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REVIEW OF THE ECOLOGICAL EFFECTS OF POISONING ON MIGRATORY BIRDS

REPORT

Summary:

The present Report has been elaborated by the CMS Preventing Poisoning Working Group to serve as background information to the draft Resolution on "Preventing Poisoning of Migratory Birds" and the "Guidelines to Prevent Poisoning of Migratory Birds" submitted to COP11 in document UNEP/CMS/COP11/Doc.23.1.2.

The Report was tabled at the 18th Meeting of the Scientific Council (Bonn, 1-3 July 2014) as document UNEP/CMS/ScC18/Inf.10.9.1. After the discussion at the Council it has been reviewed and updated to incorporate the latest information.

Convention on the Conservation of Migratory Species of Wild Animals

Secretariat provided by the United Nations Environment Programme

UNEP/CMS Preventing Poisoning Working Group

Review of the ecological effects of poisoning on migratory birds

UNEP/Convention on Migratory Species
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Executive Summary

1. Introduction and scope

The project objectives, as defined by UNEP/CMS Resolution 10.26 (adopted in Bergen at the 2011 Conference of the Parties), are to undertake a detailed assessment of:

1. the scope and severity of poisoning for migratory bird species globally and how this varies geographically and across taxa;
2. significant knowledge gaps,[1] either across range states, or in specific areas; and
3. where sufficient evidence exists, to recommend suitable responses to address the problems, potentially including:
    i. areas where enhanced legislation may be required;
    ii. features of effective regulatory regimes; and
    iii. understanding of socio-economic drivers of poisoning.

This project, including the technical background review and guidelines with recommendations for adoption at the Conference of the Parties in 2014, will focus on migratory bird species. The priority categories of poisoning addressed by this study are those most likely to affect migratory bird populations in line with UNEP/CMS Resolution 10.26.[2]

These were selected by the working group at the workshop held in Tunisia in May 2013 using the criteria in the Toxins Matrix (Table 1) as well as identifying the socio-economic drivers of poisoning (Table 4). These are poison-baits, lead ammunition/shot, veterinary pharmaceuticals, agricultural insecticides and rodenticides. There are other potentially significant poisons that, while not covered in this initial review, could be covered in the next triennial period, subject to Scientific Council agreeing the remit and appropriate funding being found.

This review analyses direct lethal and sub-lethal poisoning with the potential to lead to population decline, (e.g., egg shell thinning resulting in reduced breeding success) to migratory birds through both deliberate poisoning and incidental/accidental poisoning. Direct lethal and sub-lethal effects can occur through *primary poisoning* (direct ingestion of poison) and *secondary poisoning* (when predators are exposed to physiologically damaging concentrations of poisons by eating contaminated prey, insects or worms).

Whether lethal and sub-lethal effects of poisoning are likely to lead to population declines is a function of (1) the likelihood that migratory birds will be exposed to poison; and (2) the toxicity of the poison to migratory birds. A global assessment on the scale and severity (likelihood of population effects) of each of the priority categories of poisoning on migratory birds is included in this review. A summary of the findings are discussed below.

[1] Significant knowledge gaps will include identification of (1) the extent of impacts; and (2) the range of different types of effects of poisons on migratory birds.

[2] http://www.cms.int/sites/default/files/document/10_26_poisoning_e_0_0.pdf.

2. Poison-baits

Predator control using poison-baits occurs on a global scale, particularly in areas with game management and livestock farming. Predator and scavenger bird species are at risk of poisoning from poison-baits targeting them directly, and also from baits targeting mammalian species (with birds becoming by-catch through secondary poisoning). The effects on species, other than birds of prey, is largely unknown and further research is needed to understand this.

The risk of poisoning from harvesting for human consumption and traditional medicine appears to be much more isolated. Using poisons to harvest migratory bird species for consumption and/or traditional medicine may be limited to particular areas in Africa and Asia.

Due to the indiscriminate nature of many of the substances used in poison-baits, any birds are at risk of poisoning if they come into contact with poison-baits. The most common substances are rodenticides and insecticides, usually those that are known to farmers in the area as highly toxic. Carbofuran appears to be used in poison-baits in many areas around the world.

Many birds of prey populations are in decline as a result of illegal poison-baits, particularly vultures. This suggests that further work needs to be developed to understand why poison-baits continue to be used and create effective solutions.

3. Lead ammunition/shot

Lead is highly toxic to birds causing, at higher concentrations mortality and at lower concentrations a range of sub-lethal impacts. Wherever there is anthropogenic use of lead which is available to migratory birds, poisoning can potentially occur. Thus, it should be noted that although surveillance and research reports on lead poisoning from most sources are mainly from North America and Europe, this is unlikely to reflect distribution of the problem.

Lead poisoning, whether primary or secondary, through ingestion of shot and bullets has been recorded in at least 20 countries with greatest reporting in North America and Europe. However, lead poisoning in migratory birds can be expected to occur wherever lead ammunition is used for hunting. It follows that wherever lead shot is used, it will accumulate within the environment and the degree of contamination will be directly proportional to the intensity of use.

Certain taxa, namely wildfowl and raptors, including threatened species, are more greatly affected than other groups of birds and losses can be high. Population effects are difficult to quantify for a number of reasons, including, lack of robust surveillance and gaps in knowledge of ingestion rates and subsequent survival. Sub-lethal impacts are particularly difficult to quantify. In most countries there are also gaps in knowledge of the efficacy of restrictive regulations.

The effects of lead poisoning from fishing weights on migratory birds are restricted to certain susceptible species and to certain geographical areas where discarded and lost weights are available. A number of migratory species are known to suffer from lead poisoning following the ingestion of discarded or lost lead fishing weights. In principle, most birds feeding in currently or historically fished water bodies or near-shore soils and sediments are at risk of being exposed to and ingesting lead. Species likely to feed in areas exposed to lead fishing weights and that have physiological mechanisms that assist lead absorption, are therefore, most at risk of suffering from lead ingestion

and poisoning. For these reasons, lead weight related poisoning has been widely reported in waterbirds. Although it is difficult to assess population-level effects of lead poisoning from fishing weights, there is some evidence that such effects can occur in species particularly sensitive to lead poisoning such as the mute swan and the common loon. Furthermore, significant mortality of threatened species from lead poisoning is a cause for concern.

4. Pesticides

Most bird species that use agricultural landscapes are in decline in Europe and North America as a result of the direct and indirect effects of land use intensification, habitat modification, pesticides, and other factors.[3] Often, these declines are related to intensification of management practices associated with the modernisation of agriculture.

Three quarters of all pesticides used are in agriculture.[4] The pesticide use often associated with modern agriculture can threaten ecosystem viability through a reduction in biodiversity (flora and fauna) and pollution of natural resources, such as groundwater, that impact human health and communities, as well as the natural environment. Indirect effects of pesticides on birds, such as the loss of habitat/cover and invertebrates, which lead to reduced feeding opportunities and breeding success, are well documented,[5] but will not be considered in detail here, as indirect effects are beyond the focus of this review. This study seeks to understand the scale and severity of the direct effects of pesticides on migratory birds.

Effects on birds arising unintentionally from the legal use of pesticides in agriculture are inherently variable.[6] However, insecticides and rodenticides are the main agricultural pesticides likely to result in direct lethal or sub-lethal poisoning of migratory birds (see Introduction and Scope).

4.1. Insecticides

Insecticides account for less than 20 per cent of pesticide use generally (in North America), but are more prevalent in developing countries.[7] Bird species that inhabit farmland or use farmland during migration are at risk of exposure to insecticides. Waterfowl and some gamebirds which feed on agricultural foliage are at potential risk. Granivorous passerines are attracted to pesticide-treated seeds. Birds that feed on agricultural pests, such as grasshoppers and earthworms, are at risk if

[3] Mineau, P., & Whiteside, M. (2006). Lethal risk to birds from insecticide use in the United States—a spatial and temporal analysis. *Environmental toxicology and chemistry*, *25*(5), 1214-1222 and Guerrero, I., Morales, M. B., Oñate, J. J., Geiger, F., Berendse, F., Snoo, G. D., ... & Tscharntke, T. (2012). Response of ground-nesting farmland birds to agricultural intensification across Europe: Landscape and field level management factors. *Biological Conservation*, *152*, 74-80.

[4] Sánchez-Bayo, F. (2011). Impacts of agricultural pesticides on terrestrial ecosystems. *Ecological Impacts of Toxic Chemicals. Bentham Science Publishers, Online*, 63-87.

[5] Devine, G. J., & Furlong, M. J. (2007). Insecticide use: contexts and ecological consequences. *Agriculture and Human Values*, *24*(3), 281-306.

[6] Hart, A. D. M. (2008). The assessment of pesticide hazards to birds: the problem of variable effects. *Ibis*, *132*(2), 192-204.

[7] Herbicides account for nearly half of the pesticides used in North America, insecticides 19%, fungicides 13%, with the remaining 22% including a variety of other products. Gianessi LP, Silvers CS. Trends in crop pesticide use: comparing 1992 and 1997: Office of Pest Management Policy, U.S. Department of Agriculture; 2000.

feeding on contaminated insects.[8] Scavengers and predators are poisoned when they consume contaminated prey.[9]

The likelihood of exposure to insecticides is influenced by a number of factors, including cultivation practices, pest types, crop types, pesticide form, and migratory bird ecology (diet and habitat preferences). Exposure may be reduced by using particular forms of pesticides, e.g., liquid forms over granular forms, and changing application periods for when migratory birds are not likely to be present (which can be effective given the low persistence of many of the second generation pesticides).

If a migratory bird is likely to be exposed, the toxicity of the pesticide is significant. The broad spectrum nature of organophosphates and carbamates (the most common insecticides) makes any bird at risk of lethal or sub-lethal effects if they happen to be in the vicinity at the time of application, or shortly thereafter, or if they come into contact with exposed prey.

Many of the highly toxic insecticides to birds, such as carbofuran, have been removed from the market in developed countries as a result of population declines in some bird species. Much of the effects, both sub-lethal and lethal, recorded in the literature are related to the use of these now highly regulated compounds. This could indicate that the situation has improved in areas where these highly toxic compounds are no longer used or that other substances have not yet been studied.

The implications of sub-lethal effects from exposure to second generation agricultural insecticides are little understood and are difficult to study in the field. Migratory birds may be particularly susceptible to sub-lethal effects from insecticides, which may cause reduced movement and affect migratory orientation. Further research should focus on assessing these effects on populations.

Neonicotinoids have become a main replacement for the carbamates and organophosphates in many countries. Further research is needed to understand their impacts on birds.

4.2. Rodenticides

Rodenticides are most commonly used for agricultural purposes, such as the protection of crops and grain storage from rodent pests. Anticoagulant rodenticides (ARs) are the most widely used rodenticide to control rodent pests worldwide.[10] They are also an integral component of modern agriculture for the control of rodent populations.[11]

Migratory birds are exposed to ARs through the consumption of contaminated baits (primary) or by the consumption of contaminated prey (secondary). Widespread exposure in birds to rodenticides has been detected through wildlife monitoring programmes in Europe and North America. For example, high detection rates of anticoagulant rodenticides have been reported in birds of prey

[8] Szabo, J. K., Davy, P. J., Hooper, M. J., & Astheimer, L. B. (2009). Predicting avian distributions to evaluate spatiotemporal overlap with locust control operations in eastern Australia. *Ecological Applications*, *19*(8), 2026-2037.

[9] Mineau, P. (2009). Birds and pesticides: is the threat of a silent spring really behind us? *Pesticides News*, (86), 12-18.

[10] Sánchez-Barbudo, I. S., Camarero, P. R., & Mateo, R. (2012). Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain. Science of the Total Environment, 420, 280-288.

[11] Tosh, D. G., Shore, R. F., Jess, S., Withers, A., Bearhop, S., Ian Montgomery, W., & McDonald, R. A. (2011). User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use. Journal of environmental management, 92(6), 1503-1508.

collected through wildlife monitoring programmes in New York (49 per cent of 265 raptors between 1998-2001),[12] France (73 per cent of 30 raptors, 2003),[13] Great Britain (37 per cent of 351 owls and kestrels, 2003-2005),[14] and Western Canada (70 per cent of 164 owls, 1988-2003).[15] However, birds submitted to monitoring programmes (e.g., dead birds found by members of the public) are unlikely to accurately represent the prevalence of exposure in the wild, as the sampling will be biased towards sick and dead individuals, and it is therefore difficult to estimate exposure rates for populations of migratory birds.

Birds that forage in agricultural landscapes are most likely to be exposed to anticoagulant rodenticides, as use of these products is primarily in agricultural areas. However, some species' ecology will make them more likely to be exposed than others within these areas. Many raptor species are especially likely to be exposed to rodenticides due to a regular diet of rodents. Scavenging species may be especially at risk because they feed on carcasses that could be contaminated with rodenticides. The red kite, for example, may be particularly susceptible to secondary poisoning because of the high proportion of carrion in its diet, including rat carcasses.[16]

If exposure to anticoagulant rodenticides is likely to occur, the toxicity level of the AR will greatly influence the corresponding effect – whether lethal or sub-lethal. The effects, particularly sub-lethal effects, of exposure to ARs on species at both the individual and population level remain poorly understood.[17] Sub-lethal exposure to second generation ARs (which are more commonly used and more toxic to birds than first generation ARs) may hinder the recovery of birds from non-fatal collisions or accidents. They may also impair hunting ability through behavioural changes, such as lethargy, thus increasing the probability of starvation. However, there is limited evidence of these effects occurring in the field. [18]

There is wide-spread exposure of raptors to rodenticides where second-generation anticoagulant rodenticides are used in agriculture, but the ecologically-significant effects (both lethal and sub-lethal) from exposure are largely unknown. Additionally, it is unknown whether there are any population level effects from exposure. There is also scant knowledge of SGAR exposure rates in birds outside Europe, North America and Australasia.

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12 Stone, W. B., Okoniewski, J. C., & Stedelin, J. R. (2003). Anticoagulant rodenticides and raptors: recent findings from New York, 1998–2001. *Bulletin of environmental contamination and toxicology*, *70*(1), 0034-0040.

13 Lambert, O., Pouliquen, H., Larhantec, M., Thorin, C., & L'Hostis, M. (2007). Exposure of raptors and waterbirds to anticoagulant rodenticides (difenacoum, bromadiolone, coumatetralyl, coumafen, brodifacoum): epidemiological survey in Loire Atlantique (France). *Bulletin of environmental contamination and toxicology*, *79*(1), 91-94.

14 Walker, L. A., Turk, A., Long, S. M., Wienburg, C. L., Best, J., & Shore, R. F. (2008). Second generation anticoagulant rodenticides in tawny owls (Strix aluco) from Great Britain. *Science of the Total Environment*, *392*(1), 93-98.

15 Albert, C. A., Wilson, L. K., Mineau, P., Trudeau, S., & Elliott, J. E. (2010). Anticoagulant rodenticides in three owl species from western Canada, 1988–2003. *Archives of environmental contamination and toxicology*, *58*(2), 451-459.

16 Carter, I., & Burn, A. (2000). Problems with rodenticides: the threat to red kites and other wildlife. British Wildlife, 11(3), 192-197.

17 Burn, A. J., Carter, I., & Shore, R. F. (2002). The threats to birds of prey in the UK from second-generation rodenticides. *Aspects of Applied Biology*, *67*, 203-212; Knopper, L. D., Mineau, P., Walker, L. A., & Shore, R. F. (2007). Bone density and breaking strength in UK raptors exposed to second generation anticoagulant rodenticides. *Bulletin of environmental contamination and toxicology*, *78*(3), 249-251.

18 Thomas, P. J., Mineau, P., Shore, R. F., Champoux, L., Martin, P. A., Wilson, L. K., ... & Elliott, J. E. (2011). Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada. *Environment international*, *37*(5), 914-920.

In addition to research needed to determine whether there are population effects resulting from widespread exposure in some species, further research is also needed to identify the exposure rate of rodenticides in species other than raptors as some evidence indicates that grain-based baits could result in exposure of granivorous bird species.

5. Veterinary pharmaceuticals (NSAIDs)

Non-steroidal anti-inflammatories (NSAIDs) are used to treat domestic livestock for inflammation and pain relief. Diclofenac, a previously popular NSAID for veterinary care of cattle in India, Pakistan, Bangladesh, and Nepal, is toxic to a number of vulture species and *Aquila* eagles. It resulted in the poisoning of scavenging vultures throughout India, Pakistan, Bangladesh and Nepal by contaminating domestic livestock carcasses traditionally fed on by vultures. Prior to the ban of diclofenac in these countries, it was prevalent in livestock carcasses and caused substantial population declines of three Gyps vulture species in South Asia. Research is ongoing to determine the effectiveness of the ban. Diclofenac has now been licensed for use in some European countries and presents a risk to various raptor species there.

The use of diclofenac in regions outside South Asia, such as Europe, may pose a risk of poisoning to other vultures and *Aquila* eagles which are present on a number of continents. For example, the promotion of diclofenac on the African continent could pose a risk to vultures in this region, including the African white-backed vulture (*Gyps africanus*) and the endangered Cape Griffon vulture (*Gyps coprotheres*) due to these species' sensitivity to diclofenac. Although, exposure levels may be different in Africa, through, for example, the removal of cattle carcasses from open areas and variation in vulture diet.

The next steps are to (1) evaluate the effects of other NSAIDs on birds of prey/scavenging birds; (2) identify vulture-safe alternatives (so far only meloxicam has been shown to have low toxicity to *Gyps* vultures); (3) determine whether diclofenac/NSAIDs are toxic to other vultures and birds of prey; and (4) assess the effects of diclofenac/NSAIDs on vultures in areas outside South Asia, especially in areas where domestic ungulate carcasses are likely to be available for scavenging.

Introduction and project scope

1. Project objectives

The project objectives, as defined by UNEP/CMS Resolution 10.26 (agreed at the 2011 Conference of the Parties), are to undertake a detailed assessment of:

1. the scope and severity of poisoning for migratory bird species globally and how this varies geographically and across taxa;
2. significant knowledge gaps,[19] either across range states, or in specific areas; and
3. where sufficient evidence exists, to recommend suitable responses to address the problems, potentially including:
    i. areas where enhanced legislation may be required;
    ii. features of effective regulatory regimes, and
    iii. understanding of socio-economic drivers of poisoning.

In addition to regulatory changes, an assessment will be done of areas where enhanced education and awareness raising, voluntary codes and best practice may be useful.

2. What is poisoning for the purposes of this review?

This review addresses poisoning with the potential to cause direct lethal effects and/or sub-lethal effects with the potential to lead to population decline of migratory birds through both deliberate poisoning and accidental/incidental poisoning. Deliberate poisoning results from the intentional use of poisons to target migratory birds – generally illegal actions intended to harm or exploit migratory birds.[20] Incidental poisoning arises from the mis-use of poisons or through labelled/routine use with insufficient precautions (or where risk assessment didn't capture the full extent of the risk on migratory birds post-registration) taken during a generally legal activity (eg, rodent control) that results in harm to migratory birds.

Both primary and secondary sources of poisoning are included in this study. Primary poisoning occurs as a result of direct ingestion of poison;[21] whereas, secondary poisoning occurs when predators are exposed to physiologically damaging concentrations of poisons by eating contaminated prey.[22]

[19] Significant knowledge gaps will include identification of (1) the extent of impacts; and (2) the range of different types of effects of poisons on migratory birds.

[20] As defined by *Mahler v United States Forest Service*, 927 F Supp at 1579 in relation to the Migratory Bird Treaty Act (MBTA) in the United States.

[21] Sánchez-Bayo, F. (2011). Impacts of agricultural pesticides on terrestrial ecosystems. *Ecological Impacts of Toxic Chemicals. Bentham Science Publishers, Online*, 63-87.

[22] S purgeon, D. J., & Hopkin, S. P. (1996). Risk assessment of the threat of secondary poisoning by metals to predators of earthworms in the vicinity of a primary smelting works. Science of the total environment, 187(3), 167-183.

3. What toxins will be covered in this study?

The priority categories of poisoning addressed by this study are those most likely to affect migratory bird populations in line with UNEP/CMS Resolution 10.26.[23] These were selected by the working group at the workshop held in Tunisia in May 2013 using the criteria in the Toxins Matrix (see Appendix I). These are poison-baits, lead ammunition/shot, veterinary pharmaceuticals, agricultural insecticides and rodenticides. There are other potentially significant poisons that, while not covered in this initial review, could be covered in the next triennial period, subject to Scientific Council agreeing the remit and appropriate funding being found.

4. Study limitations

Some types of poisoning affecting migratory birds will be excluded from this study due to occurrence in limited or isolated instances (less likely for population impact), and/or because they fall outside the definition of poisoning used in this project. A relatively tight definition has been chosen for practical reasons, to allow resource to be focussed in a realistic way on delivering against the mandate established by UNEP/CMS Resolution 10.26. Sources of poisoning excluded from this study:

- *Poisoning as a result of oil spills, industrial chemicals, eg, those released into the air and water, irrigation and drain water contaminants, and general pollution* to migratory birds – effects may be significant, but it is outside the definition of poisoning used for this limited study; for example, PCBs as industrial chemicals are less toxic to birds than mammals.[24]
- *Poisoning as a result of heavy metals*, such as by-products of mining stored in "sludge ponds" (other than lead ammunition and sinkers) – instances of poisoning on migratory birds are relatively infrequent and are often diffused into soil and water (classified as pollution, which is excluded by this study).
    - With the exception of lead, concentrations of cadmium, mercury and selenium in eggs and tissues appear to be below toxic thresholds for waterbirds in the United States.[25]
    - Lead ammunition and fishing weights are the primary sources of lead poisoning for birds. Other sources of lead, such as activities relating to mining, can also cause poisoning of birds, but are more limited (see Appendix II).
- *Poisoning as a result of human disease control*, e.g., malaria and other vector-borne diseases (alternative solution not readily available/tractable at this time).
- *Poisoning of non-target species arising from invasive species management/eradication programmes* – generally has a limited negative effect on migratory species populations compared to the beneficial contribution to biodiversity conservation objectives of the removal of invasive alien species.[26] Best practice guidance exists to minimise risk of

[23] http://www.cms.int/sites/default/files/document/10_26_poisoning_e_0_0.pdf.

[24] Rattner, B. A., & McGowan, P. C. (2007). Potential hazards of environmental contaminants to avifauna residing in the Chesapeake Bay estuary. *Waterbirds*, *30*(sp1), 63-81.

[25] Rattner, B. A., & McGowan, P. C. (2007). Potential hazards of environmental contaminants to avifauna residing in the Chesapeake Bay estuary. *Waterbirds*, *30*(sp1), 63-81.

[26] Pimentel, D., Zuniga, R., & Morrison, D. (2005). Update on the environmental and economic costs associated with alien-invasive species in the United States. *Ecological economics*, *52*(3), 273-288; Fisher, P., Griffiths, R., Speedy, C., & Broome, K. (2011). Environmental monitoring for brodifacoum residues after aerial application of baits for rodent eradication. *Island invasives: eradication and management. IUCN (International Union for Conservation of Nature), Gland, Switzerland*, 278-282; and Hoare, J. M., & Hare, K. M. (2006). The impact of brodifacoum on non-target wildlife: gaps in knowledge. *New Zealand Journal of Ecology*, *30*(2), 157-167.

poisoning in invasive species management.[27] Further study on the adherence to best practice could be promoted; and a minimum baseline of compliance with best practice could be established through, for example, the proposed European Invasive Species Directive.

