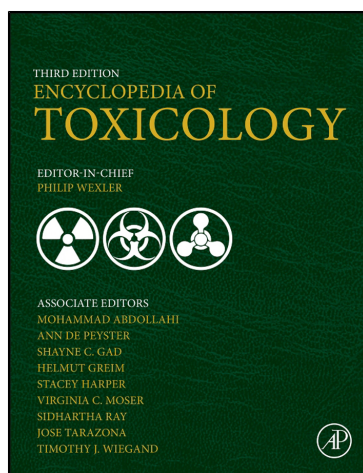


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Ecotoxicology, Avian

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Ecotoxicology is an interdisciplinary science that studies the movement of environmental contaminants through ecosystems and their harmful effects on biota. Therefore, avian ecotoxicology is the subset that concerns itself with birds as part of the ecosystems. Avian ecotoxicology forms part of the lowest level into this hierarchical science, organismal ecotoxicology, which explores toxicant effects to individual and, where possible, links them to population and communities. For this purpose, avian ecotoxicology, as the other branches of ecotoxicology, is built on sciences such as toxicology, analytical chemistry, ecology, physiology, pathology, behavioral sciences, etc.

Avian ecotoxicology probably began in the late nineteenth century with the early references about wildlife toxicology concerning pheasant and waterfowl mortality related to ingestion of lead pellets. In the following century, by the 1930s, this fact was recognized as a common cause of death in waterfowl. Also in the early twentieth century, the first experimental studies with fowls, both domestic and wild, were conducted. However, it was later, in the 1940s, when the first studies of environmental contaminants using captive waterfowl were carried out. Lead, arsenic, strychnine, and white phosphorous were the first toxic compounds studied and analyzed in avian tissues. At the same time, in the late 1930s, during the Third North American Wildlife Conference, application of pesticides was also acknowledged as a potential inductor of adverse effects to wildlife, especially to birds. In those days, the insecticidal properties of dichlorodiphenyltrichloroethane (DDT) had not been still discovered. During the second part of the 1940s and the early 1950s, the first reports about the DDT effects on avian species were written. These reports related an increase of dead birds to applications of high concentrations of DDT in agricultural practices. Convincing evidences emerged worldwide about population declines of raptors and fish-eating birds induced by DDT and its degradation product, DDE (1,1-dichloro-2,2-bis-(*p*-chlorophenyl)-ethene). Nevertheless, organochlorine compounds were not the only toxic products sprayed on the earth: organophosphorous pesticides (e.g., schradan, parathion), and rodenticides such as compound 1080 were also widely used during the 1940s and later on. Actually, after World War II, reduced numbers of some species of birds and mortality episodes were noted in the areas where different pesticides such as DDT, parathion, aldrin, dieldrin, schradan, chlordane, heptachlor, toxaphene, and others had been widely used. Some of them (e.g., aldrin or dieldrin) used as seed dressings were found to be highly hazardous to passerine and game birds. For this reason, widespread applications of DDT had also unacceptable consequences to birds of prey populations. Therefore, early problems involving pollutants focused not only on consequences for individuals or organisms, but also on consequences for populations. Toxic effects in individuals are able to explain some changes observed in field populations; for

example, DDT-induced change in calcium-dependent ATPase in the eggshell gland is an individual sublethal effect with consequences on bird population failure. Controversial reports regarding the relationship between pesticide use and wildlife mortalities, especially bird populations, prompted experimental studies and controlled field studies relying on tissue concentrations as one of the most relevant measurement endpoint. Measuring tissue residues of pollutants in birds is relevant to ecotoxicology, both for understanding the exposure and kinetics of contaminants within organisms and their movements through food chains, and for understanding the adverse effects in organisms and their populations. In this sense, in the early 1950s, both acute and chronic exposure studies were carried out on captive game birds reporting relevant information on signs of intoxication, lethality, and accumulation of contaminants in tissues. The most frequent approach was a combined laboratory–field approach in which concentrations of contaminants found in dead or intoxicated birds were compared with those obtained in experimental studies conducted on dosed animals.

