affect fish and birds
Spinosyns	spinosad	activation of nicotinic acetylcholine receptors	derived from soil bacterium, low toxicity to mammals and birds, moderately toxic to fish
Avermectins	ivermectin, abamectin	activate glutamate chloride receptor	used widely on animals for insect and nematode control but hazardous to mammals and fish
Phenylpyrazoles	fipronil	block GABA chloride receptor	low mammalian toxicity, but affect fish and birds
Anthranilic diamides	rynaxypry	binds to ryanodine receptors in muscles	low toxicity to nearly all except some insects
Insecticides that are insect growth regulators			
Juvenoids	methoprene, hydroprene, fenoxycarb	bind juvenile hormone receptors	keep insects in juvenile form, selective, low toxicity to mammals
Chitin synthesis inhibitors	diflubenzuron, hexaflumuron, buprofezin	inhibit chitin formation	interfere with molting, low toxicity to mammals, birds, and fish

Table 4. The toxicity of some common insecticides (products with low numerical values are more toxic).

Insecticide	Toxicity (mg/kg oral LD)
Organochlorines[a]	
DDT	113
Aldrin	38
Chlordane	250
Dieldrin	40
Endrin	7
Heptachlor	40
Lindane	88
Methoxychlor	6000
Toxaphene	49
Organophosphates	
Acephate	945
Chlorpyrifos	135
Diazinon	1250
Dichlorvos	50
Dimethoate	387
Fenthion	250
Fonofos	8
Malathion	1375
Methyl parathion	3
Monocrotophos	165
Parathion	2
Phorate	1.6
Trichlorfon	250
Carbamates	
Aldicarb	0.9
Carbaryl	500
Carbofuran	8
Methiocarb	20
Methomyl	17
Propoxur	50
Pyrethroids	
Allethrin	860
Bifenthrin	55
Cyfluthrin	500
Cyhalothrin	56
Cypermethrin	250
Esfenvalerate	75
Permethrin	430
Avermectins	
Abamectin	11
Emamectin	93
Nicotinoids	
Acetamiprid	146

Insecticide	Toxicity (mg/kg oral LD)
Imidacloprid	424
Thiamethoxam	1563
Spinosyns	
Spinosad	3738
Phenylpyrazoles	
Fipronil	97
Anthranilic diamides	
Rynaxypyr	>5000
Insect Growth Regulators	
Methoprene	34,600
Hydroprene	5,500
Fenoxycarb	10,000
Cyromazine	3387
"Natural" Products	
Bacillus thuringiensis	>15,000
Azadirachtin	5000
Rotenone	132
Pyrethrin	200
Nicotine	55
Ryania	1200
Sabadilla	4000
Spinosad	5000
Requiem	>5000
Petroleum oil	15,000
[a] nearly all US registrations have been cancelled for these products.	