This study excludes the indirect effect of poisons in ecosystems on migratory birds, such as the reduction in availability of particular food species or other resources utilised by migratory birds.[28]

4.1. Certainty of evidence

This study will apply the precautionary principle in determining treatment of cases where the evidence is less than certain. Where uncertainty exists about population level impacts as a result of poisoning, but the risk of harm to migratory birds is significant, the poisoning issue may still be addressed by this review.

5. What species will be included in this study?

This project, including the technical background review and guidelines with recommendations for adoption at the Conference of the Parties in 2014, will focus on migratory bird species. Migratory bird species, for the purposes of this project, are those where a significant proportion of the population "cyclically and predictably cross one or more national jurisdictional boundaries."[29] Specifically, the project will focus on those species listed on the CMS Appendices, given those are of highest conservation concern. Examples of poisoning of non-migratory species may be included where it can be reasonably be postulated that the mechanism of poisoning does or could also impact migratory species.

[27] Best practice guidance is available through the Pacific Invasives Initiative: http://www.pacificinvasivesinitiative.org/rk/index.html (accessed on 10 May 2013).

[28] Herbicides and avermectin residues (used as worming livestock agents) affect birds indirectly by reducing food abundance (Vickery JA, et al, The management of lowland neutral grasslands in Britain: effects of agricultural practices on birds and their food resources, Journal of Applied Ecology 38(3): 647-664, 2001.).

[29] Convention on Migratory Species, Article 1.1(a).

Effects of crop protection using insecticides

1. Introduction

Most bird species that use agricultural landscapes are in decline in Europe and North America as a result of the direct and indirect effects of land use intensification, habitat modification, pesticides, and other factors.[30] Often, these declines are related to intensification of management practices associated with the modernisation of agriculture. The pesticide use usually associated with modern agriculture can threaten ecosystem viability through a reduction in biodiversity (flora and fauna) and pollution of natural resources, such as groundwater, that impact human health and communities, as well as the natural environment.

Indirect effects of pesticides on birds, such as the loss of habitat/cover and invertebrates which lead to reduced feeding opportunities and breeding success, are well documented,[31] but will not be considered in detail here, as indirect effects are beyond the focus of this review. This study seeks to understand the scale and severity of the direct effects of pesticides on migratory birds.

Conventional modern agriculture uses irrigation, synthetic pesticides, and fertilisers to increase crop yields thus leading to an intensification of agricultural practices. Approximately 40 per cent of global food production is supported by conventional modern agriculture.[32] The FAO projects that if current patterns in food consumption persist, globally 60 per cent more food will need to be produced by 2050 (compared with 2005-2007).

The area treated with insecticides has remained static over the last decade for the developed world (although, there have been changes in the types of pesticides used). However, pesticide usage in developing countries is expected to increase with the growth of food production in those countries.[33]

1.1. Net costs and benefits of pesticides

Without pesticides, crop yields could drop by as much as a third and food prices could increase by as much as 75 per cent.[34] Yields from organic plots are often competitive with those of conventional plots, but are more unpredictable and labour intensive.[35] Investment in pesticides is economically beneficial – in US agriculture systems, each $1 invested in pesticide management returns $4.[36]

[30] Mineau, P., & Whiteside, M. (2006). Lethal risk to birds from insecticide use in the United States—a spatial and temporal analysis. *Environmental toxicology and chemistry*, *25*(5), 1214-1222 and Guerrero, I., Morales, M. B., Oñate, J. J., Geiger, F., Berendse, F., Snoo, G. D., ... & Tscharntke, T. (2012). Response of ground-nesting farmland birds to agricultural intensification across Europe: Landscape and field level management factors. *Biological Conservation*, *152*, 74-80.

[31] Devine, G. J., & Furlong, M. J. (2007). Insecticide use: contexts and ecological consequences. *Agriculture and Human Values*, *24*(3), 281-306.

[32] FAO 2003.

[33] Davies, I. (2005) UK Self-sufficiency drops by 12%. In *Farmers Weekly*, 4 February 2005.

[34] Trewavas, A. (2001). Urban myths of organic farming. *Nature*, *410*(6827), 409-410.

[35] Pimentel, D., Hepperly, P., Hanson, J., Douds, D., & Seidel, R. (2005). Environmental, energetic, and economic comparisons of organic and conventional farming systems. *BioScience*, *55*(7), 573-582.

[36] Pimentel, Environmental and economic costs of the application of pesticides primarily in the United States.

However, these figures exclude the environmental and social costs of pesticide use, which are estimated to be $12 billion in the US alone: $2.2 billion for bird, fish and other wildlife losses; $2 billion for water monitoring and pesticide clean-up; $1.1 billion crop losses caused by pesticides; $1.5 billion pesticide resistance in pests; $1.1 billion for public health.[37] These costs amount to 4 per cent of farm revenues.[38] Pesticide management attempts to minimise these costs.[39]

1.2.Agricultural pesticides of risk to migratory birds

Insecticides and rodenticides are the key pesticides of risk of direct harm to birds (see Introduction). Rodenticides are covered in a separate section herein. Insecticides account for less than 20 per cent of pesticide use generally (in North America), but are more prevalent in developing countries.[40] Bird species that inhabit farmland or use farmland during migration are at risk. Waterfowl and some gamebirds which feed on agricultural foliage are at potential risk. Granivorous passerines are attracted to pesticide-treated seeds. Birds that feed on agricultural pests, such as grasshoppers and earthworms, are at risk if feeding on contaminated insects. Scavengers and predators are poisoned when they consume contaminated prey.[41]

1.3.Mode of poisoning

Agricultural pesticides are typically applied directly to crops in a liquid spray form, as granules buried in the soil, or as seed dressings. The form of application greatly determines the exposure rate.[42] The effect of the form of pesticides is discussed below. Birds may incorporate pesticides through ingestion (either directly or through consuming contaminated species), inhalation, or eye or skin contact.[43]

- *First generation insecticides (organochlorines)*: mode of action in birds is generally presented through reproductive effects, such as eggshell thinning, or acute mortality.
- *Second generation insecticides (organophosphates and carbamates)*: mode of action is cholinesterase-inhibiting, which causes neurological effects leading to lethal or sub-lethal effects.

Small amounts of these chemicals can cause sub-lethal effects, such as reduced activity in birds, which spend more time resting or perching than foraging or reproducing.[44] For example, raptors that consume high levels of these substances lose the ability to fly and coordinate muscles[45] until paralysis of the respiratory muscles causes death.[46]

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37 Ibid.

38 Färe, R, Grosskopf, S., & Weber, W. L. (2006). Shadow prices and pollution costs in US agriculture. *Ecological economics*, *56*(1), 89-103.

39 See, for example, Reichenberger, S., Bach, M., Skitschak, A., & Frede, H. G. (2007). Mitigation strategies to reduce pesticide inputs into ground-and surface water and their effectiveness; A review. *Science of the Total Environment*, *384*(1), 1-35; Hoy, M. A., & Hoy, M. A. (1998). Myths, models and mitigation of resistance to pesticides. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *353*(1376), 1787-1795.

40 Herbicides account for nearly half of the pesticides used in North America, insecticides 19%, fungicides 13%, with the remaining 22% including a variety of other products. Gianessi LP, Silvers CS. Trends in crop pesticide use: comparing 1992 and 1997: Office of Pest Management Policy, U.S. Department of Agriculture; 2000.

41 Mineau, P. (2009). Birds and pesticides: is the threat of a silent spring really behind us? *Pesticides News*, (86), 12-18.

42 Sánchez-Bayo, *Impacts of agricultural pesticides on terrestrial ecosystems*.

43 Pisani, J. M., Grant, W. E., & Mora, M. A. (2008). Simulating the impact of cholinesterase-inhibiting pesticides on non-target wildlife in irrigated crops. Ecological Modelling, 210(1), 179-192.

44 Walker, C. H. (2003). Neurotoxic pesticides and behavioural effects upon birds. *Ecotoxicology*, *12*(1), 307-316.

45 Ostrowski, S., & Shobrak, M. (2001). Poisoning by acetylcholinesterase inhibiting pesticides in free-ranging raptors: a case reported from Saudi Arabia. *Falco*. The newsletter of the Middle East Falcon Research Group 20:8-9.

46 Fleischli, M. A., Franson, J. C., Thomas, N. J., Finley, D. L., & Riley, W. (2004). Avian mortality events in the United States caused by anticholinesterase pesticides: a retrospective summary of National Wildlife Health Center records from 1980 to 2000. *Archives of environmental contamination and toxicology*, *46*(4), 542-550.

1.4.Scope of poisoning via agricultural pesticides

The main threat to bird populations from pesticide use will, in most cases, be via the removal of their food sources from the countryside. This issue will not be considered further in the current paper, which focuses on risks of direct poisoning.

This study is limited to the direct effects of pesticide poisoning – therefore, only species that use agricultural landscapes for breeding, migration stopovers, or wintering habitats, or bird species that feed on species that have been in contact with pesticides in agricultural areas will be addressed herein. It excludes the indirect effects of pesticides that, as a result of soil run-off, erosion, spray-drift (and others), contaminate freshwater resources and the sea. However, these factors must be considered in the development of solutions to minimise poisoning.

1.5.Availability of pesticides toxic to birds

The availability of particular pesticides for agricultural use is generally regulated at a national level (and European Union level), but global regulation of pesticides is growing.[47] Effects of pesticides on wildlife are often considered before registration is authorised and many regimes require post-registration consideration of effects (eg, Canada). However, some countires, such as Japan, do not require consideration of effects on birds before being able to register a new pesticide. The regulation of the use of insecticides is not always effective in minimising the risk to wildlife, and can be scant in developing countries. Therefore, pesticides toxic to birds are often readily available for use.

1.6.Population-level effects

Effects on birds arising unintentionally from the approved use of pesticides in agriculture are inherently variable.[48] One study estimated between 0.25 and 8.9 birds per hectare of agricultural area are killed each year by pesticides.[49] It is unknown how much of this is due to mis-use of the pesticide labelled requirements. Labelled uses (legal use per label requirements) of pesticides in North America, Canada and the United Kingdom contributed to 181/736, 92/126, and 7/136, respectively, of documented raptor deaths reported by the specific country monitory schemes between 1985 and 1995.[50]

The vast majority of insecticide poisoning events are likely to go unrecorded or reported.[51] Additionally, scavenging of carcasses may bias estimates of mortality following the use of pesticides.[52] There are also problems with the detection of poisoned birds which may travel far from

[47] Hough, P. (2003). Poisons in the system: the global regulation of hazardous pesticides. Global Environmental Politics, 3(2), 11-24.

[48] Hart, A. D. M. (2008). The assessment of pesticide hazards to birds: the problem of variable effects. *Ibis*, *132*(2), 192-204.

[49] Pimentel, D., Acquay, H., Biltonen, M., Rice, P., Silva, M., Nelson, J. & D'amore, M. (1992). Environmental and economic costs of pesticide use. *BioScience*, *42*(10), 750-760.

[50] Mineau, P., Fletcher, M. R., Glaser, L. C., Thomas, N. J., Brassard, Candace, Wilson, L. K., & Porter, S. L. (1999). Poisoning of raptors with organophosphorus and carbamate pesticides with emphasis on Canada, US and UK. *Journal of Raptor Research*, *33*, 1-37.

[51] Kostecke, R. M., Linz, G. M., & Bleier, W. J. (2001). Survival of avian carcasses and photographic evidence of predators and scavengers. *Journal of Field Ornithology*, *72*(3), 439-447.

[52] Kostecke et al., *Survival of avian carcasses and photographic evidence of predators and scavengers*.

place of exposure, and often fields are left alone after application of pesticides further inhibiting the likelihood of detection.[53]

Whether these incidents are sufficient to cause population decline is unknown, which is dependent on exposure and toxicity.[54] This section reviews whether agricultural pesticides are causing, incidentally, population-level decline of migratory birds, which is a function of (1) the likelihood that migratory birds will be exposed to pesticides; and (2) toxicity of pesticides to migratory birds. Each of these components is discussed in more detail below.

2. Likelihood of exposure

The likelihood of exposure is influenced by a number of factors, including cultivation practices, pest types, crop types, pesticide form, and migratory bird ecology (diet and habitat preferences), which are each discussed below.

2.1. Cultivation practices, such as timing of application, may influence likelihood of exposure

Agricultural cultivation practices influence exposure rate of birds. In one study, the exposure of ducks was correlated with the timing of insecticide applications to agricultural fields.[55] Most second generation pesticides have limited persistence, although some have been shown to last a few months in certain situations, such as water-logged fields,[56] and therefore the likelihood of exposure is increased when birds are present within a few days or weeks of application.

Pesticide exposure can vary with seasons; for example, regular die-offs of raptors occurred every spring in Switzerland when crops were sown. This was linked to the general agricultural practice to simultaneously apply granular carbofuran for seed protection at planting.[57] The application of insecticides in North American canola fields in mid-to-late May to early-June coincides with the intensive use of canola fields by migratory songbirds and geese.[58]

2.2. Pest types that increase risks of insecticide poisoning of birds

Species that rely on particular types of insect pests may be more likely to be exposed to insecticides because of, for example, (1) the insect's behavioural reaction to particular substances making it more likely to be preyed upon; (2); insects that are the target of particular pesticides make up a large proportion of a bird's diet; and (3) the insect occurs in relative abundance at particular times (eg, pest outbreaks) making it more likely that a large quantity of that pest to be ingested by birds.

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53 Dietrich, D. R., Schmid, P., Zweifel, U., Schlatter, C., Jenni-Eiermann, S., Bachmann, H., ... & Zbinden, N. (1995). Mortality of birds of prey following field application of granular carbofuran: a case study. *Archives of Environmental Contamination and Toxicology*, *29*(1), 140-145.

54 Schmolke, A., Thorbek, P., Chapman, P., & Grimm, V. (2010). Ecological models and pesticide risk assessment: current modeling practice. *Environmental Toxicology and Chemistry*, *29*(4), 1006-1012.

55 Osten, J. R. V., Soares, A. M., & Guilhermino, L. (2005). Black-bellied whistling duck (*Dendrocygna autumnalis*) brain cholinesterase characterization and diagnosis of anticholinesterase pesticide exposure in wild populations from Mexico. *Environmental toxicology and chemistry*, *24*(2), 313-317.

56 Mineau et al., *Poisoning of raptors with organophosphorus and carbamate pesticides with emphasis on Canada, US and UK*.

57 Dietrich, D. R., Schmid, P., Zweifel, U., Schlatter, C., Jenni-Eiermann, S., Bachmann, H., ... & Zbinden, N. (1995). Mortality of birds of prey following field application of granular carbofuran: a case study. *Archives of Environmental Contamination and Toxicology*, *29*(1), 140-145.

58 Mineau, P., Downes, C. M., Kirk, D. A., Bayne, E., & Csizy, M. (2005). Patterns of bird species abundance in relation to granular insecticide use in the Canadian prairies. *Ecoscience*, *12*(2), 267-278.

The presence of these insects may increase the likelihood of exposure to some species:[59]

- *Earthworms:* carbamates are particularly toxic to earthworms. Earthworms exposed to carbamates are more likely to attract predators because they often exhibit violent coiling behaviour at the soil surface after exposure, increasing their visibility. Species, that regularly feed on earthworms are therefore more likely to be poisoned as a result of carbamate use. This has been documented in birds of prey such as buzzards and kites.[60]

- *Locusts and grasshoppers*: locust and grasshopper control differs from ordinary crop protection in that spraying operations often take place in habitats outside of croplands. Also, application must be conducted on a large scale to be effective. This means that species that forage outside of agricultural fields may also be exposed.

Outbreaks of pests attract birds and so may increase the likelihood of exposure of birds to pesticides. Many species of raptor are killed through consumption of contaminated invertebrates. European species such as black kites (*Milvus migrans*) feed on locusts in the Sahel or Southern Africa and North American Swainson's hawks (*Buteo swainsoni*) that historically fed on locusts now eat grasshoppers and other insect species in Argentina. The Swainson's hawk may be particularly vulnerable to poisoning because of its ability to target pest outbreaks in agricultural crops.[61] For example, grasshopper control in Argentina using the organophosphate monocrotophos killed at least 5,000 Swainson's hawks during the austral summer of 1995-1996.[62] Most pesticides are applied as a preventative measure, but those that target outbreaks of pests may pose more risk to bird species.

Spraying of carbofuran for grasshopper control resulted in the disappearance of burrowing owls in Canada, but the use of carbaryl, a grasshopper insecticide of lower acute toxicity, did not lead to mortalities.[63] The rate of application of carbofuran implicated in the owls' disappearance was one of the lowest rates registered anywhere in the world.

Highly mobile pests are more likely to be poisoned, which increases the risk to birds that feed on those pests. For example, waterfowl feeding on those pests were disproportionally killed through secondary poisoning.[64]

2.3.Crops that increase likelihood of exposure

Particular types of crops may increase likelihood of exposure to birds. Some crops are associated with use of pesticides that are more acutely toxic to birds and/or particular forms of pesticides that are more likely to result in exposure. Additionally, some crops may be associated with cultivation practices of more frequent applications or quantities of pesticides than others. Other types of crops are more attractive to birds as foraging areas, either due to the direct palatability of the crop as a food source, or because of other resources likely to be present within that particular crop.

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59 Note, the literature is very raptor focussed and this may not be a true indication of the species variation for the effects of pest types on exposure in birds.

60 Mineau et al., *Poisoning of raptors with organophosphorus and carbamate pesticides with emphasis on Canada, US and UK*.

61 Ibid.

62 Goldstein, M. I., Lacher, T. E., Woodbridge, B., Bechard, M. J., Canavelli, S. B., Zaccagnini, M. E., ... & Hooper, M. J. (1999). Monocrotophos-induced mass mortality of Swainson's Hawks in Argentina, 1995–96. *Ecotoxicology*, *8*(3), 201-214.

63 Fox, G. A., Mineau, P., Collins, B., & James, P. C. (1989). *The impact of the insecticide carbofuran (Furadan 480F) on the burrowing owl in Canada*. Environment Canada, Canadian Wildlife Service.

64 Osten et al., *Black-bellied whistling duck (Dendrocygna autumnalis) brain cholinesterase characterization and diagnosis of anticholinesterase pesticide exposure in wild populations from Mexico*.

For example, rice is one of the crops with the highest cumulative risk to birds. Ten percent of rice cultivation areas receive pesticide treatments likely to cause avian deaths in the United States.[65]

Rice fields and turf grass farms provide important alternative wintering habitat and migratory stopover habitats for shorebirds. These areas pose a risk of exposure to organophosphates and carbamates to birds using them as stopover sites. This risk is increased by the loss of natural wetland habitats, with rice fields becoming attractive alternatives.[66]

2.4.Forms of insecticides that increase risk of exposure

2.4.1.Granular forms

Granular insecticides are extremely concentrated sources of insecticides, commonly used in agriculture to protect crops from particular pests, and are often implicated in the deaths of songbirds, shorebirds and waterfowl (as well as small mammals). There are several means by which birds may be exposed to granular pesticides, but of particular concern is the possibility that birds may ingest the granules either inadvertently or intentionally. The granules are usually buried below the soil surface during application, but often some particles are left uncovered.[67]

Seed-eating birds and birds foraging for grit may be at risk of exposure to granular pesticides through primary ingestion.[68] Granular insecticides are particularly attractive to songbirds, either as grit or as food.[69] This route of exposure may also impact on waterfowl ingesting grit to aid digestion. Waterfowl are exposed to granular insecticides when they sift sediments and crop residues in puddles and waterlogged soils, or through accidental ingestion when picking up grit. Extensive kills of waterfowl have occurred in potato and root crops, and in partially flooded corn, winter wheat and rice fields in the US and Canada.[70] Primary poisoning occurs in birds and mammals by ingestion of organophosphate and carbamate granules or coated seeds. [71] Granular insecticides also lead to secondary poisoning of other bird species that consume birds or other prey which have ingested or been exposed to the granules. [72] The most common form of secondary poisoning in raptors is associated with granular insecticides. Carbofuran granules have been shown to cause regional population declines in some species (see Figure 1) and have therefore been removed from the market for agricultural purposes in a number of developed countries.

65 Parsons, K. C., Mineau, P., & Renfrew, R. B. (2010). Effects of pesticide use in rice fields on birds. *Waterbirds*, *33*(sp1), 193-218.

66 Strum, K. M., Alfaro, M., Haase, B., Hooper, M. J., Johnson, K. A., Lanctot, R. B., ... & Zaccagnini10, M. E. (2008). Plasma cholinesterases for monitoring pesticide exposure in Nearctic-Neotropical migratory shorebirds. *Ornitología Neotropical*, *19*, 641-651.

67 Erbach, D. C., & Tollefson, J. J. (1991). Application of corn insecticide granules to minimize hazard to birds. *Applied engineering in agriculture*, *7*.

68 Best, L. B. (1995). Grit-Use Behavior in Birds: A Review of Research to Develop Safer Granular Pesticides. In *National Wildlife Research Center Repellents Conference 1995* (p. 6).

69 Best, L. B., & Fischer, D. L. (1992). Granular insecticides and birds: factors to be considered in understanding exposure and reducing risk. *Environmental toxicology and chemistry*, *11*(10), 1495-1508.

70 Mineau et al., *Poisoning of raptors with organophosphorus and carbamate pesticides with emphasis on Canada, US and UK*.

71 Sánchez-Bayo, F., Tennekes, H. A., & Goka, K. (2013). Impact of Systemic Insecticides on Organisms and Ecosystems.

72 Ibid.

Figure 1: Examples of bird poisoning from granular insecticides

Some of the largest mortality incidents related to carbofuran granules have occurred in horned larks (*Eremophila alpestris*) (800 individuals) and more than 2,000 Lapland longspurs (*Calcarius lapponicus*) in the Canadian prairies. Regional population effects were found in horned lark and house sparrow (*Passer domesticus*) from granular carbofuran used in Canadian agriculture. At the peak of carbofuran's popularity in the early-1980s, a sand-based formulation caused the annual death of between 17-91 million songbirds and waterfowl in the US corn belt. [73]

In the UK between 1985 and 1995, there were very few raptor deaths as a result of labelled pesticide use, but three out of four incidents associated with labelled use were caused by granular carbofuran.[74]

The hazard associated with granular insecticides may be more dependent on which species (e.g., size and feeding behaviour) inhabit a treated area than on the actual application rate.[75]

2.4.2. Seeds

In the United Kingdom, one study found some seed treatments will poison birds, but only occasionally under particular circumstances. Whether poisoning from treated seeds will occur is dependent on the area sown, toxicity of the pesticide, its concentration on the seed, density of the exposed seed, availability of other foods, and ability of the birds to selectively avoid treated seed.

The results showed that fonofos-treated wheat (now withdrawn by manufacturer in the UK) will poison birds, but only when both rapid feeding is possible and there is a high concentration of residues on the seeds. This is most likely to occur when seed is split before sowing, as this produces dense patches of seed which enables rapid feeding.[76] Another study estimated the rate of poisoning by insecticide-treated seeds in a bird population resulting in morality from exposure was likely to lie in the range 0-5 per cent.[77]

2.4.3. Liquid forms

The effect of spray-form insecticides on mortality of birds is dependent on its toxicity (addressed in the next section) and its rate of application.[78]

A study on the effects of repeated liquid sprays of methiocarb (a carbamate) found only sub-lethal effects on the dozens of bird species monitored in a UK orchard.[79] However, the extent of sub-lethal effects on overall population health was not assessed in that study.