In 1962, Rachel Carson's *Silent Spring* was published and issues that had only been debated by experts and scientists in specific meetings began to be discussed in all sectors of the society. Although the society was moderately aware on the hazards and risks associated to toxic compounds, especially pesticides, a new conception and other viewpoints regarding the risks associated to chemicals were clearly highlighted in Carson's book. The effects of pesticides on nontarget organisms, the chemical persistence in the environment, the resistance to the effects of pesticides, and the most alarming issue, the human safety concerns, were now in the center of the public scene. A few years later, new legislation was published and governmental agencies, such as US Environmental Protection Agency (US EPA), were established to deal with environmental contamination.

Birds as Sentinels of Environmental and Human Health

The value of birds as biomonitors of environmental quality has been broadly recognized as an important tool for environmental management and several government established monitoring programs are a proof of this. Among the multitude of aims of monitoring using sentinel animals, comes the collection of data in order to estimate human health risks, identify food chain contaminants, determine levels of environmental contamination, and identify adverse effects on the animals themselves.

In the last two decades, evidence has been accumulated indicating that humans and wildlife, and specially wild birds, may be affected by industrial chemicals and pesticides that interfere with hormones affecting reproduction via nonspecific

morbidity or increased stress, provoking cessation of egg-laying, interruption of incubation, or reduced care of chicks. In 1991, the Committee on Animals as Monitors of Environmental Hazards, from the National Research Council, puts forward some recommendations for the use of sentinel animals in risk assessment, and finally concluded that the implementation of these recommendations would greatly enhance and improve human risk assessment. Also in 1991, during the Wingspread Conference 'chemically-induced alterations in sexual development,' five facts were included as certain statements by consensus: (1) a large number of artificial chemicals that had been released into the environment, have the potential to disrupt the endocrine system of animals, including humans, (2) many wildlife populations (wild birds included) are already affected by these compounds, (3) the pattern for effects vary among species and among compounds, (4) laboratory studies corroborate the abnormal sexual development observed in the field and provide biological mechanisms to explain the observations in wildlife, and (5) humans have also been affected by compounds of this nature. Finally, a major conference on endocrine disrupting chemicals was held between 2 and 4 December 1996, in Weybridge, England. This conference was organized by several European organizations and national environmental agencies. Numerous recommendations were identified during the workshop and some of them directly concerned wildlife, especially wild birds. Some examples are the need to conduct field studies at locations where endocrine disruption may occur, the need to identify which species should be studied in order to determine whether the environment was being affected (sentinel animals), and the need to look for biomarkers to predict detrimental effects on reproduction.

Among birds, raptors, other top predators, and scavengers (carrion-feeders) are especially suitable for monitoring persistent and bioaccumulative contaminants. These bird species, located at the top of food chains, are able to integrate contaminant exposure both over time and relatively large spatial areas. Moreover, they are relatively easily captured and censed (especially nestlings), which facilitates collection of nondestructive samples (blood, feather, preen gland oil, unhatched eggs, or pellets). Many species of birds, especially top predators, are also known to have measurable responses to persistent contaminants, ranging from residue accumulation to population decline. Actually, it was the dramatic population decline noted in the bald eagle (*Haliaeetus leucocephalus*) in the USA, or in several species in the UK, such as Peregrine falcon (*Falco peregrinus*), Eurasian sparrowhawk (*Accipiter nisus*) and golden eagle (*Aquila chrysaetos*), which clearly demonstrated the high value of birds of prey as powerful sentinels of environmental quality.

Persistent Organic Pollutants

According to the Stockholm Convention, persistent organic pollutants (POPs) are toxic, resistant to degradation, can bioaccumulate, and are transported through air, water, and migratory species across international boundaries and deposited far from their releasing areas, where they accumulate in terrestrial and aquatic ecosystems. Many species of birds have

migratory habits, being this fact to take into account when exposure to these compounds and environmental quality are being evaluated or monitored. POPs encompass an array of anthropogenic organic and elemental substances, and their degradation and metabolic by-products that have been found in the tissues of exposed birds, especially POPs are categorized as organohalogen compounds (OHCs). These are global contaminants which are widely distributed within ecosystems and include organochlorine compounds such as polychlorinated biphenyls (PCBs), organochlorine pesticides (OC), hexa- and penta-chlorobenzenes, and polychloro dibenzo-*p*-dioxins (PCDDs) and polychloro-dibenzo-furans (PCDFs); brominated flame retardants such as polybrominated diphenyl ethers (PBDEs) and polybrominated biphenyls; and perfluorinated compounds such as perfluorooctane sulfonate and perfluorooctanoic acid. Many of these have been synthesized for industrial use (e.g., PCBs, PBDEs) or as agrochemical products (e.g., DDT, lindane, chlordane), but there are also compounds such as PCDDs and PCDFs that are unintentionally released to the environment.

Exposure to OHCs in birds has frequently been associated with toxic effects (e.g., embryotoxicity, eggshell thinning, or behavioral alterations) that have impaired reproductive success. The population consequences of DDT or DDE inhibition of Ca-ATPase in the eggshell gland of birds, resulting in thin-shelled eggs that break before full development and hatching are probably the most studied and cited effect in the literature in this regard. As a consequence, several bird species have suffered declines in their populations. Such environmental risks have led to the ban or restrictions of use of most of these compounds and a subsequent decrease of their concentrations both in the environment and bird tissues. However, due to their high persistence and ability to bioaccumulate and biomagnify through the trophic chain, environmental concentrations of these compounds may still be important and exert potential risks in bird populations.

Recent studies have demonstrated that accumulation of these compounds is not uniform among bird species, but it varies according to differences in diet and biotransformation abilities. Frequently, OC concentrations detected in different bird tissue samples are not considered directly responsible for organism death. Nevertheless, these compounds are able to produce chronic effects that lead them to be considered hormone disruptors, immunosuppressants, and the cause of adverse effects on the nervous and reproductive systems, as mentioned above. Moreover, interactions with other environmental pollutants may occur and thus be indirectly responsible for detrimental health effects. For example, it has been shown that PBDEs can interact with PCBs to cause developmental neurotoxic effects in mice when exposed during a critical period of neonatal brain development.

Apart from the species, other factors such as sex, age, feeding habits, body condition, migratory habits, etc. influence the exposure and kinetics of these compounds into the organisms, and consequently the accumulation in tissues, fluids, and bird products. Several studies have concluded that females have lower organochlorine concentrations than males, probably due to the transfer of these compounds from mother to egg. The higher levels in the fat tissues of older birds

may reflect a longer period of exposure in these individuals and a low elimination rate of these compounds. Organochlorine concentrations in birds depend, among other factors, on trophic level and feeding habits of each species. In studies with birds of different feeding habits, the highest OC levels were found in piscivorous birds, followed by insectivores, omnivores, and herbivores. Changes in feeding behavior can also influence OC exposure. Furthermore, body condition is also a determining factor in OC levels. The mobilization of organochlorines from depleting fat stores related to higher concentrations of these compounds in body organs (e.g., the liver). During periods of distress, such as migration or breeding, the stored body fat is metabolized and the lipophilic OC are mobilized and distributed through the bloodstream to highly active organs, i.e., the liver, which exhibits only a modest change in lipid content. In the case of migration, this is a period of exceptional energy demand. Thus, before migration, birds deposit substantial fat stores to meet this high demand, which may reach 50% of the total body mass in long-distance intercontinental migrants.

Other Nonhalogenated Pesticides

In the late 1960s and 1970s, many OCs were replaced in industrialized countries by compounds that were less persistent in the environment and less bioaccumulative into the organisms, such as organophosphorus and methyl carbamates. In spite of this, the high toxicity showed by some of them, their massive and uncontrolled use in some cases, and their nonspecific mechanism of toxicity (inhibition of acetylcholinesterase provoking alteration in nerve transmission and functioning of neuromuscular junctions) led to massive songbird mortalities all around the world. Moreover, many of them have been widely used in poisoned baits to deliberately kill large birds of prey and other 'undesirable' wild and domestic animals. On the other hand, these compounds are often involved in secondary poisonings in nontarget predatory wildlife, mainly large birds of prey and carrion-feeders, such as vultures.