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[73] Mineau, P., Downes, C. M., Kirk, D. A., Bayne, E., & Csizy, M. (2005). Patterns of bird species abundance in relation to granular insecticide use in the Canadian prairies. *Ecoscience*, *12*(2), 267-278; Balcomb, R., Bowen, C. A., Wright, D., & Law, M. (1984). Effects on wildlife of at-planting corn applications of granular carbofuran. *The Journal of wildlife management*, 1353-1359.

[74] Mineau et al., *Poisoning of raptors with organophosphorus and carbamate pesticides with emphasis on Canada, US and UK*.

[75] Hill, E. F., & Camardese, M. B. (1984). Toxicity of anticholinesterase insecticides to birds: technical grade versus granular formulations.*Ecotoxicology and environmental safety*, *8*(6), 551-563.

[76] Hart, A., Fryday, S., McKay, H., Pascual, J., & Prosser, P. (1999). Understanding risks to birds from pesticide-treated seeds. In *Proceedings of the 22nd International Ornithology Congress* (pp. 1070-1087).

[77] Prosser, P. J., Hart, A. D., Langton, S. D., McKay, H. V., & Cooke, A. S. (2006). Estimating the rate of poisoning by insecticide-treated seeds in a bird population. *Ecotoxicology*, *15*(8), 657-664.

[78] Mineau, P. (2002). Estimating the probability of bird mortality from pesticide sprays on the basis of the field study record. *Environmental Toxicology and Chemistry*, *21*(7), 1497-1506.

[79] Hardy, A. R., Westlake, G. E., Lloyd, G. A., Brown, P. M., Greig-Smith, P. W., Fletcher, M. R., ... & Stanley, P. I. (1993). An intensive field trial to assess hazards to birds and mammals from the use of methiocarb as a bird repellent on ripening cherries. *Ecotoxicology*, *2*(1), 1-31.

Ingestion of caterpillars taken from a cotton field sprayed with parathion (an organophosphate) killed 16-18 Mississippi kites (*Ictinia mississippiensis*).[80] Phosphamidon, an organophosphate, sprayed in forests in Switzerland caused significant mortality across a range of species[81] and widespread bird mortality was also observed in Canadian spruce forests sprayed with phosphamidon, particularly among insectivorous warblers, as a result of birds picking up the insecticide from the sprayed foliage within a few hours of application.[82] Carbofuran and phosphamidon were the most common pesticides implicated in deaths of wild birds in Korea between 1998-2002,[83] and ducklings died in large numbers when phorate, an organophosphate, was applied to South Dakota wetlands.[84]

In orchards sprayed with methomyl (carbamate), oxamyl (carbamate, granular form banned in the United States) or dimethoate (organophosphate), the daily survival rates for nests of Pennsylvania mourning dove (*Zenaida macroura*) and American robin (*Turdus migratorius*) were significantly lower than in non-treated orchards. Repeated applications of these and other insecticides significantly reduced the reproductive success of doves and robins.[85]

Aerial sprays of pesticides for locust control, as discussed above, may affect species other than those who inhabitat agricultural areas because of the wide-range of application.

2.4.4. Migratory bird ecology: diet, foraging behaviour, habitat preferences, and migration behaviour may influence likelihood of exposure

The broad-spectrum nature of organophosphates and carbamates means that any bird in the vicinity of where pesticides are applied is at risk of exposure – typically the likelihood of exposure is increased for birds that use agricultural areas for foraging.

Bird migration behaviour, such as late-staying spring migrants are at greater risk of exposure, e.g., to snow geese (*Chen caerulescens*) in dieldrin treated rice fields in Texas.[86] Dieldrin (no longer used in the US) was shown to cause mortality of geese in rice habitats in Texas and a large die-off of nesting white-faced ibis (*Plegadis chihi*).[87]

One study that simulated foraging in insecticide-treated fields in Texas, USA, found that foraging location is more likely to influence exposure than diet preferences or daily intake rate.[88] Exposure is therefore, the result of a temporal and spatial overlap between species occurrence and principle areas and timings of insecticide application. For example, birds that forage more frequently in grassland areas are less likely to be exposed to insecticides because no insecticide applications occur

[80] Franson, J. C. (1994). Parathion poisoning of Mississippi kites in Oklahoma. *J Raptor Res*, *28*, 108-109.

[81] Schneider, F. (1966). Some pesticide-wildlife problems in Switzerland. *Journal of Applied Ecology*, 15-20.

[82] Fowle, C. D. (1966). The Effects of Phosphamidon on Birds in New Brunswick Forests. *Journal of Applied Ecology*, 169-170.

[83] Kwon, Y. K., Wee, S. H., & Kim, J. H. (2004). Pesticide poisoning events in wild birds in Korea from 1998 to 2002. *Journal of wildlife diseases*, *40*(4), 737-740.

[84] Dieter, C. D., Flake, L. D., & Duffy, W. G. (1995). Effects of phorate on ducklings in northern prairie wetlands. *The Journal of wildlife management*, 498-505.

[85] Fluetsch, K. M., & Sparling, D. W. (2009). Avian nesting success and diversity in conventionally and organically managed apple orchards. *Environmental toxicology and chemistry*, *13*(10), 1651-1659.

[86] Flickinger, E. L. (1979). Effects of aldrin exposure on snow geese in Texas rice fields. *The Journal of Wildlife Management*, 94-101.

[87] Flickinger, E. L., & Meeker, D. L. (1972). Pesticide mortality of young White-faced Ibis in Texas. *Bulletin of Environmental Contamination and Toxicology*, *8*(3), 165-168.

[88] Corson, M. S., Mora, M. A., & Grant, W. E. (1998). Simulating cholinesterase inhibition in birds caused by dietary insecticide exposure. *Ecological modelling*, *105*(2), 299-323.

there. In some areas, exposure risk decreases as crops grow because certain species generally spend less time in crop fields.[89] Therefore, pesticides applied at the time of planting may pose the highest risk of exposure to birds.

Organophosphates and carbamates may have a larger effect on small birds. In a northbound migration, small-bodied migratory shorebirds had more significant effects of exposure.[90] Another study on migratory shorebirds found birds were exposed to organophosphates and carbamates at wintering areas in South America, but not at migratory stopover sites.[91]

Raptors appear to be more sensitive than other bird species to organophosphates and carbamates.[92] The risk of poisoning to raptors is increased as a result of their ecology, such as insectivory, opportunistic taking of debilitated prey, scavenging, presence in agricultural areas and bioaccumulation of some types of insecticides.

The use of crop areas can vary widely between birds, over time, and in response to local conditions, such as the relative availability of food from other sources. Some species may be at risk of exposure to pesticides when their usual food sources are depleted. For example, pink-footed geese (*Anser brachyrhynchus*) and greylag geese (*Anser anser*) wintering in Scotland prefer to feed on harvest spillage, root crops and grass, and may only turn to newly sown cereals when the alternatives are depleted. The organophosphate pesticide carbophenothion was used as an insecticide seed treatment on cereals, which resulted in the poisoning of these geese. In this case, the effects only occurred on a small proportion of the occasions when the chemical was used, but were considered sufficiently severe to require regulatory action in Scotland.[93]

3. Toxicity: the level of toxicity influences effects

If exposure to insecticides is likely to occur, the toxicity level of the insecticide will greatly influence the corresponding effect. Some substances are acutely toxic to birds generally; other substances are more likely to affect particular bird species because of their unique physiology.

Overall, there has been a net reduction in the average avian acute toxicity of insecticides over time and most current problems stem from a failure to remove “old dangerous products” from the market.[94] For example, agricultural pesticide use statistics from the United States suggest the lethal risk to birds in our farmland has been generally decreasing over the last decade, with a few exceptions. The risk appears highest in the south-eastern United States, where the area is heavily used by birds as a breeding, stopover and wintering area.[95] However, this trend is unlikely in countries with less developed pesticide regulatory systems.

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89 Ibid.

90 Strum, K. M., Alfaro, M., Haase, B., Hooper, M. J., Johnson, K. A., Lanctot, R. B., ... & Zaccagnini10, M. E. (2008). Plasma cholinesterases for monitoring pesticide exposure in Nearctic-Neotropical migratory shorebirds. *Ornitología Neotropical*, *19*, 641-651.

91 Strum, K. M., Hooper, M. J., Johnson, K. A., Lanctot, R. B., Zaccagnini, M. E., & Sandercock, B. K. (2010). Exposure of Nonbreeding Migratory Shorebirds to Cholinesterase Inhibiting Contaminants in the Western Hemisphere. *The Condor*, *112*(1), 15-28.

92 Mineau et al., *Poisoning of raptors with organophosphorus and carbamate pesticides with emphasis on Canada, US and UK*.

93 Hart, A. D. M. (2008). The assessment of pesticide hazards to birds: the problem of variable effects. *Ibis*, *132*(2), 192-204.

94 Mineau, P., & Whiteside, M. (2006). Lethal risk to birds from insecticide use in the United States—a spatial and temporal analysis. *Environmental toxicology and chemistry*, *25*(5), 1214-1222.

95 Ibid.

Sub-lethal effects on birds exposed to insecticides are more common than lethal effects, although they are generally more difficult to document and quantify. Many studies have shown that sub-lethal doses of organophosphates can cause behavioural effects in birds (see Figure 2). Effects are variable and can include reductions in food consumption that leads to weight loss, lack of aggressive behaviour, memory impairment that can compromise survival, immobility on the ground that increases predation risk, apathy in incubation, nest defence and care for nestlings leading to fewer nestlings and hence reduced productivity,[96] and fertility.[97] Sub-lethal toxicity associated with exposure to organophosphates and carbamates can also lead to alteration in migratory behaviour, such as a lack of migratory orientation.[98]

Many of these effects are transient, but those affecting, for example, reproduction, can impact on the long-term viability of a species, even if there might not be apparent short-term population declines.[99] However, behavioural effects are difficult to quantify and there is limited evidence linking them to population declines.[100]

Figure 2: Range of sub-lethal effects that could lead to population declines

Reproduction
One study found population-level effects as a result of sub-lethal exposure, which caused behavioural changes such as inability to defend territories, disruption of incubation patterns and clutch desertion (the reproductive success was 25 per cent less as a result of two consecutive sprays), from spraying a forest with fenithrothion (an organophosphate).[101]

Avoidance of predation
One study found significantly lower survival rates in birds exposed to organophosphates because of greater susceptibility to predation.[102]

Feeding
Direct effects may occur on one or several components of feeding behaviour, including encountering, choosing, capturing and handling of prey.[103] Effects such as loss of coordination, disorientation and convulsions could impair hunting skills.[104]

Low-level acute exposure to organophosphates can produce long-term changes to feeding patterns. In a field study, red-winged blackbirds (*Agelaius phoeniceus*) exposed to prey contaminated with parathion developed taste aversion to the prey species in question even where uncontaminated by the chemical.[105] This could result in reduced food intake and hence body condition, but it could also minimise the risk of poisoning.

[96] Grue, C. E., George v. N. Powell, & McChesney, M. J. (1982). Care of nestlings by wild female starlings exposed to an organophosphate pesticide. *Journal of Applied Ecology*, 327-335.

[97] Sánchez-Bayo, *Impacts of agricultural pesticides on terrestrial ecosystems*.

[98] Vyas, N. B., Hill, E. F., Sauer, J. R., & Kuenzel, W. J. (1995). Acephate affects migratory orientation of the white-throated sparrow (Zonotrichia albicollis). *Environmental toxicology and chemistry*, *14*(11), 1961-1965.

[99] Fluetsch, K. M., & Sparling, D. W. (2009). Avian nesting success and diversity in conventionally and organically managed apple orchards. *Environmental toxicology and chemistry*, *13*(10), 1651-1659.

[100] Walker, Neurotoxic pesticides and behavioural effects upon birds; Peakall, D. B. (1985). Behavioral responses of birds to pesticides and other contaminants. *Residue reviews*, *96*, 45-77.

[101] Busby, D. G., White, L. M., & Pearce, P. A. (1990). Effects of aerial spraying of fenitrothion on breeding white-throated sparrows. *Journal of Applied Ecology*, 743-755.

[102] Brewer, L. W., Driver, C. J., Kendall, R. J., Zenier, C., & Lacher, T. E. (1988). Effects of methyl parathion in ducks and duck broods. *Environmental toxicology and chemistry*, *7*(5), 375-379.

[103] Atchison, G. J., Sandheinrich, M. B., & Bryan, M. D. (1996). Effects of environmental stressors on interspecific interactions of aquatic animals. *Ecotoxicology: A hierarchical treatment. Lewis Publishers, Boca Raton, Florida*, 319-345.

[104] Sharma, R. P. (1976). Influence of dieldrin on serotonin turnover and 5-hydroxyindole acetic acid efflux in mouse brain. *Life sciences*, *19*(4), 537-541.

[105] Nicolaus, L. K., & Lee, H. (1999). Low acute exposure to organophosphate produces long-term changes in bird feeding behavior. *Ecological Applications*, *9*(3), 1039-1049.

The likelihood of sub-lethal or lethal effects occurring is strongly influenced by the toxicity of the insecticide, which varies between first generation and second generation insecticides, with the latter often showing significantly greater toxicity. There is also significant variation between the compounds in each of these groups, which is discussed below.

3.1. First generation insecticides: regular agricultural use causes both sub-lethal and lethal effects leading to population declines

Organochlorines, such as DDT, aldrin and dieldrin, were one of the earliest generations of synthetic pesticides. A number of elements make organochlorines highly risky to birds and ecosystems. DDT and other organochlorines are characterised by their environmental persistence (remain in the environment for a long period of time), toxicity to organisms and their ability to accumulate in the tissues of birds and other wildlife resulting in an increased concentration with each step up the food chain.[106]

Organochlorines are widely documented as causing population-level effects in birds as a result of their use in agriculture. [107] One of the most widespread organochlorines used in agriculture is DDT, which causes harmful effects to birds, such as reproductive decline from eggshell thinning and direct mortality in adults.[108] While direct exposure to DDT is not highly toxic to birds, heavy and repetitive use of the pesticide is, as it causes increased exposure due to bioaccumulation of the compound.[109] Therefore, the effects are the greatest on top predators in the food chain due to bioaccumulation of the pesticides within the tissues of the bird. Raptors are particularly vulnerable because of their position in the food chain and diet preferences, which makes them vulnerable to bioaccumulation of the substance.

The most persistent organochlorines were subject to a voluntary withdrawal in the 1960s and banned in Europe and North America for agricultural purposes in the 1970s as a result of harmful effects to humans and wildlife.[110] Many continue to be used in Asia, Africa and South America and they are still a concern in some areas as a result of heavy contamination from prior use.[111]

After the ban in Europe and North America, populations of birds of prey, such as peregrine falcons (*Falco peregrinus*) and sparrowhawks (*Accipiter spp*.), have started to recover despite some persistent and ongoing effects of DDT in those environments. Despite the ban on persistent organochlorines in most of the developed nations since the early 1970s, their usage continued until very recently in many developing countries for agricultural and public health purposes.[112] The use of organochlorine insecticides, such as DDT, has been recorded in many developing countries including Mexico, India, Thailand and Vietnam – where it was banned for agricultural use in 1993, but is still

106 Newton, I. (1984). Uses and effects on bird populations of organochlorine pesticides.
107 Newton, I. (1998). Pollutants and pesticides. *Conservation Science and Action*, 66-89.
108 Kiff, L. F., Peakall, D. B., & Wilbur, S. R. (1979). Recent changes in California Condor eggshells. *Condor*, 166-172.
109 Fry, D. M. (1995). Reproductive effects in birds exposed to pesticides and industrial chemicals. *Environmental Health Perspectives*, *103* (Suppl 7), 165.
110 Mineau, *Birds and pesticides: is the threat of a silent spring really behind us?*
111 Ecobichon, D. J. (2001). Pesticide use in developing countries. *Toxicology*, *160*(1), 27-33.
112 Minh, N. H., Someya, M., Minh, T. B., Kunisue, T., Iwata, H., Watanabe, M. & Tuyen, B. C. (2004). Persistent organochlorine residues in human breast milk from Hanoi and Hochiminh city, Vietnam: contamination, accumulation kinetics and risk assessment for infants. *Environmental Pollution*, *129*(3), 431-441.

used for malaria control.[113] However, use has been substantially reduced in agriculture and levels of DDT in soils have been declining in places such as China.[114]

Many important wintering areas in Mexico and Latin America are located near major agricultural areas, where pesticide applications are often high. The overall use of DDT in Mexico has been substantially reduced, but it remains legal for agricultural purposes in Mexico and throughout Latin America. However, studies of migrants have generally found no difference in DDT levels between birds wintering in the southwest United States and Mexico.[115] The cotton producing areas of Mexico, such as Chiapas which may legally still use DDT in agriculture, need further study, particularly as DDT was applied there heavily in the past.[116]

In Vietnam, recent input of organochlorines has been identified in both northern and southern parts of the country, potentially as a result of DDT for malaria control, with relatively high levels of organochlorines found in birds.[117] Migratory birds from the Philippines and Vietnam, including plovers, terns, and sandpipers, had higher levels of DDT than migratory birds in India and Lake Baikal, Russia.[118]

In a study of 16 species in India, no serious threats associated with organochlorines were found.[119] In New Zealand, the residual organochlorine concentrations found in sooty shearwater (*Puffinus griseus*), a migratory seabird, were all below the levels associated with physiological impairment/mortality.[120] In Africa, since the late-1980s, organochlorines were largely replaced by organophosphates, carbamates and pyrethroids (not highly toxic to birds) and are generally no longer used in agriculture, including for locust control in Africa.[121] A similar situation exists in Australia.[122]

[113] Minh, T. B., Kunisue, T., Yen, et al. (2002). Persistent organochlorine residues and their bioaccumulation profiles in resident and migratory birds from North Vietnam. *Environmental toxicology and chemistry*, *21*(10), 2108-2118; Pandit, G. G., Sharma, et al. (2002). Persistent organochlorine pesticide residues in milk and dairy products in India. *Food Additives & Contaminants*, *19*(2), 153-157; Stuetz, W., Prapamontol, T., et al. (2001). Organochlorine pesticide residues in human milk of a Hmong hill tribe living in Northern Thailand. *Science of the total environment*, *273*(1), 53-60.; Nhan, D. D., Carvalho, F. P., Tuan, et al. (2001). Chlorinated pesticides and PCBs in sediments and molluscs from freshwater canals in the Hanoi region. *Environmental Pollution*, *112*(3), 311-320.

[114] Kunisue, T., Watanabe, M., Subramanian, A., Sethuraman, A., Titenko, A. M., Qui, V., ... & Tanabe, S. (2003). Accumulation features of persistent organochlorines in resident and migratory birds from Asia. *Environmental pollution*, *125*(2), 157-172.

[115] Mora, M. A. (2009). Transboundary pollution: persistent organochlorine pesticides in migrant birds of the Southwestern United States and Mexico. *Environmental Toxicology and Chemistry*, *16*(1), 3-11.

[116] Ibid.

[117] Minh et al., *Persistent organochlorine residues and their bioaccumulation profiles in resident and migratory birds from North Vietnam*; Nhan et al., *Chlorinated pesticides and PCBs in sediments and molluscs from freshwater canals in the Hanoi region*.

[118] Kunisue, T., Watanabe, M., Subramanian, A., Sethuraman, A., Titenko, A. M., Qui, V. & Tanabe, S. (2003). Accumulation features of persistent organochlorines in resident and migratory birds from Asia. *Environmental pollution*, *125*(2), 157-172.

[119] Dhananjayan, V. (2012). Accumulation pattern of persistent organochlorine pesticides in liver tissues of various species of birds from India. *Environmental Science and Pollution Research*, 1-8.

[120] El-Din Bekhit, A., Al-Amer, S., Gooneratne, R., Mason, S. L., Osman, K. A., & Clucas, L. (2011). Concentrations of trace elementals and organochlorines in Mutton bird (Puffinus griseus). *Ecotoxicology and environmental safety*, *74*(6), 1742-1746.

[121] Peveling, R., Attignon, S., Langewald, J., & Ouambama, Z. (1999). An assessment of the impact of biological and chemical grasshopper control agents on ground-dwelling arthropods in Niger, based on presence/absence sampling. *Crop protection (Guildford, Surrey)*, *18*(5), 323-339.

[122] Szabo, J. K., Davy, P. J., Hooper, M. J., & Astheimer, L. B. (2009). Predicting avian distributions to evaluate spatiotemporal overlap with locust control operations in eastern Australia. *Ecological Applications*, *19*(8), 2026-2037.

3.2. Second generation insecticides: the effects depend on toxicity level of individual insecticide, sub-lethal effects more common than lethal effects, sub-lethal effects may affect population levels but harder to detect in field

After bans of the persistent pesticides (organochlorines) were introduced in some areas, less persistent pesticides – organophosphates and carbamates, also known as cholinesterase-inhibiting insecticides – were introduced. They only stay active in the environment for days or weeks at a time and do not bioaccummulate in the food chain.[123] Although these insecticides have limited persistence, decreasing risk of exposure, in the environment, they have an elevated toxicity – particularly to birds.[124] Both sets of compounds are acutely toxic (neurotoxic) to birds, often at very low doses.[125]

Organophosphates and carbamates are the most commonly used pesticides throughout the world (in both developed and developing countries) since the 1980s – they make up 54 per cent and 22 per cent, respectively, of all insecticides applied in the United States.[126] In general, the use of oldest and most toxic of these insecticides is decreasing, but they still retain a 50 per cent market share worldwide.[127]

Both of these pesticides affect a wide variety of organisms, not just insects (including mammals and birds).[128] Although organophosphates and carbamates have the same mode of action, different organisms can be resistant to one class of compounds but not the other. For example, some pests are resistant to organophosphates (eg, white flies, leaf miners, ants, scale insects, cockroaches, wasps and aphids), but can be controlled by carbamates.[129]

The timing of pesticide application is closely associated with poisoning because organophosphate and carbamate pesticides are short-lived compounds in the environment. These short-lived chemicals can also make detecting the compounds in dead birds difficult because of the rapid breakdown process (metabolism in the body).[130] Degradable compounds may be prolonged due to interaction with other pesticides, and there is evidence that effects may persist longer than the active form of the insecticide.[131]

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123 Mitra, A., Chatterjee, C., & Mandal, F. B. (2011). Synthetic chemical pesticides and their effects on birds. *Res. J. Environ. Toxicol*, *5*(2), 81-96.

124 Pisani, J. M., Grant, W. E., & Mora, M. A. (2008). Simulating the impact of cholinesterase-inhibiting pesticides on non-target wildlife in irrigated crops. *Ecological Modelling*, *210*(1), 179-192.

125 Cox, C. (1991). Pesticides and birds: from DDT to today's poisons. *Wildl. Dis*, *24*(1), 51-61.

126 Gianessi, L. P., & Silvers, C. S. (2000). *Trends in crop pesticide use: Comparing 1992 and 1997* (p. 165). Washington, DC: National Center for Food and Agricultural Policy.

127 Devine, G. J., & Furlong, M. J. (2007). Insecticide use: contexts and ecological consequences. *Agriculture and Human Values*, *24*(3), 281-306.

128 Donovan, S., Taggart, M., & Richards, N. (2012). 1 An overview of the chemistry, manufacture, environmental fate and detection of carbofuran. *Carbofuran and Wildlife Poisoning*, 1-18.

129 Ibid.

130 Kwon, Y. K., Wee, S. H., & Kim, J. H. (2004). Pesticide poisoning events in wild birds in Korea from 1998 to 2002. *Journal of wildlife diseases*, *40*(4), 737-740.

131 Walker, C.H. (2001). Organic Pollutants: An Ecotoxicological Perspective. London: Taylor and Francis.

Second generation pesticides often present less obvious effects, such as transitory behavioural disturbances, which could be of ecological significance. [132] Some of the second generation insecticides have been shown to be very toxic to birds and are linked with population declines. For example, granular carbofuran applied at seeding in canola fields resulted in reduced abundance and declining population trends of common agricultural species such as the horned lark, house sparrow, western meadowlark (*Sturnella neglecta*), American robin, and mourning dove in the Canadian prairies.[133]

Organophosphates have been implicated in 335 separate mortality events causing the deaths of approximately 9,000 birds between 1980 and 2000 in the United States.[134] Secondary poisoning by carbamate and organophosphate insecticides have been attributed as the cause of mortality in barn owls (*Tyto alba*), American kestrels (*Falco sparverius*), red-tailed hawks (*Buteo jamaicensis*), great horned owls (*Bubo virginianus*) and bald eagles (*Haliaeetus leucocephalus*).[135]

Many of these highly toxic substances have been removed from the agricultural pesticide market or are regulated to some extent in most developed countries. For example in most developed countries, carbofuran and monocrotophos are no longer used as agricultural pesticides (their illegal use is discussed in another section). Much of the evidence of poisoning of birds in agricultural areas is related to these compounds. In less developed regions, these compounds are often still used in agriculture.

Chlorfenapyr is very toxic to birds, affecting reproduction, and has been denied registration in the USA, but is now registered in a number of other countries.[136] In Bolivia, monocrotophos is still the dominant insecticide in rice cultivation. The last major desert locust outbreak in the Sahel region (2003-2005) was treated with organophosphate insecticides – notably chlorpyrifos and malathion and to a lesser extent fenitrothion – putting birds at risk.

Carbofuran has been used worldwide for control of pests in sugarcane, sugar beet, maize, rice and coffee and is very effective in controlling rice pests. It is very toxic to birds with more than 80 bird species in farmland the US and Canada known to have been killed by this insecticide.[137] The use of this insecticide has now been restricted or banned for agricultural purposes in Europe, Canada and USA. It is still used widely throughout Mexico,[138] South America, Africa and Asia. Carbofuran constitutes a high proportion of insecticides in some markets, such as in Korea.[139] In some regions,

[132] Rattner, B. A., & McGowan, P. C. (2007). Potential hazards of environmental contaminants to avifauna residing in the Chesapeake Bay estuary. *Waterbirds*, *30*(sp1), 63-81; Lee, C. (1972). Death in the potato fields. *Florida Nat*, *45*, 60-61; Fleischli et al., *Avian mortality events in the United States caused by anticholinesterase pesticides: a retrospective summary of National Wildlife Health Center records from 1980 to 2000*; Elliott et al., *Secondary poisoning of birds of prey by the organophosphorus insecticide, phorate*.

[133] Mineau, P., & Whiteside, M. (2006). Lethal risk to birds from insecticide use in the United States—a spatial and temporal analysis. *Environmental toxicology and chemistry*, *25*(5), 1214-1222.

[134] Fleischli et al., *Avian mortality events in the United States caused by anticholinesterase pesticides: a retrospective summary of National Wildlife Health Center records from 1980 to 2000*.

[135] Mineau et al., *Poisoning of raptors with organophosphorus and carbamate pesticides with emphasis on Canada, US and UK*.

[136] Mineau, *Birds and pesticides: is the threat of a silent spring really behind us?*

[137] Ibid.

[138] Osten et al., *Black-bellied whistling duck (Dendrocygna autumnalis) brain cholinesterase characterization and diagnosis of anticholinesterase pesticide exposure in wild populations from Mexico*.

[139] Park, H. H. (2009). Impact of pesticide treatment on an arthropod community in the Korean rice ecosystem. *Journal of Ecology and field biology*, *32*(1), 19-25.

its use is expanding; for example, in Kenya carbofuran use has recently expanded from rice to maize and wheat and is sold over the counter in spray, granules or seed dressing form.[140]

The detection of sub-lethal effects may suffer from a sampling bias, which arises because birds which are most severely affected by the pesticide, may be less active, more secretive and less likely to be collected than other birds which are less affected.[141] Sampling bias may cause the true range of variation in effects to be under-estimated by failure to recover the worst-affected individuals.

Figure 3: Knowledge gaps in the literature on effects of insecticides on migratory birds

- Is the likelihood of exposure related to the volume of insecticide use or how insecticides are used?
  - Do certain methods of agriculture increase the effects on birds, eg, require more/less pesticides, frequency of application?
  - Are certain types of insecticides more likely to result in exposure of migratory birds, eg, require more frequent treatments?
- Does exposure to multiple pesticides have a cumulative effect on bird mortality? Does pesticide exposure interact with other stressors, eg, disease, to increase mortality?
- Potential significance of sub-lethal poisoning?
- Are insecticides more likely to result in exposure of migratory birds in certain climates or landscapes, eg, climates with higher numbers of insects may require more intensive insecticide use? Variation between seasons?
- How frequently does poisoning occur through negligent/mis-use of pesticides (versus labelled use)?
- Is illegal or legal use of pesticides causing more bird deaths?
- Much of the literature is focussed on North America and Europe, how do the effects vary in other parts of the world?
- Raptors appear to be the most studied, how do the effects from exposure to insecticides vary in other species?
- Effects of new insecticides on migratory birds, eg, likelihood of exposure and toxicity?
- Where likelihood of exposure is high and toxicity of the insecticide is also high, how likely are population-level effects?
- How do direct effects (primary or secondary poisoning) compare with indirect effects of insecticide use (eg, changes in food source)?
- How do the effects of insecticides on birds compare with other causes of population decline, such as habitat loss?
- What are the effects of new types of pesticides on birds?

4. Conclusion

The use of agricultural pesticides has historically caused the decline of several bird species, particularly birds of prey, with the use of organochlorine insecticides, such as DDT. Organochlorine insecticides are no longer regularly used in agriculture in many areas and while there are residual effects on populations (and some concern with ongoing DDT use in malaria prevention) because of the persistent nature of the compound, there is no longer the immediate concern of dramatic widespread population declines associated with the use of agricultural pesticides.

However, the significant decline of many farmland bird species raises the conservation significance of any potential source of additional mortality or reduced breeding success. Pesticides used in

[140] Otieno, P. O., Lalah, J. O., Virani, M., Jondiko, I. O., & Schramm, K. W. (2010). Soil and water contamination with carbofuran residues in agricultural farmlands in Kenya following the application of the technical formulation Furadan. *Journal of Environmental Science and Health Part B*, *45*(2), 137-144.

[141] Mineau, P., & Peakall, D. B. (2009). An evaluation of avian impact assessment techniques following broad-scale forest insecticide sprays. *Environmental toxicology and chemistry*, *6*(10), 781-791.

routine agricultural practices (primarily, second generation insecticides – organophosphates and carbamates) and their direct effects on bird species are varied and depend on the likelihood of exposure and the toxicity level of the pesticide. Both the likelihood of exposure and effects are difficult to determine because of the difficulty of studying effects in the field. However, the broad spectrum nature of organophosphates and carbamates makes any bird at risk of exposure if they happen to be in the vicinity at the time of application, or shortly thereafter, or if they come into contact with a prey species that was in contact with pesticides.

Likelihood of exposure is linked to agricultural cultivation practices, the form of the pesticide and the particular ecology and behaviour of migratory birds. The likelihood of exposure can be reduced by using particular forms of pesticides, eg, liquid forms over granular forms, and changing application periods for when migratory birds are not likely to be present (which can be effective given the low persistence of many of the second generation pesticides).

If a bird is likely to be exposed, which is often a matter of being in the wrong place at the wrong time, the toxicity level of the pesticide is significant as to whether exposure will result in any effect – either lethal or sub-lethal. Many of the highly toxic insecticides, such as carbofuran and monocrotophos, have been removed from the market in developed countries because of the significant lethal risk to birds. Single events can cause population-level effects, such as the Swainson's hawks incident in Argentina.

Sub-lethal effects are more common as a result of exposure to second generation pesticides. The implications of the sub-lethal effects are little understood and are difficult to study in the field. Migratory birds may be particularly susceptible to sub-lethal effects because of the reaction to second generation insecticides, which causes reduced movement and affects migratory behaviour. Any reduction in energy levels can have significant effects on migratory birds because of their high energy requirements for migration. Further research should focus on assessing these effects on populations and model simulations may play a valuable role in this area.

Much of the effects, both sub-lethal and lethal, recorded in the literature are related to the use of these now highly regulated compounds. This could indicate that the situation has improved in areas where these highly toxic compounds are no longer used or that newer substances have not yet been studied. Therefore, efforts to remove these highly toxic compounds from markets where they are still used would likely make the most significant contribution to reducing this route of poisoning of migratory birds. This is particularly true where likelihood of exposure is high and the use of highly toxic insecticides occurs.

Neonicotinoids have become a main replacement for the carbamates and organophosphates in many countries. Further research is needed to understand their direct impacts on birds.

Effects of crop protection/harvests using rodenticides

1. Introduction

This is a review on the effects on migratory birds from the approved (legal) use of rodenticides (illegal use is covered in a separate section herein) to control rodent populations. Mice, rats, and other rodents can threaten food production and act as reservoirs for disease throughout the world. Rodenticides are commonly used to prevent rodent infestations that cause spoiling of crops and grain storage, but are also used in urban areas for human health, and the protection of buildings from damage.[142] The effects on wildlife from urban use are largely unknown and therefore, this review will focus on the effects associated with use in more rural, often agricultural, areas, which are more likely to affect migratory bird species directly.

Rodents are one of the most significant agricultural pests globally. In many developing countries, farmers consider rodents the main impediment to higher yields.[143] Each year, rats in Asia destroy food crops that could feed 200 million people for an entire year.[144] In Africa, damage to corn crops in Tanzania by rodents costs $45 million.[145] Rodent damage in South America can vary between 5-90 per cent of total crop production.[146]

Anticoagulant rodenticides are the most widely used rodenticides to control rodent pests worldwide.[147] In many countries, they are also an integral component of modern agriculture for the control of rodent populations.[148] In some areas they may be over-used (tending towards countries with high GDP) and in other areas may not be used much (tending towards countries with low GDP).[149] All anticoagulant rodenticides may pose a risk to predatory and scavenging birds on a worldwide scale, with the active substance a key determinant of risk. [150]

[142] Tosh, D. G., Shore, R. F., Jess, S., Withers, A., Bearhop, S., Ian Montgomery, W., & McDonald, R. A. (2011). User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use. Journal of environmental management, 92(6), 1503-1508.

[143] Makundi, R. H., Oguge, N. O., & Mwanjabe, P. S. (1999). 22. Rodent Pest Management in East Africa—an Ecological Approach.

[144] Singleton, G. R. (2003). Impacts of rodents on rice production in Asia. *IRRI Discuss. Pap. Ser*, *45*, 30.

[145] Stenseth, N. C., Leirs, H., Skonhoft, A., Davis, S. A., Pech, R. P., Andreassen, H. P., ... & Wan, X. (2003). Mice, rats, and people: the bio-economics of agricultural rodent pests. *Frontiers in Ecology and the Environment*, *1*(7), 367-375.

[146] Ibid.

[147] Sánchez-Barbudo, I. S., Camarero, P. R., & Mateo, R. (2012). Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain. Science of the Total Environment, 420, 280-288.

[148] Tosh et al., User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use.

[149] Ghimire, N., & Woodward, R. T. (2013). Under-and over-use of pesticides: An international analysis. Ecological Economics, 89, 73-81.

[150] Rattner, B. A., Horak, K. E., Warner, S. E., Day, D. D., Meteyer, C. U., Volker, S. F., ... & Johnston, J. J. (2011). Acute toxicity, histopathology, and coagulopathy in American kestrels (*Falco sparverius*) following administration of the rodenticide diphacinone. Environmental Toxicology and Chemistry, 30(5), 1213-1222.

Although anticoagulant rodenticides were designed to control commensal rodents, they have a non-specific mode of action and can affect a wide variety of wildlife, including birds. [151] These substances inhibit the vitamin K cycle in the liver, which is typically associated with lethal haemorrhaging.[152]

This report assesses the risk of rodenticides (normal use) on migratory bird populations. Population-level effects are determined by a function of (1) the likelihood that migratory birds will be exposed to rodenticides; (2) the toxicity of the rodenticide to migratory birds. Each of these components are discussed in more detail below.

2. Likelihood of exposure

Migratory birds can be exposed to anticoagulant rodenticides (ARs) through the consumption of contaminated baits (primary) or by the consumption of contaminated prey (secondary). Widespread exposure in birds to rodenticides has been detected through wildlife monitoring programmes in Europe and North America. Most of the studies have focused on birds of prey, as top predators can act as sentinel species for the integrity of the food chain. For example, high detection rates of anticoagulant rodenticides have been reported in birds of prey collected through wildlife monitoring programmes in Canada (70 per cent of 164 owls of various species) and 60 per cent red-tailed hawks exposed[153]), USA (86 per cent of 161 birds tested had liver residues[154]), United Kingdom (90 per cent of 96 birds -- barn owls, red kites and kestrels -- exposed[155]), Norway (53 per cent of golden eagles and eagle owls exposed[156]), Denmark (92 per cent of 430 birds exposed from 11 species[157]), France (44 per cent of red kites indicated AR poisoning[158]), and Spain (9 per cent indicated AR poisoning[159]). A number of examples of exposure are listed in Table 1. The rates of exposure in other parts of the world are largely unknown. However, birds submitted to monitoring programmes (typically dead birds found by members of the public) may underestimate the extent of exposure as fatally exposed birds may die in tree roosts or other locations where they are not readily found. The effects of exposure on species at both the individual and population level remain poorly understood.[160]

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151 Lambert, O., Pouliquen, H., Larhantec, M., Thorin, C., & L'Hostis, M. (2007). Exposure of raptors and waterbirds to anticoagulant rodenticides (difenacoum, bromadiolone, coumatetralyl, coumafen, brodifacoum): epidemiological survey in Loire Atlantique (France). Bulletin of environmental contamination and toxicology, 79(1), 91-94.

152 Lethal haemorrhaging may occur as a result of the inhibition of vitamin K in the liver, which interferes with the clotting cascade and increases coagulation times.

153 Albert, C. A., Wilson, L. K., Mineau, P., Trudeau, S., & Elliott, J. E. (2010). Anticoagulant rodenticides in three owl species from western Canada, 1988–2003. *Archives of environmental contamination and toxicology*, *58*(2), 451-459; Thomas, P. J., Mineau, P., Shore, R. F., Champoux, L., Martin, P. A., Wilson, L. K., ... & Elliott, J. E. (2011). Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada. *Environment international*, *37*(5), 914-920.

154 Murray, M. (2011). Anticoagulant rodenticide exposure and toxicosis in four species of birds of prey presented to a wildlife clinic in Massachusetts, 2006-2010. *Journal of Zoo and Wildlife Medicine*, *42*(1), 88-97.

155 Walker, L. A., Chaplow, J. S., Llewellyn, N. R., Pereira, M. G., Potter, E. D., Sainsbury, A. W., & Shore, R. F. (2013). Anticoagulant rodenticides in predatory birds 2011: a Predatory Bird Monitoring Scheme (PBMS) report.

156 Langford, K. H., Reid, M., & Thomas, K. V. (2013). The occurrence of second generation anticoagulant rodenticides in non-target raptor species in Norway. *Science of the Total Environment*, *450*, 205-208.

157 Christensen, T. K., Lassen, P., & Elmeros, M. (2012). High Exposure Rates of Anticoagulant Rodenticides in Predatory Bird Species in Intensively Managed Landscapes in Denmark. *Archives of environmental contamination and toxicology*, *63*(3), 437-444.

158 Berny, P., & Gaillet, J. R. (2008). Acute poisoning of red kites (*Milvus milvus*) in France: data from the SAGIR network. *Journal of wildlife diseases*, *44*(2), 417-426.

159 Sánchez-Barbudo et al., *Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain*.

160 Burn, A. J., Carter, I., & Shore, R. F. (2002). The threats to birds of prey in the UK from second-generation rodenticides. *Aspects of Applied Biology*, *67*, 203-212; Knopper, L. D., Mineau, P., Walker, L. A., & Shore, R. F. (2007). Bone density

Table 1: Examples of anticoagulant exposure in birds of prey in Europe, Canada and USA

United Kingdom

In the UK, secondary exposure to ARs has been found in populations of barn owl (*Tyto alba*), tawny owl (*Strix aluco*), kestrel (*Falco tinnunculus*), buzzard (*Buteo buteo*), and red kite (*Milvus milvus*). Recent research carried out by the Predatory Bird Monitoring Scheme in the United Kingdom indicates over 90 per cent of barn owls and red kites were exposed to second generation anticoagulant rodenticides in 2010.[161]

Norway

In a study in Norway, ARs were present in five species of raptors found dead in Norway between 2009 and 2011.162 The ARs brodifacoum, bromadiolone, difenacoum and flocoumafen were detected in approximately 70 per cent of the golden eagles (*Aquila chrysaetos*) and 50 per cent of the eagle owls (*Bubo bubo*). Thirty percent (7/24) of the golden eagle and eagle owl livers contained (estimated) lethal levels. However, the overall impact on the raptor populations was not possible to determine. [163]

Canada

A sample of raptors in Quebec, Canada, found that 43 per cent of birds were exposed to ARs (13/30 birds). This study suggests a broad contamination of the food chain of hawks, particularly *Accipiters*, such as Cooper's hawk, merlins and the American kestrel. Many of these species feed predominantly on small birds (rather than rodents) and occasionally on insects.[164] Therefore, it may indicate that small birds are exposed to SGARs through insects or ingestion of grain-based baits. [165]

United States

Of 161 birds submitted to a wildlife clinic in Massachusetts, USA, between 2006-2010, 86 per cent had AR residues in their liver. Within the 86 per cent of exposed birds, nearly all of the birds (99 per cent) had brodifacoum residues. Mortality from AR toxicosis was diagnosed in only 6 per cent of the birds. No indication of sub-lethal effects of exposure were found.[166]

With anticoagulant rodenticides, a number of factors can increase the likelihood of exposure of migratory birds. These factors include the behaviour of exposed rodents, migratory bird ecology, form and type of rodenticide bait, location and rate of rodenticide application.

2.1. Rodent behaviour

Poisoned rodents, which can continue to live for 4-9 days after ingesting a lethal rodenticide dose, may be, because of slower reaction times, more likely to be captured by predators. One study found poisoned rodents are more likely to stay above ground (73 per cent) instead of in their burrows, increasing the risk of secondary poisoning of predators.[167] Also, rodents exposed to anticoagulant rodenticides show altered behaviour such as spending more time in open areas, staggering, and

and breaking strength in UK raptors exposed to second generation anticoagulant rodenticides. *Bulletin of environmental contamination and toxicology*, *78*(3), 249-251.

[161] Walker, L.A., Llewellyn, N.R., Pereira M.G., Potter, E.D., Sainsbury, A.W. & Shore, R.F. (2012). Anticoagulant rodenticides in predatory birds 2010: a Predatory Bird Monitoring Scheme (PBMS) report. Centre for Ecology & Hydrology, Lancaster, UK. 17pp.

[162] Langford, K. H., Reid, M., & Thomas, K. V. (2013). The occurrence of second generation anticoagulant rodenticides in non-target raptor species in Norway. *Science of The Total Environment*, *450*, 205-208.

[163] Ibid.

[164] Ehrlich, P., Dobkin, D. S., & Wheye, D. (1988). *Birder's Handbook: A Field Guide to the Natural History of North American Birds*. Touchstone.

[165] Thomas et al., *Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada*.

[166] Murray, M. (2011). Anticoagulant rodenticide exposure and toxicosis in four species of birds of prey presented to a wildlife clinic in Massachusetts, 2006-2010. *Journal of Zoo and Wildlife Medicine*, *42*(1), 88-97.

[167] Tuyttens, F. A., & Stuyck, J. J. (2002). Effectiveness and efficiency of chlorophacinone poisoning for the control of muskrat (Ondatra zibethicus) populations. *New Zealand Journal of Zoology*, *29*(1), 33-40.

sitting motionless before death, all of which may increase susceptibility to predation.[168] Up to twice as many successful avian predator attacks were on injured or abnormal prey.

Rodent outbreaks have been suggested as potentially increasing the likelihood of exposure. For example, in Spain in 2007, there was an increase of anticoagulant rodenticides poisoning associated with the large scale use of bromadiolone and chlorophacinone against an outbreak of common voles.[169] In some parts of Europe, voles are the most common rodent pest in productive lowland agricultural areas and therefore, birds sharing habitats with voles may be a higher risk of exposure to rodenticides.[170]

2.2. Migratory bird ecology

Birds that forage in agricultural landscapes are most likely to be exposed to anticoagulant rodenticides, as use of these products is primarily in agricultural areas. However, some species' ecology will make them more likely to be exposed than others within these areas.

2.2.1. Predator and scavenger species

Many raptor species are especially likely to be exposed to rodenticides due to a regular diet of rodents. Scavenging species may be especially at risk because they feed on carcasses that could be contaminated with rodenticides. The red kite, for example, may be particularly susceptible to secondary poisoning because of the high proportion of carrion in its diet, including rat carcasses.[171] One study found common buzzards to be the most contaminated species and more susceptible to exposure than other raptors, as a result of almost half of its diet made-up of small rodents.[172]

Raptor species are exposed through, for example, scavenging or preying upon commensal target species (rats and house mice), other mice, voles, and carrion. Up to nearly half of the local populations of non-target small mammals have been found to feed on rodenticide baits used during rat control around farm buildings and pheasant feeders [173] and therefore, may be a source of secondary exposure to predators that do not feed on commensal species, and to scavengers. Additionally, some rodents are highly mobile and can be exposed to rodenticide applications occurring in neighbouring farms.[174]

Birds that feed on non-commensal rodent species in open agricultural areas (rather than around buildings) are also subject to secondary exposure to rodenticides. For example, the migratory red kite's diet in France consists of more than 90 per cent of water voles. Water vole outbreaks are widely treated with rodenticides, and in a study of residues in water voles, over 99 per cent of voles

[168] Cox, P., & Smith, R. H. (1992). Rodenticide ecotoxicology: pre-lethal effects of anticoagulants on rat behaviour.
[169] Sánchez-Barbudo et al., *Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain*.
[170] Jacob, J., & Tkadlec, E. (2010). Rodent outbreaks in Europe: dynamics and damage. *Rodent outbreaks: ecology and impacts*, 207-223.
[171] Carter, I., & Burn, A. (2000). Problems with rodenticides: the threat to red kites and other wildlife. *British Wildlife*, *11*(3), 192-197.
[172] Génsbøl, B. (1999). Guide des rapaces diurnes: Europe, Afrique du Nord et Moyen-Orient. Delachaux et Niestlé in Lambert, O., Pouliquen, H., Larhantec, M., Thorin, C., & L'Hostis, M. (2007). Exposure of raptors and waterbirds to anticoagulant rodenticides (difenacoum, bromadiolone, coumatetralyl, coumafen, brodifacoum): epidemiological survey in Loire Atlantique (France). Bulletin of environmental contamination and toxicology, 79(1), 91-94.
[173] Brakes, C. R., & Smith, R. H. (2005). Exposure of non-target small mammals to rodenticides: short-term effects, recovery and implications for secondary poisoning. *Journal of Applied Ecology*, *42*(1), 118-128.
[174] Tosh, D. G., McDonald, R. A., Bearhop, S., Llewellyn, N. R., Montgomery, W. I., & Shore, R. F. (2012). Rodenticide exposure in wood mouse and house mouse populations on farms and potential secondary risk to predators. *Ecotoxicology*, *21*(5), 1325-1332.

found underground and 41 per cent of voles found or captured above ground contained bromadiolone residues,[175] and vole control with bromadiolone has resulted in the poisoning of red kites. [176] Between 1992 and 2002, 62 red kites with suspected poisoning were submitted to SAGIR (the French Wildlife Disease Surveillance System) and bromadiolone was confirmed as the cause of death in 24 cases and chlorophacinone in 3 other cases. The proportion of confirmed cases was significantly higher in red kites (82 per cent) compared with other wildlife (54 per cent). The study was unable to determine the impact of poisoning on the population, but estimated that there was mortality of a few individuals per thousand based on poisoning from both rodenticides and insecticides.[177]

In the United Kingdom, red kites analysed under a monitoring scheme (the Predatory Bird Monitoring Scheme) were widely exposed to ARs (approx. 74 per cent).[178] The pattern of exposure (types of compounds to which they are exposed) in red kites is similar to other avian predators in Britain and reflects usage patterns (see the discussion on how application method affects exposure below). Bromadiolone and difenacoum, the two commonly used ARs in Britain that are licensed for both indoor and outdoor use, were most frequently detected in the kites. Brodifacoum and flocoumafen (higher acute toxicity) was less common and flocoumafen occurred rarely, which could be because both of these rodenticides and/or their use has been limited to indoors only in the UK.[179] However, the proportion of kites that contained detectable residues and the magnitude of those residues were generally greater than in most other predators, except for kestrels. The relationship between AR levels and physiological effects is poorly understood and there is a large inter-species variation in the toxicity of rodenticides,[180] but residues in some kites were at levels potentially lethal to barn owls. [181] And, rodenticide-induced mortalities have been recorded in this species.