Pesticide coated seeds are commonly used in agriculture, and may be an important source of food for some birds in times of scarcity, as well as a route of pesticide ingestion. Mortality episodes of birds related to ingestion of treated seeds have been widely reported during the last four decades, and today is still a matter of less concern in comparison to other type of intoxications or poisonings. Recently, an experimental study on a game bird species, the Red-legged partridge (*Alectoris rufa*) fed with seeds coated with difenoconazole (fungicide), thiram (fungicide), or imidacloprid (insecticide) found both direct and indirect effects on the body condition, physiology, immunology, coloration, and subsequent reproduction of exposed partridges. Thiram and imidacloprid produced mortalities at the highest doses, and all pesticides provoked sublethal effects such as altered biochemical parameters, oxidative stress, and reduced carotenoid-based coloration. In addition, detrimental effects on size of eggs, fertilization, and chick survival were reported. Therefore, pesticide-treated seeds should be considered when declining bird populations in agricultural environments are being assessed.

Cadmium

Environmental pollution caused by heavy metals has been responsible for numerous pathologies in wild species. Cadmium (Cd) is widely distributed in the environment and has been described as highly toxic for living beings, affecting the survival and reproduction of birds. Birds are exposed to Cd primarily throughout their diets, but its intestinal absorption is very low (less than 7% of ingested). Cadmium levels in bird tissues are influenced by many factors such as feeding habits and diet, age, physiological status, ecosystem use, etc. Significant differences exist among species regarding Cd accumulation in tissues. In fact, pelagic species accumulate more Cd than other seabirds, terrestrial birds, freshwater birds, and shorebirds.

Diverse alterations in birds have been reported after field or experimental exposure to Cd, such as intestinal damage and altered nutrient uptake; kidney damage with alteration of vitamin D metabolism; skeletal effects such as osteomalacia, osteoporosis, or osteopenia; effects on osmoregulation, and energy metabolism; effects on reproductive organs and reproduction, such as atrophied testes, reduced egg production, or experimental eggshell thinning; endocrine disruption; anemia; behavioral alterations; and immune deficiencies.

Lead

Lead (Pb) ammunition is probably the most relevant source of Pb poisoning in birds, not only in waterfowl, but also in top predators and scavengers or carrion-feeders. Waterfowl and other bird species ingest directly Pb shotgun pellets, presumably as grit of food particles, in areas that are hunted over, including wetlands, farmland, and terrestrial game shooting areas. Moreover, some species of waterbirds, mainly diver species, also ingest metallic Pb with the angler's Pb weights representing a serious problem in areas where recreational fishing is practiced. The other group of birds affected by ingestion of ammunition includes those birds that prey upon or scavenge the flesh of game species, which is formed by raptors, vultures, and other scavenging birds. Lead pellets in the preys can be present in their gizzards or embedded in their body. Avian mortalities due to ingestion of Pb ammunition have been described in all parts of the world for over a century, and therefore the use of Pb ammunition has been restricted in almost 30 countries around the world. The restriction depends on the countries: while in some countries the restriction is only for shooting waterfowls in wetlands, it has been banned in all hunting activities in other countries.

In addition to Pb shotgun pellets, birds are exposed to Pb from other sources, such as ingestion of sediments containing Pb at locations surrounding mines and smelters, atmospheric lead in urban environments from airborne sources, ingestion of material with lead-based paints, etc.