In a sample of tawny owls in Great Britain between 1990-1993 and 2003-2005, 20 per cent of birds contained detectable residues of one or more SGARs. Of the raptors analysed to date in Britain, tawny owls had the lowest proportion of individuals with SGARs residues and therefore, may be the least vulnerable to exposure of ARs.

Pest outbreaks may pose a particular risk of exposure to birds whose feeding preferences change with prey availability. For example, red kites may target water vole outbreaks because of its flexible feeding behaviour.[182] As an opportunistic predator and scavenger, it may exhibit local diet specialisation according to prey availability. Additionally, some scavenging and predatory species

[175] Sage et al., *Kinetics of bromadiolone in rodent populations and implications for predators after field control of the water vole, Arvicola terrestris*.

[176] Coeurdassier, M., Poirson, C., Paul, J.P., Rieffel, D., Michelat, D., Reymond, D., ... & Scheifler, R. (2012). The diet of migrant Red Kites Milvus milvus during a Water Vole Arvicola terrestris outbreak in eastern France and the associated risk of secondary poisoning by the rodenticide bromadiolone. *Ibis*, *154*(1), 136-146.

[177] Berny, P., & Gaillet, J. R. (2008). Acute poisoning of red kites (Milvus milvus) in France: data from the SAGIR network. *Journal of wildlife diseases*, *44*(2), 417-426.

[178] Walker, L. A., Shore, R. F., Turk, A., Pereira, M. G., & Best, J. (2008). The predatory bird monitoring scheme: identifying chemical risks to top predators in Britain. AMBIO: A Journal of the Human Environment, 37(6), 466-471.

[179] http://www.hse.gov.uk/biocides/downloads/er-sgar.pdf

[180] World Health Organization. (1995). Anticoagulant rodenticides. *Environmental Health Criteria*, *175*.

[181] Walker, L. A., Shore, R. F., Turk, A., Pereira, M. G., & Best, J. (2008). The predatory bird monitoring scheme: identifying chemical risks to top predators in Britain. *AMBIO: A Journal of the Human Environment*, *37*(6), 466-471.

[182] Coeurdassier et al., *The diet of migrant Red Kites Milvus milvus during a Water Vole Arvicola terrestris outbreak in eastern France and the associated risk of secondary poisoning by the rodenticide bromadiolone*.

preferentially feed on the liver, which contains a higher proportion of rodenticides than other tissues, increasing the risk of exposure in those bird species.[183]

2.2.2. Insectivorous species

Insectivorous species may be susceptible to exposure through the ingestion of contaminated insects. Invertebrates can become contaminated through consumption of rodent carcasses and faeces, ingestion of soil residues, eg, earthworms, and direct consumption of poison baits, including those placed in bait stations.[184] Two unpublished studies indicated that in insects exposed to ARs, residue levels take in excess of four weeks to return to background levels, and trace levels are detectable up to ten weeks following brodifacoum baiting operations, which poses a risk to insectivorous bird species.[185] Insects may not bio-accumulate ARs after repeated exposures,[186] thereby lowering the likelihood of increased toxicity to insectivorous birds. However, as insectivorous species will feed on many invertebrates, the potential exists for bioaccumulation to occur. One study suggested that a granivorous species, the crested partridge (*Rollulus roulroul*), may have died by consuming cockroaches exposed to the SGAR brodifacoum.[187]

A study on hedgehogs (*Erinaceus europaeus*) in the UK, whose diet consists primarily of invertebrates, found that contamination of hedgehogs with ARs is commonplace.[188] Whether the exposure in hedgehogs results in any lethal or sub-lethal effects is unknown. Anticoagulant rodenticides may, therefore, pose a similar risk to other species whose diets consist of insects, such as insectivorous birds.[189] However, the impact of ARs on insectivorous species, both through primary and secondary poisoning routes, is currently a significant knowledge gap.

In wetlands, rodenticides are commonly used to protect crops, such as rice. Baits used in wetland habitats are fixed on rafts away from banks to limit the contamination of terrestrial species, but not necessarily that of waterbirds. Herbivorous bird species are at risk of exposure to rodenticides that are baited in cereal, carrot, beet or apple-based baits. The primary method of exposure of waterbirds in one study was the direct, primary consumption of treated baits (cereals, carrots or apples).[190] In the same study, the fast disappearance of bait after application suggested ingestion of the bait by waterbirds.

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[183] Sage et al., *Kinetics of bromadiolone in rodent populations and implications for predators after field control of the water vole, Arvicola terrestris*.

[184] Eason, C. T., Murphy, E. C., Wright, G. R., & Spurr, E. B. (2002). Assessment of risks of brodifacoum to non-target birds and mammals in New Zealand. Ecotoxicology, 11(1), 35-48; Dunlevy, P. A., Campbell III, E. W., & Lindsey, G. D. (2000). Broadcast application of a placebo rodenticide bait in a native Hawaiian forest. International biodeterioration & biodegradation, 45(3), 199-208; and Spurr, E. B., & Drew, K. W. (1999). Invertebrates feeding on baits used for vertebrate pest control in New Zealand. New Zealand Journal of Ecology, 23(2), 167-173.

[185] Craddock, P. (2003). *Aspects of the ecology of forest invertebrates and the use of brodifacoum* (Doctoral dissertation, ResearchSpace@ Auckland); Booth, L. H., Fisher, P., Heppelthwaite, V., & Eason, Toxicity and residues of brodifacoum in snails and earthworms.

[186] Craddock, P. (2003). *Aspects of the ecology of forest invertebrates and the use of brodifacoum* (Doctoral dissertation, ResearchSpace@ Auckland).

[187] Borst, G. H. A., & Counotte, G. H. M. (2002). Shortfalls using second-generation anticoagulant rodenticides. *Journal of Zoo and Wildlife Medicine*, *33*(1), 85-85.

[188] Dowding, C. V., Shore, R. F., Worgan, A., Baker, P. J., & Harris, S. (2010). Accumulation of anticoagulant rodenticides in a non-target insectivore, the European hedgehog (*Erinaceus europaeus*). *Environmental Pollution*, *158*(1), 161-166.

[189] Ibid.

[190] Berny, P. J., Buronfosse, T., Buronfosse, F., Lamarque, F., & Lorgue, G. (1997). Field evidence of secondary poisoning of foxes (Vulpes vulpes) and buzzards (Buteo buteo) by bromadiolone, a 4-year survey. *Chemosphere*, *35*(8), 1817-1829 in Lambert, O., Pouliquen, H., Larhantec, M., Thorin, C., & L'Hostis, M. (2007). Exposure of raptors and waterbirds to anticoagulant

2.3. Form of bait

Commercial anticoagulant rodenticide baits often used by farmers can be purchased in the form of pellets, loose meal, paraffin blocks or packet baits, and are available from various companies and in varying concentrations. [191]

Grain-based rodenticide baits have the potential to expose granivorous birds. For example, when treated-grain is distributed on the surface with spreader machines, there is the potential to seriously affect populations of granivorous birds.[192]

Grain and fruit-based rodenticide baits may not result in exposure of birds of prey. Raptors, as predators and scavengers, are unlikely to suffer from primary poisoning because of their feeding ecology, especially if baits are treated cereals, carrots or apples. Birds of prey may be less at risk of primary exposure to treated-grain as they generally do not consume pellets or grain-type foods. However, they are more likely to be exposed secondarily as a result of consumption of poisoned prey.

2.4. Application types

Practices such as the lack of protection of bait stations, broadcast baiting, permanent baiting, and failure to remove bait at the end of baiting campaigns are likely to increase the risk of primary and secondary poisoning.[193] Indeed, how the rodenticide is applied may have more of an effect than how often it is applied. For example, during the Foot and Mouth outbreaks in the United Kingdom, large amounts of ARs were used on farms without increasing exposure in buzzards and barn owls.[194]

A broadcast method of rodenticide dispersal, commonly employed in France to tackle vole outbreaks is associated with a particularly high risk to non-target wildlife. This untargeted approach is believed to be the primary reason why secondary poisoning by rodenticides is an issue of conservation concern in France, but generally not in other countries of the species' range, where deliberate illegal poisoning is a more significant factor.[195]

In a monitoring study in Spain, granivorous birds showed the highest prevalence of primary anticoagulant rodenticide exposure, especially to chlorophacinone in a region treated against a vole population in 2007. [196] Nocturnal raptors had the highest rate of secondary exposure in the same study. The risk of primary poisoning of granivorous species may be reduced by placing baits in areas only accessible by the target rodent, such as burrows (see below).

rodenticides (difenacoum, bromadiolone, coumatetralyl, coumafen, brodifacoum): epidemiological survey in Loire Atlantique (France). *Bulletin of environmental contamination and toxicology*, *79*(1), 91-94.

[191] Albert et al., *Anticoagulant rodenticides in three owl species from western Canada, 1988–2003*.

[192] Sánchez-Barbudo et al., *Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain*.

[193] Shore, R. F., Pereira, G. M., Potter, E. D., and Walker, L. A. (2013). Monitoring rodenticides residues in wildlife (*in press*).

[194] Shore, R.F., Malcolm, H.M., McLennan, D., Turk, A., Walker, L.A., Wienburg, C.L., and Burn, A.J. (2006). Did Foot-and-Mouth Disease-Control Operations Affect Rodenticide Exposure in Raptors?. The Journal of Wildlife Management, 70: 588–593.

[195] Knott, J, P. Newbery, and B. Barov (2009). Action plan for the red kite Milvus milvus in the European Union, 55 p. ISSN (…)

[196] Sánchez-Barbudo et al., *Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain*.

The rate of application may also influence the likelihood of exposure in target and non-target species. For example, the risk of secondary poisoning to predators of wood mice was suggested to be lower 2-3 months after initiation of seasonal baiting on farms compared with immediately after onset.[197]

The use of tamper-proof bait stations may reduce primary poisoning of non-target wildlife. In Canada, rodenticide labels are required to include that either tamper-proof bait stations must be used or bait placed in locations not accessible to children, pets or livestock. Generally in Canada, second-generation anticoagulants are largely restricted to indoor use or against the outside walls of buildings.[198] The extent to which bait stations are used and the rate of compliance with label restrictions is unknown. In addition, the use of bait stations may lower uptake by rodents, necessitating longer baiting periods. The potential impact of this on exposure rates is poorly understood.

Permanent baiting stations will also significantly increase risk to non-target wildlife, both by direct exposure (primary or secondary poisoning) and through increased the likelihood of resistance developing in the local rodent population, necessitating use of more toxic products, associated with greater risk to non-target wildlife, in order to deliver effective rodent control.

A survey of farms in Northern Ireland showed that most farmers baited for rodents every year, and many baited for prolonged periods or permanently. Complete compliance with best practice guidance for baiting on farms was rare, which may increase the likelihood of exposure of non-target species.[199]

2.5. Application location: areas where lower likelihood of exposure

2.5.1. Climate and geography

Certain climates and geographical features may influence the likelihood of exposure. For example, in areas with cold climates and lower human population densities there may be less need for rodent control as fewer rodents are likely to come into contact with people and be subject to control in these areas. However, one study found similar rates of exposure to SGARs of raptors sampled in monitoring programmes in Europe and Canada, despite the potential for lower AR use in the harsh Canadian climate and lower human population density. The similar rate of exposure may be due to a sampling bias as the sampled areas of Canada were those with higher population densities,[200] or due the wide ranging behaviour of the raptors involved.

2.5.2. Indoor versus outdoor use

Rodenticide baits used in-and-around farmyard buildings may affect wildlife differently than baits used in fields to protect crops. Baits used in-and-around buildings target commensal rodent species and are, therefore, most likely to affect bird species that prey on or scavenge commensal rodents (eg, red kite). Rodenticides that are restricted to indoor-use only are less likely to be encountered by non-target small mammals, and, therefore, pose a relatively low likelihood of primary exposure of wildlife. For example, a study on barn owls in New Jersey, USA, found no adverse impacts associated with the use of SGARs (brodifacoum) treated baits on farms because the birds tended to feed in

[197] Tosh et al., *Rodenticide exposure in wood mouse and house mouse populations on farms and potential secondary risk to predators*.
[198] Albert et al., *Anticoagulant rodenticides in three owl species from western Canada, 1988–2003*.
[199] Tosh et al., *User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use*.
[200] Thomas et al., *Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada*.

grasslands away from the buildings were the applications were being made.[201] Additionally, as birds are less likely to have direct access to these rodenticides (if indoors or used in bait stations), they are less likely to be primarily exposed. And, in the United Kingdom, compounds licensed for use outside are more frequently detected in some wildlife than compounds that are licensed for indoor and outdoor use.[202] Although, it is unclear whether this difference reflects where compounds are used or differences in the amounts used.[203] However, unless rodenticides are deployed in a largely closed system (e.g., sewers), use inside buildings may result in exposure to birds. Exposure may occur when the poisoned rodents spend time outside of the building in which the bait is deployed. In this way, indoor use can be said to lower risk, but not eliminate it.

In contrast, rodenticides used in fields and other open areas, may affect a wide variety of bird species that prey on rodents and other small mammals. Grain-based baits also have the potential to affect granivorous birds through direct primary poisoning. Baits in agricultural fields may also expose insects and therefore, putting insectivorous birds at risk of exposure.

Further restrictions on anticoagulant rodenticides (and other control agents, such as zinc phosphide) were introduced in the USA in 2012, including the requirement for bait stations for some ARs used in outdoor agricultural areas, such as brodifacoum.[204] These new restrictions may change the likelihood of exposure in birds. In Europe, the Biocidal Products Directive (98/8/EEC) has recently been enacted to harmonise approvals for rodenticides and other biocides across the European Union. This will significantly alter the approvals allowed for AR use. For example, in the UK, products previously allowed to be used in open areas will be restricted to use in-and-around buildings, while others only allowed to be used inside, will now be available for use in-and-around buildings. These details are currently subject to public consultation in the UK and no details on likely or documented changes to risk profiles are currently available.

2.6. Crop types

Certain types of crops are more likely to require rodent control and/or are more likely to attract bird species to a foraging location. For example, the rice field rat (*Rattus argentiventer*) is a serious pest in Malaysia where rice fields attract large numbers of rats. As a result, there is often widespread use of rodenticides in rice fields[205] In India, sugarcane crops are a readily available source of food and cover for feeding, burrowing and breeding activities of rodents nearly year-round and rodents are often controlled using ARs.[206]

3. Toxicity

If exposure to anticoagulant rodenticides is likely to occur, the toxicity level of the AR will greatly influence the corresponding effect – whether lethal or sub-lethal. Additionally, certain bird species

[201] Hegdal, P. L., & Blaskiewicz, R. W. (1984). Evaluation of the potential hazard to barn owls of Talon (brodifacoum bait) used to control rats and house mice. *Environmental toxicology and chemistry*, *3*(1), 167-179.
[202] Dowding et al., *Accumulation of anticoagulant rodenticides in a non-target insectivore, the European hedgehog* (*Erinaceus europaeus*).
[203] Ibid.
[204] http://www.epa.gov/oppsrrd1/reregistration/rodenticides/finalriskdecision.htm
[205] Lam, Y. M. (1988). Rice as a trap crop for the rice field rat in Malaysia.
[206] Singla, N., & Babbar, B. K. (2012). Critical Timings of Rodenticide Bait Application for Controlling Rodents in Sugarcane Crop Grown in Situations Like Punjab, India. Sugar Tech, 14(1), 76-82.

are more likely to be affected by ARs because of their unique physiology. The toxicity of first and second generation anticoagulant rodenticides and species at risk of experiencing adverse effects as a result of exposure are discussed below.

Anticoagulant rodenticides are distinguished by first or second generation: first-generation anticoagulants (FGARs), developed after World War II, typically require multiple feedings to result in mortality; and second-generation anticoagulants (SGARs) are more recently developed and have a greater toxicity, such that typically only a single feed is required to result in mortality .[207] FGARs have, in many areas, been superseded to a large extent by second-generation anticoagulant rodenticides which were developed in response to the emergence of genetic resistance to warfarin in rats and mice.[208]

3.1. First-generation anticoagulant rodenticides

First-generation anticoagulants include warfarin, pival, coumafuryl, coumachlor, coumatetralyl, diphacinone, and chlorophacinone. These compounds act as chronic toxicants, requiring multiple exposures over a short period of time (days) to be effective in causing mortality.[209] Generally, the first generation anticoagulants have relatively low persistence. For example, warfarin has a half-life of between 5-28 hours in animal tissue. Due to their low toxicity and more limited persistence, the likelihood of toxicity from FGARS is lower than that from second generation anticoagulants.[210] For example, one study of tawny owls found a low probability of secondary poisoning from first generation compounds.[211] Also, FGAR residues in prey are likely to pose a lower risk of secondary poisoning than SGAR residues because of their more limited persistence.[212]

Many commensal rodents have developed resistance to FGARs, which means they can ingest large amounts of rodenticides yet survive.[213]

The restrictions on the use of certain SGARs in the US may result in an increased use of other acute toxicants and other anticoagulant rodenticides. [214] Therefore, policy-makers need to ensure the SGAR restrictions do not result in the use of riskier alternative substances.

However, there have been few documented non-target wildlife poisoning incidents involving first-generation anticoagulants and limited studies on their potential risks to birds in the field. Due to their shorter persistence in tissues, there is a lack of good information on residues of this group of

[207] Endepols, S., Prescott, C. V., Klemann, N., & Buckle, A. P. (2007). Susceptibility to the anticoagulants bromadiolone and coumatetralyl in wild Norway rats (Rattus norvegicus) from the UK and Germany. International Journal of Pest Management, 53(4), 285-290.

[208] Tosh, D. G., McDonald, R. A., Bearhop, S., Lllewellyn, N. R., Fee, S., Sharp, E. A., ... & Shore, R. F. (2011). Does small mammal prey guild affect the exposure of predators to anticoagulant rodenticides?. Environmental Pollution, 159(10), 3106-3112.

[209] Hadler, M. R., & Buckle, A. P. (1992). Forty five years of anticoagulant rodenticides—past, present and future trends.

[210] Ibid.; Hosea, R. C. (2000, March). Exposure of non-target wildlife to anticoagulant rodenticides in California. In Proceedings of the 19th Vertebrate Pest Conference (Vol. 19, pp. 236-244).

[211] Townsend, M. G., Fletcher, M. R., Odam, E. M., & Stanley, P. I. (1981). An assessment of the secondary poisoning hazard of warfarin to tawny owls. The Journal of Wildlife Management, 45(1), 242-248.

[212] Burn et al., *The threats to birds of prey in the UK from second-generation rodenticides*.

[213] Baert, K., Stuyck, J., Breyne, P., Maes, D., & Casaer, J. (2012). Distribution of anticoagulant resistance in the brown rat in Belgium. Belg. J. Zool, 142(1), 39-48.

[214] Rattner et al., *Acute toxicity, histopathology, and coagulopathy in American kestrels (Falco sparverius) following administration of the rodenticide diphacinone*.

rodenticides in wildlife.[215] However, due to the lower toxicity and persistence, it is generally accepted that FGARs represent a significantly lower risk to non-target wildlife than equivalent use of SGARs.

As a result of FGARs being largely replaced by SGARs, this review will focus on the toxicity of SGARs, which is discussed below.

3.2. Second-generation anticoagulants

Commonly used SGARs, such as brodifacoum, bromadiolone, flocoumafen, and difenacoum, are classified as second-generation anticoagulant rodenticides (SGARs). SGARs were introduced in the 1970s following widespread development of rodent resistance to first-generation anticoagulant rodenticides. SGARs are the primary means of controlling rodents in many developed countries and are used worldwide.[216] This class of rodenticides are much more acutely toxic than first-generation anticoagulant rodenticides, generally providing a lethal dose to rodents after a single feeding. They also tend to be more persistent in animal tissues and have a higher affinity for liver tissue, which is often an attractive food source for predators/scavengers. Based on brodifacoum and bromadiolone, it was predicted the likely persistence of residues after a sub-lethal dose in target and non-target animals could be up to 24 months.[217] As a result, second generation anticoagulant rodenticides represent a greater risk of secondary poisoning than FGARs to migratory birds because of their toxicity and biophysical persistence.

Table 2: Relative toxicity to birds of common second-generation anticoagulant rodenticides

A number of studies have indicated that brodifacoum appears to have the greatest potential for non-target wildlife mortality due to its physiological persistence in body tissues and acute toxicity.[218]
Several of the most highly toxic SGARs are restricted to particular uses, eg, use limited to indoors or around buildings in certain countries. For example, a risk assessment conducted by the United States Environmental Protection Agency in 2002 identified several rodenticides that pose significant risk to birds and non-target mammals, in part because of their toxicity and persistence. As a result, in 2008, the US imposed restrictions on the sale, distribution and packaging of brodifacoum, difethialone, bromadiolone and difenacoum. [219]
For similar reasons, legislation in the UK currently draws a distinction between brodifacoum, flocoumafen and difethialone (only licensed for indoor use), and difenacoum and bromadiolone (licensed for outdoor use), although this distinction may be removed under a proposed new approvals system.[220] At the time of this study, the majority of farmers and pest controllers in the UK use the outdoor licensed SGARs: difenacoum and bromadiolone.[221] The SGARs that are licensed for indoor use only (flocoumafen, brodifacoum and difethialone) are used less extensively on farms (<3 per cent of farms and <1 per cent of total amount of active substance[222]), except in Northern Ireland where higher usage occurs.[223]

[215] Burn et al., *The threats to birds of prey in the UK from second-generation rodenticides*.
[216] Tosh et al., *User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use*.
[217] Eason, C., Miller, A., Ogilvie, S., & Fairweather, A. (2011). An updated review of the toxicology and ecotoxicology of sodium fluoroacetate (1080) in relation to its use as a pest control tool in New Zealand. *New Zealand Journal of Ecology*, *35*(1), 1-20.
[218] Stone, W. B., Okoniewski, J. C., & Stedelin, J. R. (2003). Anticoagulant rodenticides and raptors: recent findings from New York, 1998–2001. *Bulletin of environmental contamination and toxicology*, *70*(1), 0034-0040 and Stone, W. B., & Okoniewski, J. C. (1999). Poisoning of wildlife with anticoagulant rodenticides in New York. *Journal of Wildlife Diseases*, *35*(2), 187-193.
[219] Rattner et al., *Acute toxicity, histopathology, and coagulopathy in American kestrels (Falco sparverius) following administration of the rodenticide diphacinone*.
[220] http://www.cieh.org/assets/0/72/1126/1132/06170d08-8f18-4023-bc24-3a6380f108fb.pdf
[221] Dawson, A., Bankes, J., & Garthwaite, D. (2001). *Rodenticide use on farms in Great Britain growing arable crops 2000*. Defra.
[222] Ibid.

With the use of SGARs, rodents survive for several days after consuming a lethal dose and often will continue feeding on the bait.[224] This increases the likelihood that the body burden in poisoned rodents may significantly exceed the lethal dose needed to kill them (and so present a greater poisoning risk to predators), and poisoned animals may remain active and available for capture by predators for some period after ingestion of the rodenticide.[225] Therefore, predatory or scavenging birds may be more likely to ingest a high dose of SGARs from feeding on contaminated rodents, which, in-turn, may increase the likelihood of lethal or sub-lethal effects in birds. For example, the barn owl (*Tyto alba*) population in oil palm plantations on the Malaysian peninsula declined dramatically following the replacement of a first generation rodenticide (warfarin) with baits containing SGARs (brodifacoum).[226]

The most frequent anticoagulant detected in the liver of birds in a study in France was bromadiolone.[227] Bromadiolone was rarely detected in herbivorous species like mallards.[228] In a New York study, brodifacoum was the most frequently detected anticoagulant (84 per cent) in 265 raptors.[229] The presence of brodifacoum and bromadiolone in non-target species has continued in the United States despite the restriction of their use to in-and-around structures.

Exposure to anticoagulant rodenticides may be occurring more frequently than are detected. [230] It has been suggested that birds suffering from adverse exposure to anticoagulant rodenticides may be likely to die undiscovered in their roosts, as a period of lethargy may precede death. Therefore, while long-running monitoring scheme may assess changes in exposure rates over time, it is difficult to accurately estimate effects on bird populations.

One model estimates the use of SGARs may result in a mortality rate of 11 per cent for great horned owls (based upon a 65 per cent likelihood of being exposed to SGARs and 17 per cent likelihood of exhibiting toxic effects as a result of the exposure). [231] However, this is the first estimate of population effects for a wild raptor population and its estimate may be too low (or too high) as an unknown number of birds die out of sight as a result of SGAR exposure (as a period of lethargy may precede death),[232] which is not reflected in the model. It also does not include consideration of sub-lethal effects of SGAR exposure, which are largely unknown. But since a high proportion of birds are exposed to sub-lethal amounts of SGARs, any effects could be widespread. However, this is only a preliminary study and the uncertainties around it are unquantified to some extent. Therefore, the true level of mortality may be greater or lesser, but it is the only estimate that we have to date. It is unknown whether this level of individual mortality would result in population effects.

[223] Tosh et al., *User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use*.

[224] Cox and Smith, *Rodenticide ecotoxicology: pre-lethal effects of anticoagulants on rat behaviour*.

[225] Thomas et al., *Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada*.

[226] Duckett, J. E. (1984). Barn owls (Tyto alba) and the 'second generation rat-baits utilised in oil palm plantations in Peninsular Malaysia. *Planter, Kuala Lumpur*, *60*, 3-11.

[227] Lambert, O., Pouliquen, H., Larhantec, M., Thorin, C., & L'Hostis, M. (2007). Exposure of raptors and waterbirds to anticoagulant rodenticides (difenacoum, bromadiolone, coumatetralyl, coumafen, brodifacoum): epidemiological survey in Loire Atlantique (France). *Bulletin of environmental contamination and toxicology*, *79*(1), 91-94.

[228] Ibid.

[229] Stone, W. B., Okoniewski, J. C., & Stedelin, J. R. (2003). Anticoagulant rodenticides and raptors: recent findings from New York, 1998–2001. *Bulletin of environmental contamination and toxicology*, *70*(1), 0034-0040.

[230] Albert et al., *Anticoagulant rodenticides in three owl species from western Canada, 1988–2003*.

[231] Thomas et al., *Second generation anticoagulant rodenticides in predatory birds*.

[232] Newton, I., Wyllie, I., & Freestone, P. (1990). Rodenticides in British barn owls. *Environmental pollution*, *68*(1), 101-117.

3.2.1. Species particularly susceptible based on physiology

Variation between species in their accumulation of liver SGAR residues may, in part, be due to species differences in metabolism and excretion but is also likely to result from differences in dietary exposure.[233]

Species vary widely in their sensitivity to SGARs, and very little is known about the relative sensitivity of different avian species.[234] A study suggested significant differences exist among raptor species.[235] A potential toxic range in the magnitude of liver residues that may be associated with mortality has been suggested for barn owls but this gives no indication of likelihood of effects in individual birds.[236]

Some studies indicate that raptors are considerably more sensitive to diphacinone than bobwhite and mallards, and effective protection from secondary poisoning risks may require more substantial safety margins than afforded by avian game bird species traditionally used in pesticide registration studies. For example, one study found the acute oral toxicity of the anticoagulant rodenticide diphacinone to be over 20 times greater in American kestrels (*Falco sparverius*) than the northern bobwhite (*Colinus virginianus*) and mallards (*Anas platyrhynchos*). [237]

3.2.2. Sub-lethal effects

There is a lack of published information on the effects of sub-lethal doses of second generation anticoagulant rodenticides, which are largely unknown. [238] Sub-lethal effects could include haemorrhages which interfere with locomotion, predisposing animals to predation; accidental trauma; toxic injury to the liver and reduced food intake. Inadequate nutrition may then predispose birds to infectious and parasitic disease, hypothermia, or poisoning with pesticides stored in fat.[239]

Sub-lethal exposure to SGARs may hinder the recovery of birds from non-fatal collisions or accidents if they lead to sub-lethal but prolonged clotting times. They may also impair hunting ability through behavioural changes, such as lethargy, potentially increasing the probability of starvation. However, there is limited evidence of sub-lethal effects occurring in the field, and those attributed to anticoagulants may be due to other correlative factors. [240] In laboratory rats, sub-lethal exposure to rodenticides resulted in increased abortion rates, but these types of reproductive effects have not been studied in birds. [241]

[233] Tosh et al., *Does small mammal prey guild affect the exposure of predators to anticoagulant rodenticides?*
[234] Walker, L. A., Turk, A., Long, S. M., Wienburg, C. L., Best, J., & Shore, R. F. (2008). Second generation anticoagulant rodenticides in tawny owls (< i> Strix aluco</i>) from Great Britain. *Science of the Total Environment*, *392*(1), 93-98.
[235] Thomas et al., *Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada*.
[236] Newton, I., Shore, R. F., Wyllie, I., Birks, J. D. S., & Dale, L. (1999). Empirical evidence of side-effects of rodenticides on some predatory birds and mammals. *Advances in vertebrate pest management*, *1*, 347.
[237] Rattner et al., *Acute toxicity, histopathology, and coagulopathy in American kestrels (Falco sparverius) following administration of the rodenticide diphacinone*.
[238] Burn et al., *The threats to birds of prey in the UK from second-generation rodenticides*.
[239] Albert et al., *Anticoagulant rodenticides in three owl species from western Canada, 1988–2003*.
[240] Thomas et al., *Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada*.
[241] Burn et al., *The threats to birds of prey in the UK from second-generation rodenticides*.

Table 3: Knowledge Gaps/discussion questions

➢ What is the variation in exposure rates of birds that prey upon commensal rodent species versus wild rodent species?
➢ How does the restriction of rodenticides to in-and-around buildings affect exposure rates of rodents and non-target birds?
➢ What are the behavioural effects of exposure to sub-lethal doses of SGARs?
➢ Does a higher proportion of diet of rodents increase the risk of exposure to SGARs?
➢ How do SGARs affect species other than raptors? Do some bird species' physiology make them particularly susceptible to lethal or sub-lethal effects as a result of anticoagulant rodenticide exposure?
➢ Does the likelihood of exposure to SGARs vary by the type of agriculture? Do some require more intensive use of SGAR rodenticides? Vary by season, eg, different application rates at different times of the year?
➢ Does the likelihood of exposure to SGARs vary by the type of rodents present?
➢ Does exposure to SGARs vary with rodenticide application practice? Annually versus permanent baits?
➢ Does exposure to SGARs vary with the use of bait protection equipment? Use of bait stations vs broadcast dispersal?
➢ How much of exposure is due to mis-use of labelled requirements for SGAR use?
➢ How geographically widespread is this an issue? Although rodenticides are used globally, evidence for impacts on non-target wildlife comes almost exclusively from Europe and North America.

4. Conclusion

There is wide-spread exposure of raptors to rodenticides where second-generation anticoagulant rodenticides are used in agriculture, but the ecologically-significant effects (both lethal and sub-lethal) from exposure are largely unknown. Additionally, it is unknown whether there are any population level effects from exposure. There is also scant knowledge of SGAR exposure rates in birds outside Europe, North America and Australasia.

In addition to research needed to determine whether there are population effects resulting from widespread exposure in some species, further research is also needed to identify the exposure rate of rodenticides in species other than raptors as some evidence indicates that grain-based baits could result in exposure of granivorous bird species. Even between raptor species, the variation of effects is little understood.

Effects of predator control and harvesting of birds using poison-baits

1. Introduction

Predator control by humans is as old as livestock husbandry. The control of many mammalian and avian predator species has been a feature of human development for centuries.[242] Poison baiting is probably the most widely used predator eradication method worldwide.[243] It is also illegal to use poison-baits in many countries.

The use of poison-baits to control predators is illegal in the European Union through the Bern Convention and the Birds Directive. Illegal baiters in the United States can be prosecuted under the Federal Insecticide, Fungicide and Rodenticide Act 1947 for using any registered pesticide in a manner inconsistent with its labelling.[244] If bird of prey carcasses are found in the vicinity of the bait site, the suspects may also be charged with violations of the Migratory Bird Treaty Act, the Bald and Golden Eagle Protection Act, the Endangered Species Act, and various state laws.

Migratory birds are exposed to poison-baits that are used for predator control (avian and mammalian) and also for those used to harvest birds for human consumption or traditional medicine. Poisoning of scavenging birds occurs when they eat the poisoned-bait (primary poisoning), and also through consumption of poisoned carcasses (secondary poisoning).

Baits to attract the avian and mammalian predators include, large animal carcasses (eg, deer, cow, sheep, and lamb carcasses), small animal carcasses (eg, chicken and rabbit carcasses), animal parts, fish, eggs, and commercial pet food that are laced with high concentrations of liquid, granular or powder forms of highly toxic insecticides (often that are not registered for predator control).[245] The insecticides may be topically applied to the surface of the bait, injected or placed into slits cut in the bait, or soaked into the bait.

This report assesses the risk of poison-baits to migratory bird populations. Population-level effects are determined by a function of (1) the likelihood that migratory birds will be exposed to poison-baits; (2) the toxicity of the poison-baits to migratory birds. Each of these – exposure and toxicity – are discussed in more detail below.

[242] Reynolds, J. C., & Tapper, S. C. (1996). Control of mammalian predators in game management and conservation. *Mammal Review*, *26*(2-3), 127-155.

[243] Herranz, J. (2000). *Efectos de la depredación y del control de predadores sobre la caza menor en Castilla-La Mancha* (Doctoral dissertation, Ph. D. thesis, Autónoma University. Madrid) in Márquez, C., Vargas, J. M., Villafuerte, R., & Fa, J. E. (2012). Understanding the propensity of wild predators to illegal poison baiting. *Animal Conservation*.

[244] United States Code, Title 7, Section 136j, 1972.

[245] Vyas, N. B., Spann, J. W., Hulse, C. S., Bauer, W., & Olson, S. (2005). Carbofuran detected on weathered raptor carcass feet. *Wildlife Society Bulletin*, *33*(3), 1178-1182.

2. Exposure: predator control

Illegal poisoning is a form of persecution usually generated by conflicts with human interests associated with livestock rearing or game management for hunting, and indiscriminately affects birds or mammals that occasionally or regularly feed on carcasses, or other poison-laced baits.[246]

2.1. Mammalian predators

The presence of large mammalian predators may increase the likelihood of exposure of migratory birds to poison-baits.

Carnivore poisoning continues to be common, especially in Europe and Africa, but also occurs outside these areas. In Europe, poisoning is used to kill predators of game animals (e.g., rabbits, pheasants, and partridges) because hunters believe carnivores such as foxes and mongooses reduce their hunting success. In both Europe and Africa, poisoning is used to "protect" livestock from predators. Small-stock (sheep and goats) farmers may use more poison than large-stock (cattle) farmers.[247] For example, most farmers in Kenya consider birds of prey nuisance birds because some species prey on domestic fowl.[248]

Poisoning, resulting from illegal poison-baits, is the main threat for the conservation of different species of raptors in Europe.[249] In Hungary, the mortality of birds of prey due to poisoning has markedly increased since 2007. The study suggested this is most likely related to the use illegal use of carbofuran-laced baits for predator control.[250] In Italy, illegal poison-baits are commonly used.[251] In Mediterranean countries, the use of poison-baits to control predators is a frequent practice that affects several species of vulture. In Greece, poisoning has resulted in the decline of the bearded vulture (*Gypaetus barbatus*).[252] Similar problems exist in many other countries, such as the Netherlands, Spain, Croatia, Austria and the Czech Republic.[253]

The mortality of fifty-four golden eagles and bald eagles in Canada due to poisoning may have been related to the illegal poisoning of coyotes, black bears and skunks.[254] The illegal use of poison-baits

[246] Whitfield, D. P., McLeod, D. R., Watson, J., Fielding, A. H., & Haworth, P. F. (2003). The association of grouse moor in Scotland with the illegal use of poisons to control predators. *Biological Conservation*, *114*(2), 157-163; González, L. M., Margalida, A., Manosa, S., Sánchez, R., Oria, J., Molina, J. I., ... & Prada, L. (2007). Causes and spatio-temporal variations of non-natural mortality in the Vulnerable Spanish imperial eagle Aquila adalberti during a recovery period. *Oryx*, *41*(04), 495-502.

[247] Ledger, J. A. (1985). The poison people. *Afr. Wildl*, *39*, 6-8.

[248] Ogada, D. L., & Kibuthu, P. M. (2008). Conserving Mackinder's eagle owls in farmlands of Kenya: assessing the influence of pesticide use, tourism and local knowledge of owl habits in protecting a culturally loathed species. *Environmental Conservation*, *35*(03), 252-260.

[249] Margalida, A., Heredia, R., Razin, M., & Hernández, M. (2008). Sources of variation in mortality of the Bearded vulture Gypaetus barbatus in Europe. *Bird Conservation International*, *18*(1), 1.

[250] Lehel, J., Laczay, P., Déri, J., Darin, E. G., & Budai, P. (2010). Model study on the clinical signs and residue concentrations of sub-lethal carbofuran poisoning in birds. *Journal of Wildlife Diseases*, *46*(4), 1274-1278.

[251] Giorgi, M., & Mengozzi, G. (2011). Malicious animal intoxications: poisoned baits. *Veterinarni Medicina*, *56*(4), 173-179.

[252] Xirouchakis, S., Sakoulis, A., & Andreou, G. (2001). The decline of the Bearded Vulture Gypaetus barbatus in Greece. *Ardeola*, *48*(2), 183-190.

[253] Richards, N. (Ed.). (2011). *Carbofuran and wildlife poisoning: global perspectives and forensic approaches*. Wiley.

[254] Wobeser, G., Bollinger, T., Leighton, F. A., Blakley, B., & Mineau, P. (2004). Secondary poisoning of eagles following intentional poisoning of coyotes with anticholinesterase pesticides in western Canada. *Journal of Wildlife Diseases*, *40*(2), 163-172.

(laced with insecticides) for controlling avian and mammalian predators is used in the United States by some farms, including poultry and gamebird farmers, and hunting establishments.[255]

2.2. Migratory bird ecology

Certain life history traits and patterns of habitat use may make some scavenging species more susceptible to poisoning.[256] Poison-related mortality often affects breeding adults, and several studies have documented the detrimental effects of illegal poisoning on population dynamics, especially for long-lived species with low reproductive rates and delayed maturity.[257] Direct persecution, including illegal poisoning, affects adult survival, so increasing the replacement of adult breeders creates ecological traps, which attract adult floaters and immature birds, and increases pre-adult mortality in birds that may originate from persecution-free areas.[258]

Figure 1: Examples of species susceptible to poison-baits

Studies in South Africa found threats to the survival of savannah eagles (bateleur *Terathopius ecaudatus*, tawny *Aquila rapax* and martial *Polemaetus bellicosus* eagles) to include inadvertent poisoning from strychnine most likely as baits targeting predators.[259]

An analysis of 267 records of non-natural mortality of the Spanish imperial eagle (*Aquila adalberti*) collected between 1989 and 2004, found poisoning was the main cause of death in approximately 31 per cent of the birds.[260] Of the total birds found dead, 41 per cent were associated with predator control (game practices and livestock protection).[261] The illegal use of poison to control predators was the main cause of mortality (94 per cent) in the Egyptian vulture in Spain, particularly in small hunting reserves (75 per cent).[262]

Vultures are particularly vulnerable to exposure to poison-baits due to a combination of foraging behaviours and life-history traits found collectively only in vultures.[263] Presently, 61 per cent of vulture species worldwide are threatened with extinction, and the most rapid declines have occurred in the vulture-rich regions of Asia and Africa. The reasons for the population declines are varied, but poisoning or human persecution, or both, feature in the list of nearly every declining species. Deliberate poisoning of carnivores is likely to be the most widespread cause of vulture poisoning. Human persecution of vultures has occurred for centuries, and shooting and deliberate poisoning are the most widely practiced activities.[264]

[255] Vyas, N. B., Spann, J. W., Hulse, C. S., Bauer, W., & Olson, S. (2005). Carbofuran detected on weathered raptor carcass feet. *Wildlife Society Bulletin*, *33*(3), 1178-1182.

[256] Virani, M. Z., Kendall, C., Njoroge, P., & Thomsett, S. (2011). Major declines in the abundance of vultures and other scavenging raptors in and around the Masai Mara ecosystem, Kenya. *Biological Conservation*, *144*(2), 746-752; Herholdt, J. J., & Kemp, A. C. (1997). Breeding status and ecology of the Martial Eagle in the Kalahari Gemsbok National Park, South Africa. *Ostrich*, *68*(2-4), 80-85.

[257] Hernández, M., & Margalida, A. (2009). Poison-related mortality effects in the endangered Egyptian vulture (Neophron percnopterus) population in Spain. *European Journal of Wildlife Research*, *55*(4), 415-423.

[258] Whitfield, D. P., Fielding, A. H., McLeod, D. R. A., & Haworth, P. F. (2004). The effects of persecution on age of breeding and territory occupation in golden eagles in Scotland. *Biological Conservation*, *118*(2), 249-259.

[259] Herholdt, J. J. (1998). Survival threats and conservation management of raptors in the Kgalagadi Transfrontier Park. *Transactions of the Royal Society of South Africa*, *53*(2), 201-218.

[260] González, L. M., Margalida, A., Manosa, S., Sánchez, R., Oria, J., Molina, J. I., ... & Prada, L. (2007). Causes and spatio-temporal variations of non-natural mortality in the Vulnerable Spanish imperial eagle Aquila adalberti during a recovery period. *Oryx*, *41*(04), 495-502.

[261] Ibid.

[262] Hernández and Margalida, *Poison-related mortality effects in the endangered Egyptian vulture (Neophron percnopterus) population in Spain*.

[263] Otieno, P. O., Lalah, J. O., Virani, M., Jondiko, I. O., & Schramm, K. W. (2010). Carbofuran and its toxic metabolites provide forensic evidence for Furadan exposure in vultures (*Gyps* africanus) in Kenya. *Bulletin of Environmental Contamination and Toxicology*, *84*(5), 536-544; Houston, D. C. (1996). The effect of altered environments on vultures. *Raptors in Human Landscapes. Academic Press, London, United Kingdom*, 327-336.

[264] Ogada, D. L., Keesing, F., & Virani, M. Z. (2012). Dropping dead: causes and consequences of vulture population declines worldwide. *Annals of the New York Academy of Sciences*, *1249*(1), 57-71.

In Kenya, there have been significant declines of *Gyps* vultures in certain areas and one of the factors relating to the decline may be feeding on poisoned carcasses.[265] During the last ten years, there has been an increase in the use of poisons to kill generalist predators, such as lions and hyenas, often in retaliation for livestock predation in East Africa.[266]

Poisoning of carcasses and harvesting for traditional medicine have become major causes of vulture decline in Southern and Western Africa and have been reported in East Africa as well.[267]

Birds that do not feed on carrion may not be susceptible to poisoning targeted at the control of large predators.[268] For example, the eagle owl (*Bubo bubo*) is not a scavenging bird species and therefore, may not be likely to be affected by poison-baits as other raptors. However, particular poison-baits, such as carbofuran laced seed, may expose seed-eating birds. It is also one of several birds singled out by governments and hunters as a source of conflict with game interests.[269] An analysis of 1,576 birds admitted to wildlife rescue centres in Spain between 1989 and 2003, revealed human persecution was the main cause of death in 20 per cent of the birds; however, persecution by poisoning only occurred in 1.2 per cent of the dead birds, with hunting being the most common form of persecution.[270]

2.3. Grain crop areas

Pesticides have been used to target avian pest species in rice, and in some areas this practice continues, usually illegally. Organophosphates, such as monocrotophos and parathion, were used to control dickcissels (*Spiza Americana*), a small seed-eating bird, in Venezuela in rice and sorghum crops. Die-offs as large as 1,000 birds were documented.[271] In China, poison-baits used by farmers to protect wheat fields from nomad sheep may expose migratory birds.[272]

2.4. Game management areas

In some cases, game management areas can increase the likelihood of migratory birds' exposure to poison-baits. Game management focuses on ensuring there is a large potential harvest of game species, such as rabbits in Spain, is available for the hunting season. As a result, birds of prey and other predators may be targeted with poison-baits to limit any effects on game species.

In the United Kingdom, there is a human-wildlife conflict between the commercial management of red grouse (*Lagopus lagopus scotica*) for hunting and the conservation of legally protected raptors,

[265] Virani, M. Z., Kendall, C., Njoroge, P., & Thomsett, S. (2011). Major declines in the abundance of vultures and other scavenging raptors in and around the Masai Mara ecosystem, Kenya. *Biological Conservation*, *144*(2), 746-752.