Similarly to mammals, lead accumulation follows a multi-compartmental kinetic model once it has been absorbed and is transported through the bloodstream. Probably the most relevant differences between birds and mammals are related to the type of lead ingested (diet) and the absorption rate. In birds, lead absorption greatly varies due to several factors including

age, gender, breeding condition, feeding habits and diet, anatomy and physiology of digestive tract, etc. In healthy adults, lead is poorly absorbed in the gizzard in the metallic form, while the absorption rate is slightly greater when lead is biologically incorporated to food (e.g., birds of prey that ingest preys highly exposed to lead). In any case, only a small fraction of lead ingested arrives to the bloodstream, because it is mainly eliminated with feces. In general, lead in bone is more frequently accumulated in females than in males, and in laying females more than nonlaying ones. This is probably due to mobilization of calcium from medullary bone for eggshell formation, and to an increase of intestinal absorption of calcium (and concurrently lead). On the other hand, anatomical differences of the stomach among species can also influence the retention of metallic lead objects. In this sense, granivorous and insectivorous birds have a gizzard adapted to digest hard diets, while carnivores, fish-eating birds, and scavengers are adapted for a softer diet. As mentioned above, diet could be the most important factor influencing both lead absorption and its deposition in tissues. In this sense, nutritional, physical, and chemical characteristics of diet should be taken into account when assessing the risks associated to lead poisoning in birds.

Bone concentration is an indicator of chronic exposure and thus less useful for the evaluation of toxicity at the time of sampling. However, threshold concentrations in blood, liver, and kidney have been associated with subclinical, toxic, and compatible-with-death classifications in Falconiformes. In general, birds chronically exposed to environmental lead do not usually display blood and tissue concentrations above the threshold concentrations associated with clinical toxic effects. In spite of this, blood lead level is able to offer information on recent exposure of interest in risk assessment.

Lead can cause mortality in cases of acute poisoning or can indirectly affect avian populations by altering reproductive success, behavior, immune response, and physiology in cases of chronic exposure. As in mammals, lead inhibits the activities of certain enzymes participating for the synthesis of heme group of hemoglobin, such as δ -aminolevulinic acid dehydratase (δ -ALAD) and heme synthetase. In birds, the inhibition of erythrocyte δ -ALAD activity is the first measurable biochemical change after lead absorption, and its inhibition persists for several weeks to several months, depending on blood lead concentrations. This biochemical parameter has been widely used as biomarker of exposure and effect in several bird species.

Mercury

Mercury (Hg) is a persistent, toxic, and nonessential heavy metal of special concern due to its bioaccumulation and biomagnification through the food chains. Inorganic Hg is converted into organic forms (methylmercury, MeHg), especially in aquatic ecosystems, being this organic form the most harmful and bioaccumulative in food chains. Aquatic environments are especially at high risk of Hg contamination since much of the atmospheric deposition and all industrial water-runoff culminates in these ecosystems. This is the reason for the fact that much of the effort involving Hg investigations has

disproportionately focused on seabirds and other piscivorous birds, which have been frequently used as biomonitors of marine ecosystem health quantifying mercury concentrations in tissues, feathers, eggs, and blood. Furthermore, it is generally assumed that seabirds can tolerate higher Hg concentrations, considering the range of processes which may contribute to Hg detoxification, such as the molting process, induction of synthesis and binding to metallothionein, as well as demethylation and the formation of Se–Hg complexes. Some studies have found that in large predators such as some seabirds Hg levels are 10 times higher than in medium-sized fish, the latter being components of the former's diet. It has been considered that up to 90% of a bird's total Hg body burden may be sequestered into feathers during the feather growth, where Hg binds with feather keratin in the form of MeHg. Results indicate that feathers are an excellent nondestructive tool for monitoring mercury levels in several seabird species. Although fish-eating birds generally exhibit higher exposure to dietary MeHg than terrestrial animals, evidence suggests that methylation may also occur in certain terrestrial food chains in forest habitats. Therefore, some studies have found large accumulations of this contaminant in birds of prey that feed at the top of terrestrial trophic chains. In this sense, significant differences in feather Hg concentrations were found in relation to trophic level in 18 bird species from southwest Iran, and raptors that feed from vertebrates (except fish) showed the highest level of Hg. Finally, mercury historically used in pesticide seed treatments caused severe mortality in seed-eating bird species and their predators. Sublethal toxic effects in birds related to dietary exposure and tissue concentrations of Hg have been reported, such as effects on growth, development, reproduction, immune system, metabolism, nervous system, and behavior. Although these effects have been widely described in fish-eating birds, additional studies are required to better understand the significance of elevated MeHg concentrations in terrestrial food webs.

Selenium

Selenium (Se) is a metalloid trace element that birds and animals need in small amounts for good health, being food the main source of Se accumulation for birds. In some parts of the world, Se deficiencies must be corrected by addition of Se to the diet. In spite of being an essential element, Se is very embryotoxic at high doses. The most drastic incident of Se poisoning in wild birds occurred in California, at Kesterson Reservoir (Kesterson National Wildlife Refuge), during the early and mid-1980s. Se salts from soil naturally dissolved with the water used to irrigate crops were drained from agricultural fields into Kesterson Reservoir, and plants accumulated Se in amounts that were toxic to birds. The most likely effects in field studies are reproductive impairments, which have been widely documented, including poor hatching success due to embryotoxicity, poor survival of hatched eggs, and high incidence of developmental abnormalities with visible malformations in embryos and chicks (small or missing eyes, absence of legs or toes, incomplete beak development, etc.). In spite of this, Se is also able to cause toxicosis and mortality in adult birds when exposed to high doses. However, significant interspecies

differences have been described between Black-winged stilt (*Himantopus himantopus*) and American avocet (*Recurvirostra americana*).

Anticoagulant Rodenticides

Anticoagulant rodenticides (ARs) have been extensively used for rodent control, allowing secondary exposure and poisonings in nontarget predatory wildlife species, such as birds of prey that mainly feed on rodents or small birds. In spite of this, ARs have not been routinely included in biomonitoring studies. In those countries where monitoring schemes include these compounds (e.g., UK, USA), a high frequency of detection has been reported in bird samples. However, in those countries where there is not a monitoring scheme for these compounds, data regarding rodenticide levels in raptors are only restricted to cases of suspected poisoning or after use to eradicate plagues.

There are two generations of ARs called the first generation anticoagulant rodenticides (FGARs) and the second generation anticoagulant rodenticides (SGARs). The FGARs group includes the coumarin compounds: coumatetralyl and warfarin and the 1,3-indandiones; chlorophacinone and diphacinone. On the other hand, the SGARs group was introduced in the 1970s to avoid the rodent resistance to FGARs. They are more toxic and persistent than FGARs, and are based on derivatives of 4-hydroxycoumarins and include difenacoum, brodifacoum, difethialone, flocoumafen, and bromadiolone among others. SGARs have a greater affinity to binding sites in the liver and consequently display greater acute toxicity (approximately 100–600 times), accumulation, and persistence. The secondary exposure and poisoning is more common in predators feeding on rodents poisoned by SGARs, which is due to their longer tissue half-lives and accumulation after repeated sublethal exposures. SGARs bind and inhibit vitamin K epoxide reductase and persist for at least 6 months in organs and tissues containing this enzyme, such as the liver. Therefore, these compounds act as vitamin K antagonists disrupting normal blood clotting processes, and causing lethal hemorrhage. Prior to death, birds show typical clinical symptoms that include anemia, dyspnea, and lethargy. However, it is also known that a sublethal dose of rodenticides can produce significant clotting abnormalities and hemorrhages without causing death, which may increase the likelihood of death due to other causes or environmental stressors, such as food shortage or predation. The liver concentrations of SGARs associated with adverse effects and/or mortality in birds markedly vary among both individuals and species, and have not been defined for most raptor species. In the same manner, avian acute toxicity also varies between species. For example, brodifacoum LD₅₀ values of 0.31, 0.72, and 19 mg kg⁻¹ bw were calculated for mallard duck (*Anas platyrhynchos*), laughing gull (*Leucophaeus atricilla*), and Japanese quail (*Coturnix coturnix japonica*), respectively.

Veterinary Pharmaceutical Residues

Population declines of three species of vultures endemic to South Asia were first noticed in India in the early to mid-1990s.