[266] Kissui, *Livestock predation by lions, leopards, spotted hyenas, and their vulnerability to retaliatory killing in the Maasai steppe, Tanzania*.

[267] Thiollay, J. M. (2007). Raptor population decline in West Africa. *Ostrich*, *78*(2), 405-413.

[268] Herholdt, J. J., & Anderson, M. D. (2006). Observations on the population and breeding status of the African White-backed Vulture, the Black-chested Snake Eagle, and the Secretarybird in the Kgalagadi Transfrontier Park. *Ostrich-Journal of African Ornithology*, *77*(3-4), 127-135.

[269] Kenward, R. (2002). Management tools for reconciling bird hunting and biodiversity. Centre for Ecology and Hydrology.

[270] Martinez, J. A., Martinez, J. E., Manosa, S., Zuberogoitia, I., & Calvo, J. F. (2006). How to manage human-induced mortality in the Eagle Owl Bubo bubo. *Bird Conservation International*, *16*(03), 265-278.

[271] Basili, G. D., & Temple, S. A. (1999). Dickcissels and crop damage in Venezuela: defining the problem with ecological models. *Ecological applications*, *9*(2), 732-739; Parsons, K. C., Mineau, P., & Renfrew, R. B. (2010). Effects of pesticide use in rice fields on birds. *Waterbirds*, *33*(sp1), 193-218.

[272] Lu, J., Zhu, S., & Shan, K. (1998). Current situation and protection of cranes in Huanghe River Delta. *China Crane News*, *2*(2), 8-9 in Kanai, Y., Ueta, M., Germogenov, N., Nagendran, M., Mita, N., & Higuchi, H. (2002). Migration routes and important resting areas of Siberian cranes (Grus leucogeranus) between northeastern Siberia and China as revealed by satellite tracking. *Biological Conservation*, *106*(3), 339-346.

such as the hen harrier (*Circus cyaneus*).[273] The aim of grouse management is to maximise the number of grouse available for shooting in the autumn. Higher densities of grouse are needed for driven grouse hunting compared to other hunting techniques. [274] Red grouse fall prey to a number of generalist predators, including raptors.[275] Raptors are perceived to reduce grouse harvests and, traditionally, the killing of raptors was part of grouse management.[276] The illegal killing of raptors continues in many grouse management areas and threatens the UK populations of hen harriers, red kites (*Milvus milvus*), peregrine falcons (*Falco peregrinus*), and golden eagles (*Aquila chrysaetos*).[277]

An assessment of the illegal use of poisons in Scotland between 1981 and 2000 found a correlation between poison-baits and grouse management areas.[278] Indeed, the results from that study suggested the use of poison-baits is increasingly seen only in grouse management areas and not sheep-rearing areas. This may indicate that sheep farmers are less likely to use poison-baits to control predator birds in the UK.

Illegal killing was responsible for 55 per cent of the north Scotland red kites that were recovered dead (a total of 103 red kites) between 1989 and 2006, and where the cause of death could be established.[279] The majority were killed through primary poisoning, usually by poison-baits (carrion laced with poison). The red kite population in North Scotland is established in areas surrounded by active grouse management.[280] Red kites pose little threat to game management as they feed primarily on carrion, and many of the baits they consume are likely targeting other species, such as red foxes (*Vulpes vulpes*) or other raptors. However, the indiscriminate nature of poison-baits results in exposure of non-target species.

Legislation, changes in cultural attitudes and reductions in the prevalence of game hunting in some areas have resulted in several raptor populations making some recovery in the UK.[281] However, illegal persecution of raptors still occurs with effects on raptor distribution and abundance and is often associated with game hunting in the UK. [282]

[273] Redpath, S. M., Arroyo, B. E., Leckie, F. M., Bacon, P., Bayfield, N., Gutierrez, R. J., & Thirgood, S. J. (2004). Using decision modelling with stakeholders to reduce human–wildlife conflict: a raptor–grouse case study. *Conservation Biology*, *18*(2), 350-359.

[274] Sotherton, N., Tapper, S., & Smith, A. (2009). Hen harriers and red grouse: economic aspects of red grouse shooting and the implications for moorland conservation. *Journal of Applied Ecology*, *46*(5), 955-960.

[275] Thirgood, S. J., Redpath, S. M., Rothery, P., & Aebischer, N. J. (2000). Raptor predation and population limitation in red grouse. *Journal of Animal Ecology*, *69*(3), 504-516.

[276] Redpath et al., *Using decision modeling with stakeholders to reduce human–wildlife conflict: a raptor–grouse case study*.

[277] Etheridge, B., Summers, R. W., & Green, R. E. (1997). The effects of illegal killing and destruction of nests by humans on the population dynamics of the hen harrier Circus cyaneus in Scotland. *Journal of Applied Ecology*, 1081-1105; Watson, J. (2010). *The golden eagle*. Poyser; Evans, I. M., Summers, R. W., O'toole, Lorcan, Orr-Ewing, D. C., Evans, Richard, Snell, Nigel, & Smith, J. (1999). Evaluating the success of translocating Red Kites Milvus milvus to the UK. *Bird Study*, *46*(2), 129-144; Scottish Raptor Study Groups (1997). The illegal persecution of raptors in Scotland. *Scottish Birds* 19:65-85.

[278] Whitfield, D. P., McLeod, D. R., Watson, J., Fielding, A. H., & Haworth, P. F. (2003). The association of grouse moor in Scotland with the illegal use of poisons to control predators. *Biological Conservation*, *114*(2), 157-163. Also see Thirgood 2002.

[279] Smart, J., Amar, A., et al. (2010). Illegal killing slows population recovery of a re-introduced raptor of high conservation concern–The red kite Milvus milvus. *Biological Conservation*, *143*(5), 1278-1286.

[280] Carter, I., Cross, A. V., et al. (2003). Re-introduction and conservation of the red kite (Milvus milvus) in Britain: current threats and prospects for future range expansion. *Birds of prey in a changing environment. Scottish Natural Heritage, Edinburgh, UK*, 407-416.

[281] Watson, J. (2010). *The golden eagle*. Poyser.

[282] Ibid.

Small-game hunting plays an important economic role in central Spain and some game species are key food resources for threatened predators.[283] Small carnivores may reduce numbers of important game species. To alleviate predation pressure on game populations, management is often focused on predator control. Control methods can be non-selective, and so could potentially have a negative impact on non-target carnivore species.[284]

The use of poison to control predators increased dramatically in Spain after the irruption of the rabbit haemorrhagic disease that decimated Spanish rabbit populations (a key game species) in the early-1990s.[285] A study in Central Spain found carnivore species richness to be lower in areas managed for game (approx. 70 per cent of the country is managed for game[286]). This may suggest that low carnivore species richness is linked to small-game hunting activities, which may be a result of non-selective predator control practices. [287] The correlation between illegal poison use and migratory Egyptian vulture (*Neophron percnopterus*) territories in Spain may indicate that poisoning is one of the key factors in the decline of this species.[288]

Direct persecution of Bonelli eagles (*Aquila fasciata*) was the main cause of death of death in 26 per cent of 424 Bonelli eagles found dead or fatally injured between 1990 and 1998 in Spain (particularly in Levant and Northern Spain).[289] The cause of direct persecution may be related to game hunting and pigeon competition activities.

All scavenging bird species may be at risk of exposure to poison-baits because (1) game managers may target all birds of prey due to lack of knowledge of individual species' ecology; (2) the indiscriminate nature of poison-baits laced with insecticides or rodenticides can result in exposure of any scavenger bird species.

3. Exposure: harvesting for consumption

A wide variety of species are targeted for the traditional medicine trade, with approximately 350 bird species (such as birds of prey, egrets, storks, cranes)[290] used in traditional medicine in over 25 African countries.[291] The acquisition of vulture parts for traditional medicine has been documented in West and southern Africa.[292] However, in a study in Zimbabwe, the trade of the southern ground

[283] González, L. M., Margalida, A., et al. (2007). Causes and spatio-temporal variations of non-natural mortality in the Vulnerable Spanish imperial eagle Aquila adalberti during a recovery period. *Oryx*, *41*(04), 495-502.

[284] Virgós, E., & Travaini, A. (2005). Relationship between small-game hunting and carnivore diversity in central Spain. *Biodiversity & Conservation*, *14*(14), 3475-3486.

[285] Carrete, M., Grande, J. M., et al. (2007). Habitat, human pressure, and social behavior: partialling out factors affecting large-scale territory extinction in an endangered vulture. *Biological Conservation*, *136*(1), 143-154.

[286] Villafuerte R, Viñuela J, Blanco, JC. (1998) Extensive predator persecution caused by population crash in a game species: the case of red kites and rabbits in Spain. Biol Conserv 84:181–188

[287] Virgós and Travaini, *Relationship between small-game hunting and carnivore diversity in central Spain*.

[288] Grande, J. M., Serrano, D., et al. (2009). Survival in a long-lived territorial migrant: effects of life-history traits and ecological conditions in wintering and breeding areas. *Oikos*, *118*(4), 580-590.

[289] Real, Joan, Grande, J. M., et al. (2001). Causes of death in different areas for Bonelli's Eagle Hieraaetus fasciatus in Spain. *Bird Study*, *48*(2), 221-228.

[290] Nikolaus, G. The fetish culture in West Africa: an ancient tradition as a threat to endangered birdlife? *Tropical Vertebrates in a Changing World.*

[291] Williams, V. L., Cunningham, A. B., et al. (2013). Birds of a Feather: Quantitative Assessments of the Diversity and Levels of Threat to Birds Used in African Traditional Medicine. In *Animals in Traditional Folk Medicine* (pp. 383-420). Springer Berlin Heidelberg.

[292] Nikolaus, G. (2000). Bird exploitation for traditional medicine in Nigeria. *Malimbus*, *23*, 45-55.

hornbill (*Bucorvus cafer*) for traditional medicine was unlikely to have population-level effects as harvesting was rare and opportunistic.[293]

In addition to use in traditional medicines, vultures, and in particular the hooded vulture (*Necrosyrtes monachus*),[294] are hunted for food in West Africa.[295] Though it is difficult to ascertain population-level effects of persecution on individual species, it is thought to be a significant cause of mortality for some species and populations, including European bearded vultures (*Gypaetus barbatus*), Cape Griffon vulture (*Gyps coprotheres*) in South Africa, hooded vultures in West Africa, and large vultures in Nigeria.[296]

Poison-baits (laced with carbofuran) are used to target birds for human consumption in the Bunyala Rice Irrigation Scheme, located on a key migratory flyway in Kenya. In the case of harvesting, decoys (often live-captured birds of the same species) are used to attract birds to the poison-baits. Wetland birds, including storks, egrets and waders, are commonly poisoned with potential impacts on populations.[297]

During a 2009 study in the Bunyala Rice Scheme, over 3,000 birds suffered mortalities from carbofuran poison-baits (representing 37 per cent of the birds of the birds that visited the rice fields).[298] Of the 1,000 Palaearctic migrants that were present, 452 were poisoned. The most significantly affected migratory species were the black-tailed godwit (*Limosa limosa*) and wood sandpiper (*Tringa glareola*). The poison-baits are used seasonally during migration periods. A survey of local residents found that between 15-30 per cent preferred wild-caught bird meat to other sources of meat, citing that it is more nutritious, indicating that local demand is resulting in the practice of "pesticide hunting."

Very few incidents of deliberate poisoning using carbofuran have been reported in India, despite its availability as an agricultural insecticide.[299] However, incidents could go unnoticed, unreported or not investigated. In India, the poisoning surveillance system with respect to wildlife is not well formed. Poisoning of wildlife may occur for human consumption; for example, whistling teal (*Dendrocygna javanica*) were found poisoned for human consumption in Assam.[300] There are also documented cases of large predators, such as elephants and tigers, being poisoned by insecticides,

[293] Bruyns, R. K., Williams, V. L., & Cunningham, A. B. (2013). Finely Ground-Hornbill: The Sale of Bucorvus Cafer in a Traditional Medicine Market in Bulawayo, Zimbabwe. In *Animals in Traditional Folk Medicine* (pp. 475-486). Springer Berlin Heidelberg.

[294] Nikolaus, *Bird exploitation for traditional medicine in Nigeria*.

[295] Thiollay, J. (2006). The decline of raptors in West Africa: long-term assessment and the role of protected areas.

[296] Nikolaus, *Bird exploitation for traditional medicine in Nigeria*; Thiollay, *The decline of raptors in West Africa: long-term assessment and the role of protected areas*; Margalida, *Sources of variation in mortality of the Bearded vulture Gypaetus barbatus in Europe*.

[297] Odino, M. (2010). Measuring the conservation threat to birds in Kenya from deliberate pesticide poisoning.

[298] Ibid.

[299] Ragothaman, V., & Chirukandoth, S. Mitigating Human-Wildlife Conflict and Retaliatory Poisonings in India to Preserve Biodiversity and Maintain Sustainable Livelihoods. *Carbofuran and Wildlife Poisoning: Global Perspectives and Forensic Approaches*, 99-131.

[300] Talukdar, A. (2006) Deliberate poisoning of whistling teal (Dendrocygna javanica) for consumption in Bokakhat, Assam. IFAW-WTI Emergency Relief Network Digest, 2005-6, 53-4.

which may result in the secondary poisoning of scavenger bird species (which may also go unreported).[301]

In China, migratory birds are harvested using poisons in some regions (such as waterfowl, herons, spoonbills, egrets). In the Shanghai area the use of poisons is one of the top four methods used to harvest migratory birds.[302] The extent of poisoning in China and its effects on migratory bird populations is unknown.

A study suggested that deliberate poisoning of birds (often by rice laced with pesticides), particularly waterfowl, may occur in areas that practice Chinese medicine.[303] For example, wild ducks have been traditionally used in Chinese medicine in Korea.[304] Other species affected in Korea, include the tawny owl (*Strix aluco*), cranes (*Grus monachus*, *Grus japonensis* and *Grus vipio*), hill pigeon (*Columba rupestris*), and Oriental stork (*Ciconia boyciana*).

Where harvesting of migratory bird species occurs, poison-baits are often only one of the methods used. Other methods of harvesting, such as shooting with lead shot, can also result in poisoning of birds (see section on lead).

4. Toxicity

The pesticides most frequently involved in animal poisonings are insecticides and rodenticides.[305] The use of specific types of pesticides for poison-baits varies according to several factors, including, the type of agriculture in the region, the popular knowledge of the toxicity of a specific product, and its availability in the local market. For example, Carbofuran is often used for lacing baits in the United States because of the traditional knowledge within farming communities of its high acute toxicity to birds and mammals and because of its availability.[306]

The proportion of the active ingredient in the formulations available, greatly determines the risk of lethal or sub-lethal effects if exposure occurs.[307] However, the indiscriminate nature of many of the insecticide and rodenticide substances used increases the likelihood the poison-bait will be toxic to migratory birds (even if the baits were set for mammalian predators).

[301] Ragothaman, V., & Chirukandoth, S. Mitigating Human-Wildlife Conflict and Retaliatory Poisonings in India to Preserve Biodiversity and Maintain Sustainable Livelihoods. *Carbofuran and Wildlife Poisoning: Global Perspectives and Forensic Approaches*, 99-131.

[302] Ming, M., Jianjianz, L., Tang Chengjia, S. P., & Wei, H. (1998). The contribution of shorebirds to the catches of hunters in the Shanghai area, China during 1997-1998. *Sghilz*, *33*, 32-36.

[303] Kwon, Y. K., Wee, S. H., & Kim, J. H. (2004). Pesticide poisoning events in wild birds in Korea from 1998 to 2002. *Journal of wildlife diseases*, *40*(4), 737-740.

[304] Ibid.

[305] Martínez-Haro, M., Mateo, R., Guitart, R., Soler-Rodríguez, F., Pérez-López, M., María-Mojica, P., & García-Fernández, A. J. (2008). Relationship of the toxicity of pesticide formulations and their commercial restrictions with the frequency of animal poisonings. *Ecotoxicology and Environmental Safety*, *69*(3), 396-402.

[306] Vyas, N. B., Spann, J. W., Albers, E., & Patterson, D. (2002). Presticide-Laced Predator Baits: Consideratons for Prosecution and Sentencing. *Envtl. Law.*, 9, 589.

[307] Martínez-Haro et al., *Relationship of the toxicity of pesticide formulations and their commercial restrictions with the frequency of animal poisonings*.

The likelihood of exposure to poison-baits laced with carbofuran is increased because a study found that direct and secondary poisoning can occur for at least 60 days post-placement of the bait, and may persist longer in cold weather conditions.[308]

Figure 2: Examples of commonly used substances in poison-baits

Carbofuran has been the most frequent cause of poisoning in wild predatory birds in the Czech Republic in the past 10 years.[309] Illegal use of carbofuran used to be a widespread practice in vermin (foxes, martens, etc.) control. Because of its high toxicity to birds, the most frequent reported mortalities were birds of prey.

There is widespread involvement of carbofuran with bird of prey poisoning incidents throughout Kenya, Uganda and South Africa.[310] Since 2003, in Kenya, poisoning of birds with carbofuran has been a problem. Carbofuran, sold under the trade name Furadan, is legally sold throughout Kenya, as an agricultural pesticide and its use as a cheap, effective poison is well-known among farmers. [311]

In Italy, the most frequently detected substances are carbamates and organophosphates (48 per cent) and anticoagulant rodenticides (18 per cent).[312] Aldicarb and carbofuran (carbamates) were the most commonly used substances (68 per cent) in poisoning incidents in southern Spain between 1990 and 2005.[313]

Carbofuran was the most frequently identified pesticide in 80 recovered golden eagles (*Aquila chrysaetus*) and bald eagles (*Haliaeetus leucocephalus*) in Western Canada between 1993 and 2002.[314]

Strychnine and endrin are also commonly used for predator control despite restrictions on their use in many countries in Europe and North America.[315]

5. Population-level effects

The use of poison-baits may dramatically affect populations of many species. For example, poison baiting in southern Spain has been linked with severe raptor declines, such as the red kite.[316] Particularly, the black vulture (*Aegypius monachus*), Egyptian vulture (*Neophron percnopterus*), bearded vulture, and Spanish imperial eagle (*Aquila adalberti*) have been severely affected by

[308] Allen, G. T., Veatch, J. K., Stroud, R. K., Vendel, C. G., Poppenga, R. H., Thompson, L., ... & Braselton, W. E. (1996). Winter poisoning of coyotes and raptors with Furadan-laced carcass baits. *Journal of wildlife diseases*, *32*(2), 385-389.

[309] Modrá, H., & Svobodová, Z. (2009). Incidence of animal poisoning cases in the Czech Republic: current situation. *Interdisciplinary toxicology*, *2*(2), 48-51.

[310] Virani, M. Z., Kendall, C., Njoroge, P., & Thomsett, S. (2011). Major declines in the abundance of vultures and other scavenging raptors in and around the Masai Mara ecosystem, Kenya. *Biological Conservation*, *144*(2), 746-752; Otieno, P. O., Lalah, J. O., Virani, M., Jondiko, I. O., & Schramm, K. W. (2010). Carbofuran and its toxic metabolites provide forensic evidence for Furadan exposure in vultures (*Gyps* africanus) in Kenya. *Bulletin of Environmental Contamination and Toxicology*, *84*(5), 536-544.

[311] Otieno, P. O., Lalah, J. O., Virani, M., Jondiko, I. O., & Schramm, K. W. (2010). Carbofuran and its toxic metabolites provide forensic evidence for Furadan exposure in vultures (*Gyps* africanus) in Kenya. *Bulletin of Environmental Contamination and Toxicology*, *84*(5), 536-544.

[312] Giorgi, M., & Mengozzi, G. (2011). Malicious animal intoxications: poisoned baits. *Veterinarni Medicina*, *56*(4), 173-179.

[313] Márquez, C, Vargas, J. M., Villafuerte, R., & Fa, J. E. (2013). Risk mapping of illegal poisoning of avian and mammalian predators. *The Journal of Wildlife Management*, *77*(1), 75-83.

[314] Wobeser, G., Bollinger, T., Leighton, F. A., Blakley, B., & Mineau, P. (2004). Secondary poisoning of eagles following intentional poisoning of coyotes with anticholinesterase pesticides in western Canada. *Journal of Wildlife Diseases*, *40*(2), 163-172.

[315] Martínez-Haro, M., Mateo, R., Guitart, R., Soler-Rodríguez, F., Pérez-López, M., María-Mojica, P., & García-Fernández, A. J. (2008). Relationship of the toxicity of pesticide formulations and their commercial restrictions with the frequency of animal poisonings. *Ecotoxicology and Environmental Safety*, *69*(3), 396-402.

[316] Márquez et al., *Risk mapping of illegal poisoning of avian and mammalian predators*.

poisoning.[317] Indeed, vultures and red kites have been suggested as having the highest propensity of being poisoned among any wildlife.[318]

In a study of red kites in Spain, modelling indicated mortalities caused by illegal poisoning suppressed the population by 20 per cent. However, despite this, the population was likely to increase slowly, maybe as a result of supplementary feeding. In a study of radio-tracked red kites, 53 per cent of the tagged birds died as a result of illegal poisoning. The effect of this mortality on the population was unable to be determined.[319]

Modelling has indicated an estimated 3-5 per cent annual adult mortality from persecution in golden eagles, and has suggested the population would likely grow in the absence of persecution.[320] In parts of Scotland, it has been indicated that persecution may reduce breeding productivity of golden eagles by up to 20 per cent.[321] It has also been suggested that persecution is most likely to limit raptor numbers in areas where game is hunted.[322]

The number of raptors, primarily scavenger species, declined more than 40 per cent per year over a three-year period in central Kenya.[323] During the study, the overall population of large wild herbivores showed little change, which may suggest that food shortages were not the cause of the decline. Possible causes of raptor decline include the consumption of poison-baits, which are placed by pastoralists to kill large predators that attack livestock. Further research was recommended to determine whether the declines are having a population-level effect.[324] Whether poisoning explains the rapid decline in abundance of scavenging birds is yet to be determined.

Figure 3: Knowledge gaps in the literature on effects of poison-baits on migratory birds

- Likelihood of exposure to poison-baits in species other than birds of prey
- Occurrence of harvesting using poison-baits outside of Africa and China?
- Extent of the use of poison-baits compared to other methods of predator control, such as trapping and shooting
- Frequency of the use of poison-baits in game management areas versus agricultural areas?
- Effects of poison-baits on migratory birds compared to other types of poisoning, such as agricultural pesticides and lead ammunition/shot.

[317] Hernández, M., & Margalida, A. (2008). Pesticide abuse in Europe: effects on the Cinereous vulture (Aegypius monachus) population in Spain. *Ecotoxicology*, *17*(4), 264-272; Hernández and Margalida, *Poison-related mortality effects in the endangered Egyptian vulture (Neophron percnopterus) population in Spain*.; Mariano González, L., Oria, J., Sánchez, R., Margalida, A., Aranda, A., Prada, L., ... & Ignacio Molina, J. (2008). Status and habitat changes in the endangered Spanish Imperial Eagle Aquila adalberti population during 1974–2004: implications for its recovery. *Bird Conservation International*, *18*(03), 242-259.

[318] Márquez, C. Vargas, J. M., Villafuerte, R., & Fa, J. E. (2012). Understanding the propensity of wild predators to illegal poison baiting. *Animal Conservation*.

[319] Tavecchia, G., Adrover, J., Navarro, A. M., & Pradel, R. (2012). Modelling mortality causes in longitudinal data in the presence of tag loss: application to raptor poisoning and electrocution. *Journal of Applied Ecology*, *49*(1), 297-305.