Nowadays, these species are listed as in danger of extinction. Observed rates of population decrease are probably the highest recorded for any bird species, leading to total declines of 99.9% for the Oriental White-backed vulture (*Gyps bengalensis*), and 97% for Long-billed (*Gyps indicus*) and Slender-billed (*Gyps tenuirostris*) vultures, between 1992 and 2007. In 2003, the cause of these declines was discovered: diclofenac, a non-steroidal anti-inflammatory drug (NSAID), whose manufacture and import for veterinary use was banned in India, Nepal, and Pakistan in 2006 and in Bangladesh in 2010. The poisoning was due to consumption of contaminated domestic livestock carcasses. The likelihood of exposure to veterinary pharmaceuticals used to treat domestic livestock is higher in South Asia (India, Pakistan, Nepal) due to the unique situation of large numbers of livestock carcasses left in the field to be ingested by vultures. Studies in India indicate that 10% of carcasses (the main food source of vultures in Asia) were contaminated with diclofenac.

It has been proven that diclofenac is highly toxic to at least six of the eight *Gyps* vulture species. However, some studies suggest that it is likely to be toxic to all eight *Gyps* species. In this sense, one experimental study on Cape Griffon (*Gyps coprotheres*) vulture from Africa showed that this species is as sensitive to diclofenac as the other *Gyps* species. Because toxicity to diclofenac is genus specific, not just species specific, it is possible to think in a potential risk to a wide variety of birds. On the other hand, some scavenging species appear to tolerate high levels of diclofenac, such as the Turkey vulture (*Cathartes aura*) or Pied crow (*Corvus albus*), supporting the hypothesis of species or genus specificity.

Experimental studies in four *Gyps* vulture species have shown the high lethality of diclofenac within a few days as consequence of an extensive visceral gout and kidney damage in all of these species. These findings were similar to those described in the majority of vulture carcasses collected from the wild since declines began. The NSAIDs different from diclofenac, most commonly found in carcasses available to vultures are meloxicam, ibuprofen, and ketoprofen. However, the list of NSAIDs that may be toxic to scavenging bird species, includes ketoprofen, aceclofenac, carprofen, flunixin, and acetaminophen.

Avian Toxicity Tests

Risk assessment for effects on birds is based on the same principles that are used for all ecological risk assessments, i.e., it involves a comparison of the ratio between estimated toxicity and estimated exposure in order to predict risk. Both the US EPA, and the Organization for Economic Co-operation and Development (OECD) have validated three test guidelines (TG) that use birds as experimental animals: Avian Acute Oral Toxicity Test (EPA850.2100, OECD223), Avian Dietary Toxicity Test (EPA850.2200, OECD205), and Avian Reproduction Test (EPA850.2300, OECD206). These TG are included in the Office of Chemical Safety and Pollution Prevention (OCSPP) Series 850 (Ecological Effects TG), Group B from EPA, and in the OECD-TG Section 2 devoted to 'Effects on Biotic Systems.' OECD-TG are the most relevant internationally agreed test methods used by government, industry, and

independent laboratories to determine the safety of chemicals and chemical preparations, including pesticides and industrial chemicals. These tests are applicable to evaluate the hazards and risks of industrial chemicals and pesticides to terrestrial wildlife, directly or indirectly exposed. The information and data obtained from these tests are used in ecological risk assessment of pesticides, in assessments of potential off-target injury to endangered and threatened wildlife species, and when toxicity concerns arise from incidents.

In ecological risk assessment of pesticides, long-term toxicity studies to evaluate failures in reproduction are needed. In these tests, only studied species are recommended, such as Bobwhite quail (*Colinus virginianus*), mallard duck (*A. platyrhynchos*), or Japanese quail (*C. coturnix japonica*). In this sense, differences among species should be taken into account. In these long-term tests, it is important to obtain relevant data and information on feed consumption, and the caloric value of the food and its composition. The measurement of eggshell breaking strength, as well as the standard thickness measurement is important. Data on fertility, early embryo death, gross examination of the gonads, sex determination of all F1 chicks, and examination of adults (clinical signs during the test and necropsy and gross examination at the end of the study) are needed.

See also: Organophosphorus Insecticides; Carbamate Pesticides; Organochlorine Insecticides; DDT (Dichlorodiphenyltrichloroethane); Lead; Cadmium, Mercury; Selenium; Pesticides; Polybrominated Diphenyl Ethers; Polychlorinated Biphenyls (PCBs); Polycyclic Aromatic Hydrocarbons (PAHs); Pharmaceutical Effects in the Environment.

Further Reading

Beyer, W.N., Meador, J.P., 2011. Introduction. In: Beyer, W.N., Meador, J.P. (Eds.), *Environmental Contaminants in Biota. Interpreting Tissue Concentrations*, second ed. CRC Press, Taylor & Francis, Boca Ratón, pp. 1–8.

- Cuthbert, R., Taggart, M.A., Prakash, V., et al., 2011. Effectiveness of action in India to reduce exposure of *Gyps* vultures to the toxic veterinary drug diclofenac. *PLoS One* 6 (5), e19069. <http://dx.doi.org/10.1371/journal.pone.0019069>.
- Franson, J.C., Pain, D.J., 2011. Lead in birds. In: Beyer, W.N., Meador, J.P. (Eds.), *Environmental Contaminants in Biota. Interpreting Tissue Concentrations*, second ed. CRC Press, Taylor & Francis, Boca Ratón, pp. 563–593.
- García-Fernández, A.J., Calvo, J.F., Martínez-López, E., et al., 2008. Raptor ecotoxicology in Spain. A review on persistent environmental contaminants. *Ambio* 37, 432–439.
- García-Fernández, A.J., Espín, S., Martínez-López, E., 2013. Feather as biomonitoring tool of polyhalogenated compounds: a review. *Environ. Sci. Technol.* 47 (7), 3028–3043.
- Mineau, P., 2005. Ecotoxicology, avian. In: Wexler, P. (Ed.), *Encyclopedia of Toxicology*, second ed., vol. 2. Elsevier, pp. 121–126.
- Ohlendorf, H.M., Heinz, G.H., 2011. Selenium in birds. In: Beyer, W.N., Meador, J.P. (Eds.), *Environmental Contaminants in Biota. Interpreting Tissue Concentrations*, second ed. CRC Press, Taylor & Francis, Boca Ratón, pp. 669–702.
- Rattner, B.A., Scheuhammer, A.M., Elliot, J.E., 2011. History of wildlife toxicology and the interpretation of contaminant concentrations in tissues. In: Beyer, W.N., Meador, J.P. (Eds.), *Environmental Contaminants in Biota. Interpreting Tissue Concentrations*, second ed. CRC Press, Taylor & Francis, Boca Ratón, pp. 9–46.
- Thomas, P.J., Mineau, P., Shore, R.F., et al., 2011. Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada. *Environ. Int.* 37, 914–920.
- Wayland, M., Scheuhammer, A.M., 2011. Cadmium in birds. In: Beyer, W.N., Meador, J.P. (Eds.), *Environmental Contaminants in Biota. Interpreting Tissue Concentrations*, second ed. CRC Press, Taylor & Francis, Boca Ratón, pp. 645–668.

Relevant Websites

- <http://www.oecd.org/env/ehs/testing/oecdguidelinesforthetestingofchemicals.htm> – Avian Toxicity Test Guidelines from OECD.
- http://www.epa.gov/ocspp/pubs/frs/publications/Test_Guidelines/series850.htm – Avian Toxicity Test Guidelines from U.S. Environmental Protection Agency.
- http://ec.europa.eu/environment/endocrine/index_en.htm – How the European Commission addresses challenges posed by endocrine disruptors.
- <http://www.eurapmon.net/> – Research and monitoring for and with raptors in Europe (ESF research networking programme).
- <http://ourstolenfuture.org/> – The most recent knowledges about endocrine disruption and its relation to contaminants.