[320] Whitfield, D. P., Fielding, A. H., McLeod, D. R. A., & Haworth, P. F. (2004). The effects of persecution on age of breeding and territory occupation in golden eagles in Scotland. *Biological Conservation*, *118*(2), 249-259.

[321] Scottish Raptor Study Groups (1997). The illegal persecution of raptors in Scotland. *Scottish Birds*, 19, 65-85 in Whitfield, D. P., Fielding, A. H., McLeod, D. R. A., & Haworth, P. F. (2004). The effects of persecution on age of breeding and territory occupation in golden eagles in Scotland. *Biological Conservation*, *118*(2), 249-259.

[322] Ibid.

[323] Ogada, D. L., & Keesing, F. (2010). Decline of raptors over a three-year period in Laikipia, Central Kenya. *Journal of Raptor Research*, *44*(2), 129-135.

[324] Ibid.

6. Conclusion

Predator control using poison-baits occurs on a global scale, particularly in areas with game management and livestock farming. Predator and scavenger bird species are at risk of poisoning from poison-baits targeting them directly, and also from baits targeting mammalian species (with birds becoming by-catch through secondary poisoning). The effect on species other than birds of prey is largely unknown and further research is needed in this area.

The risk of poisoning from harvesting for human consumption and traditional medicine appears to be much more isolated. Using poisons to harvest migratory bird species for consumption and/or traditional medicine may be limited to particular areas in Africa and Asia. Although, the demand for particular bird species for human medicine may cause the harvesting of these species in other parts of the world.

Due to the indiscriminate nature of many of the substances used in poison-baits, any birds are at risk of poisoning if they come into contact with poison-baits. The most common substances are rodenticides and insecticides, usually those that are known to farmers in the area as highly toxic. Carbofuran appears to be used in poison-baits in many areas around the world.

Many birds of prey populations have been affected from illegal poison-baits, particularly vultures. This suggests that further work needs to be developed to understand why poison-baits continue to be used and craft effective solutions.

Effects of veterinary treatment of livestock using pharmaceuticals

1. Introduction

Certain toxic veterinary pharmaceuticals pose a risk to birds when used to treat domestic livestock whose carcasses may become food sources for scavenging birds. Non-steroidal anti-inflammatory drugs (NSAIDs) are used to treat domestic livestock for inflammation and pain relief and are the most significant pharmaceutical of risk to birds today. Diclofenac, a previously popular NSAID for veterinary care of cattle in India, Pakistan and Nepal, is toxic to a number of vulture species and has been licensed for use in Spain and other countries in Europe. It has resulted in the poisoning of scavenging vultures throughout India, Pakistan and Nepal by contaminating domestic livestock carcasses traditionally fed on by vultures. Prior to the ban of diclofenac in these regions, it was prevalent in livestock carcasses and caused substantial population declines of three species of *Gyps* vultures in South Asia.[325]

Population declines of *Gyps* vultures were first noticed in India in the early-to-mid 1990s and the cause of the decline was discovered in 2003. Observed rates of population decrease are among the highest recorded for any bird species, leading to total declines in excess of 99.9 per cent for the Oriental white-backed vulture (*Gyps bengalensis*) in India between 1992 and 2007. Long-billed (*Gyps indicus*) and slender-billed (*Gyps tenuirostris*) vultures declined by 96.8 per cent over the same period.[326] Although these birds are not migratory, there are other similar scavenging bird species that may be at risk. The drug has now also been shown to be toxic to Aquila eagles, of which there are 14 species distributed across Asia, Africa, Australia, Europe and North America, well beyond the more restricted distribution of Gyps vultures.

While other causes of mortality, such as deliberate poisoning and changes in food availability are related to vulture declines in Europe and Africa, there is no evidence that such factors play a key role in South Asia.[327] The main contributory factor causing declines in many vulture species is the use of the veterinary drug diclofenac on domestic livestock that are likely to die before the drug is metabolised and the availability of these carcasses for vultures to feed on (ie, left in the open after death). After ingestion of livestock carcasses treated with diclofenac near to their death, birds die as a result of visceral gout that subsequently causes kidney failure. Death of the bird usually occurs

[325] Shultz, S., Baral, H. S., Charman, S., Cunningham, A. A., Das, D., Ghalsasi, G. R., ... & Prakash, V. (2004). Diclofenac poisoning is widespread in declining vulture populations across the Indian subcontinent. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, *271*(Suppl 6), S458-S460.

[326] Prakash, V., Green, R. E., Pain, D. J., Ranade, S. P., Saravanan, S., Prakash, N., ... & Cunningham, A. A. (2007). Recent changes in populations of resident *Gyps* vultures in India. *J. Bombay Nat. Hist. Soc*, *104*(2), 127-133.

[327] Green, R. E., Newton, I. A. N., Shultz, S., Cunningham, A. A., Gilbert, M., Pain, D. J., & Prakash, V. (2004). Diclofenac poisoning as a cause of vulture population declines across the Indian subcontinent. *Journal of Applied Ecology*, *41*(5), 793-800.

within a few days of exposure.[328] Small quantities of diclofenac may kill an individual or group of vultures.

This section evaluates the population effects of diclofenac on migratory birds by assessing the (1) likelihood of exposure to diclofenac; and (2) toxicity of diclofenac to migratory birds, both of which are discussed below. It also gives a preliminary review of other NSAIDs that may be toxic to birds.

2. Likelihood of exposure to diclofenac

Since the early-to-mid 1990s, diclofenac has been commonly used to treat pain and inflammation in livestock in India, Pakistan and Nepal. Dead vultures contaminated with diclofenac residues have been recovered across Pakistan, India and Nepal, and large-scale surveys of domesticated ungulate carcasses (the principal food source of vultures in Asia) across India indicate that 10-11 per cent of carcasses are contaminated with diclofenac.[329]

While diclofenac had been in use in India since about 1990, veterinary use of diclofenac in Pakistan did not begin until 1998.[330] Differences in the rate of decline of *Gyps* species between regions on the Indian subcontinent may be due to differences in diclofenac availability and use.[331] Surveys in Myanmar in late 2006 and early 2007 found no evidence that diclofenac was being used in livestock[332] and it is not in use in Cambodia.[333]

The likelihood of exposure to diclofenac and other veterinary pharmaceuticals used to treat domestic livestock may be higher in South Asia because of the unique situation of large numbers of livestock being left in the open after death.[334]

2.1. Diet of domestic ungulates increases likelihood of exposure

Wild ungulates are not a large part of the diet of *Gyps* vultures in Nepal and South Asia. In South Asia, *Gyps* vultures' diet consists mainly of domestic livestock, which increases their likelihood of exposure.

The average diclofenac concentration in livestock in India was sufficient to kill more than 10 per cent of vultures feeding from the carcass within a day or two of treatment.[335] Population modelling shows that just 0.1–0.8 per cent of carcasses need to contain lethal levels of diclofenac to have caused the observed decline in vulture numbers.[336] Therefore, very few carcasses need to be contaminated to

[328] Ibid.. *Journal of Applied Ecology*, *41*(5), 793-800.
[329] Chaudhary, A., Subedi, T.R., Giri, J.B., Baral, H.S., Bidari, B., Subedi, H., … & Cuthbert, R.J. (2012). Population trends of Critically Endangered *Gyps* vultures in the lowlands of Nepal. *Bird Conservation International*, *22*(03), 270-278.
[330] Chaudhary, M.J.I., Ogada, D.L., Malik, R.N., Virani, M.Z., & Giovanni, M.D. (2012). First evidence that populations of the critically endangered Long-billed Vulture Gyps indicus in Pakistan have increased following the ban of the toxic veterinary drug diclofenac in south Asia. *Bird Conservation International*, 22(4), 389.
[331] Harris, R. J. (2013). The conservation of Accipitridae vultures of Nepal: a review. *Journal of Threatened Taxa*, *5*(2), 3603-3619.
[332] Eames, J. C. 2007. Mega transect counts vultures across Myanmar. *The Babbler: BirdLife in Indochina*, 30.
[333] Mahood in litt. 2012.
[334] Pain, D. J., Bowden, C. G., Cunningham, A. A., Cuthbert, R., Das, D., Gilbert, M., … & Green, R. E. (2008). The race to prevent the extinction of South Asian vultures. *Bird Conservation International,*18, S30-48.
[335] Green, R. E., Taggart, M. A., Das, D., Pain, D. J., Sashi Kumar, C., Cunningham, A. A., & Cuthbert, R. (2006). Collapse of Asian vulture populations: risk of mortality from residues of the veterinary drug diclofenac in carcasses of treated cattle. *Journal of Applied Ecology*, *43*(5), 949-956.
[336] Green et al., *Diclofenac poisoning as a cause of vulture population declines across the Indian subcontinent*.

result in a population decline in vultures. This may also be partly related to the tendency of vultures to feed in flocks.

NSAIDs have a short half-life in ungulate tissues and therefore, residues are unlikely to persist in tissues after treatment for long periods of time. For example, in cattle, a standard dose of diclofenac declines to undetectable levels within a week.[337] A similar pattern is likely in water buffalo.

One study found that 10.1 per cent of domestic ungulate carcasses in India from a sample size of 1,848 had detectable concentrations of diclofenac. Diclofenac was found in cattle, water buffalo, goats and horses, but not sheep.[338]

2.2. With population declines of vultures, the number of species exposed may increase

The collapse in numbers of *Gyps* vultures across Asia now means that other scavenging birds are increasingly exposed to contaminated carcasses.[339] Whether diclofenac is affecting them is unknown, although other vultures in India (in addition to *Gyps* species) are also in rapid decline.

Excluding *Gyps* vultures, raptors and other scavenging bird species observed on carcasses include cinereous vulture (*Aegypius monachus*), Egyptian vulture (*Neophron percnopterus*), red-headed vulture (*Sarcogyps calvus*), steppe eagle (*Aquila nipalensis*), black kite (*Milvus migrans*), cattle egret (*Bubulcus ibis*), house crow (*Corvus splendens*), jungle crow/large-billed crow (*Corvus macrorhynchos*) and common mynah (*Acridotheres tristis*). Two of these species (Egyptian vulture and red-headed vulture) have recently undergone rapid population declines, possibly as a result of diclofenac poisoning.[340]

Other scavenging species within the region that may also potentially come into contact with contaminated carcasses include greater and lesser adjutants (*Leptoptilos dubius* and *Leptoptilos javanicus*), which are both globally threatened. The toxicity of NSAIDs to susceptible birds is discussed below.

3. Toxicity of diclofenac to birds

3.1. Species affected due to their physiology

To date, diclofenac has proven to be toxic in at least six of the eight *Gyps* vulture species and the domestic chicken (see Table 1).[341] Some studies suggest that diclofenac is likely to be toxic to all

[337] Green et al., *Collapse of Asian vulture populations: risk of mortality from residues of the veterinary drug diclofenac in carcasses of treated cattle*.

[338] Taggart, M. A., Senacha, K. R., Green, R. E., Jhala, Y. V., Raghavan, B., Rahmani, A. R., ... & Meharg, A. A. (2007). Diclofenac residues in carcasses of domestic ungulates available to vultures in India. *Environment international*, *33*(6), 759-765.

[339] Houston, D. C. (1983). The adaptive radiation of the griffon vultures. *Vulture biology and management*, 135-152.

[340] Cuthbert, R., Green, R. E., Ranade, S., Saravanan, S., Pain, D. J., Prakash, V., & Cunningham, A. A. (2006). Rapid population declines of Egyptian vulture (*Neophron percnopterus*) and red-headed vulture (*SarcoGyps calvus*) in India. *Animal Conservation*, *9*(3), 349-354.

[341] Oaks, J. L., Gilbert, M., Virani, M. Z., Watson, R. T., Meteyer, C. U., Rideout, B. A. & Khan, A. A. (2004). Diclofenac residues as the cause of vulture population decline in Pakistan. *Nature*, *427*(6975), 630-633; Swan, G., Naidoo, V., Cuthbert, R., Green, R. E., Pain, D. J., Swarup, D., ... & Wolter, K. (2006). Removing the threat of diclofenac to critically endangered Asian vultures. *PLoS Biology*, *4*(3), e66; Naidoo, V., Duncan, N., Bekker, L., & Swan, G. (2007). Validating the

eight *Gyps* vulture species.[342] However, information on the toxicity of diclofenac to the remaining *Gyps* vultures is lacking.[343]

Migratory species of the *Gyps* vultures include the Eurasian griffon vulture (*Gyps fulvus*) and Himalayan vulture (*Gyps himalayensis*). It is unknown whether the migratory Eurasian griffon vulture, which winters in India, is affected by diclofenac. The population of Eurasian griffon vultures shows consistent growth in the past 11 years at Jorbeer, India,[344] but they may be at risk of exposure because they feed almost exclusively on livestock carcasses. Diclofenac is toxic to the Himalayan vulture[345] and because it winters in areas of India, Pakistan and Nepal, the population levels of these species should be monitored.[346]

One study found that the Cape Griffon vulture (*Gyps coprotheres*) in the southern region of Africa is as sensitive to diclofenac as the *Gyps* vulture species that have been devastated on the Indian subcontinent.[347] In South Africa, the Cape Griffon vulture is almost wholly dependent on "vulture restaurants" which are often stocked with dead livestock from commercial farms. It is possible these animals could have been treated with NSAIDs, but no effects are documented as diclofenac is not licensed for use in South Africa. The likelihood of exposure may be increased because of the Cape Griffon vulture's wide foraging range, with birds routinely crossing borders in the Southern Africa region.[348] The use of diclofenac in surrounding countries is unknown.

The promotion of diclofenac on the African continent could pose a risk to vultures in this region, including the African white-backed vulture (*Gyps africanus*) and the endangered Cape Griffon vulture due to these species' sensitivity to diclofenac. Although, exposure levels may be different in Africa, eg, removal of cattle carcasses from open areas and variation in vulture diet.

3.2. Toxicity to species other than Gyps vultures

There is a risk that diclofenac is toxic to a wide variety of birds as it appears that toxicity to diclofenac is genus specific, not just species specific (see Table 1).

Diclofenac has now also been shown to be toxic to Aquila eagles, of which there are 14 species distributed across Asia, Africa, Australia, Europe and North America, well beyond the more restricted distribution of Gyps vultures.[349]

domestic fowl as a model to investigate the pathophysiology of diclofenac in *Gyps* vultures. *Environmental toxicology and pharmacology*, *24*(3), 260-266.

[342] Swan, G. E., Cuthbert, R., Quevedo, M., Green, R. E., Pain, D. J., Bartels, P., ... & Wolter, K. (2006). Toxicity of diclofenac to *Gyps* vultures. *Biology Letters*, *2*(2), 279-282.

[343] Das, D., Cuthbert, R.J., Jakati, R., & Prakash, V. (2011). Short communication Diclofenac is toxic to the Himalayan Vulture *Gyps* himalayensis. *Bird Conservation International*, *21*, 72-75.

[344] Khatri, P. C. (2012). Diclofenac (NSAID) is not Infectious for Eurasian Griffon Vulture (Gyps fulvus): A Study at Jorbeer, Bikaner, *International Journal of Food, Agriculture and Veterinary Sciences* 2(3), 44-49.

[345] Das, et al., *Short communication Diclofenac is toxic to the Himalayan Vulture Gyps himalayensis*.

[346] Swan et al., *Toxicity of diclofenac to Gyps vultures*.

[347] Naidoo, V., Wolter, K., Cuthbert, R., & Duncan, N. (2009). Veterinary diclofenac threatens Africa's endangered vulture species. *Regulatory toxicology and Pharmacology*, *53*(3), 205-208.

[348] Bamford, A. J., Diekmann, M., Monadjem, A., & Mendelsohn, J. (2007). Ranging behaviour of Cape Vultures *Gyps* coprotheres from an endangered population in Namibia. *Bird Conservation International*, *17*(4), 331.

[349] Sharma A.K., Saini M., Singh S.D., Prakash V., Das A., Bharathi Dasan R., Pandey S., Bohara D.L., Galligan T.H., Green R. E., Knopp D., Cuthbert R.J. (2013) Diclofenac is toxic to a non-*Gyps* vulture and an Aquila eagle: increasing the diversity of raptors under threat of NSAID misuse.

Lammergeier/Bearded vultures and Egyptian vultures are now also rapidly declining but there is no direct evidence that diclofenac is the cause. However, the geographic extent and rate of decline is similar to the *Gyps* populations, which could suggest diclofenac is the cause.[350]

Some scavenging species appear to tolerate high levels of diclofenac, such as the Turkey Vulture, a New World species unrelated to Old World *Gyps* vultures.[351] No cases of mortality (even at very high doses) have been reported for the turkey vulture (*Cathartes aura*) or pied crow (*Corvus albus*).[352] This supports the hypothesis that toxicity is related to species or genus specific metabolism.

Among the raptor species present in India, those most likely to feed on diclofenac-contaminated carcasses are Eurasian griffon vulture (*Gyps fulvus*), Himalayan griffon vulture (*Gyps himalayensis*), cinereous vulture (*Aegypius monachus*), Egyptian vulture (*Neophron percnopterus*), red-headed vulture (*Sarcogyps calvus*), steppe eagle (*Aquila nipalensis*) and black kite (*Milvus migrans*).[353] Populations of the Egyptian vulture and the red-headed vulture in India have declined markedly and rapidly, but probably with a later onset than *Gyps* vultures in the same region. It is unknown whether the declines in these two species are related to diclofenac poisoning, but the geographic extent and rate of declines is very similar to the declines in the *Gyps* vultures.[354]

Table 1: Toxicity of diclofenac to raptors

Species	Diclofenac toxic to birds?	IUCN Status	Range where affected	Migratory?
South Asia				
Oriental white-backed vulture (*Gyps bengalensis*)	✔	Critical	Pakistan, India	Resident
Long-billed vulture (*Gyps indicus)*	✔	Critical	Pakistan, India	Resident
Slender-billed vulture (*Gyps tenuirostris*)	✔	Critical	India, Nepal and Bangladesh	Resident
Eurasian griffon vulture (*Gyps fulvus*)	✔	Least concern	Not applicable	✔
Himalayan griffon vulture (*Gyps himalayensis*)	✔	In decline	Nepal	✔
Red-headed vulture (*Sarcogyps calvus*)	Unknown	Critically endangered	India, Nepal	Resident
Cinereous vulture (*Aegypius monachus*)	✔	Near threatened	India. No established link to diclofenac.	Mostly resident
Steppe eagle (*Aquila nipalensis*)	✔	Least concern	Not applicable	✔
Black kite (*Milvus migrans*)	Unknown	Least concern	Not applicable	✔
Bearded vulture	Unknown	In decline	Indian subcontinent	Resident

[350] Cuthbert et al., *Rapid population declines of Egyptian vulture (Neophron percnopterus) and red-headed vulture (SarcoGyps calvus) in India*.

[351] Rattner, B. A., Whitehead, M. A., Gasper, G., Meteyer, C. U., Link, W. A., Taggart, M. A., ... & Pain, D. J. (2008). Apparent tolerance of turkey vultures (Cathartes aura) to the non-steroidal anti-inflammatory drug diclofenac. *Environmental Toxicology and Chemistry*, *27*(11), 2341-2345.

[352] Rattner et al., *Apparent tolerance of turkey vultures (Cathartes aura) to the non-steroidal anti-inflammatory drug diclofenac*.

[353] Cuthbert et al., *Rapid population declines of Egyptian vulture (Neophron percnopterus) and red-headed vulture (SarcoGyps calvus) in India.*

[354] Ibid.

(*Gypaetus barbatus*)				
Egyptian vulture (*Neophron percnopterus*)	Unknown	Endangered	South Asia, Northern Africa, Middle East, and South-Western Europe. No established link to diclofenac.	✔
Africa				
African white-backed vulture (*Gyps africanus*)	✔	Endangered	No established link to diclofenac.	Mostly resident
Cape Griffon vulture (*Gyps coprotheres*)	✔	Vulnerable	Southern Africa. No established link to diclofenac	Resident

5. Other NSAIDs of risk to migratory birds

There are a number of NSAIDs that may be toxic to scavenging bird species, including ketoprofen, aceclofenac, carprofen, flunixin and acetaminophen, which are discussed below. The NSAIDs, other than diclofenac, most commonly found in carcasses available to vultures are meloxicam, ibuprofen and ketoprofen.[355] Meloxicam, which is also discussed below, is of low toxicity to *Gyps* vultures and some other raptor species. The effect of ibuprofen on scavenging birds is unknown.

Ketoprofen was found to be toxic to *Gyps* vultures, and perhaps a wider range of avian species.[356] At least two species of *Gyps* vultures are likely to experience toxic effects from ketoprofen at doses that birds could encounter in the wild.[357] Mortality in male eider ducks has also been associated with ketoprofen.[358]

The presence of ketoprofen was found in 0.5 per cent of carcasses available to vultures within India.[359] While the residue prevalence of ketoprofen is significantly lower than diclofenac, population modelling has demonstrated that just 0.13-0.75 per cent of carcasses need contain a lethal dose to cause population declines of 48 per cent per year in the Oriental white-backed vulture.[360] The use of ketoprofen as a veterinary NSAID for treating livestock in Southern Africa and Europe also raises concerns over the potential impact of this drug on vulture populations in these regions.

Aceclofenac, which is considered by veterinary practitioners in India as a cost-effective and clinically effective substitute for diclofenac, is converted to diclofenac and its metabolites found in all mammal species tested to date raise concern that these same pathways will be followed in livestock. If this is the case, then the use of aceclofenac as a veterinary NSAID for treating livestock in South

[355] Taggart, M. A., Senacha, K. R., Green, R. E., Cuthbert, R., Jhala, Y. V., Meharg, A. A., ... & Pain, D. J. (2009). Analysis of nine NSAIDs in ungulate tissues available to critically endangered vultures in India. *Environmental science & technology*, *43*(12), 4561-4566.

[356] Naidoo, V., Venter, L., Wolter, K., Taggart, M., & Cuthbert, R. (2010). The toxicokinetics of ketoprofen in *Gyps* coprotheres: toxicity due to zero-order metabolism. *Archives of toxicology*, *84*(10), 761-766.

[357] Naidoo, V., Wolter, K., Cromarty, D., Diekmann, M., Duncan, N., Meharg, A. A., ... & Cuthbert, R. (2010). Toxicity of non-steroidal anti-inflammatory drugs to *Gyps* vultures: a new threat from ketoprofen. *Biology Letters*, *6*(3), 339-341.

[358] Mulcahy, D. M., Tuomi, P., & Larsen, R. S. (2003). Differential mortality of male spectacled eiders (Somateria fischeri) and king eiders (Somateria spectabilis) subsequent to anesthesia with propofol, bupivacaine, and ketoprofen. *Journal of avian medicine and surgery*, *17*(3), 117-123.

[359] Taggart et al., *Analysis of nine NSAIDs in ungulate tissues available to critically endangered vultures in India*.

[360] Green et al., *Diclofenac poisoning as a cause of vulture population declines across the Indian subcontinent*.

Asia, or any countries with *Gyps* vultures, may pose a risk to vultures scavenging on domestic ungulate carcasses treated with aceclofenac prior to death.[361]

Carprofen and flunixin appear to carry a high risk of renal damage in birds. One study found mortality associated with the use of carprofen and flunixin in 30 per cent of cases of over 870 birds from 79 species.[362] Both carprofen and flunixin are used to treat livestock in Europe, although not yet in South Asia. Livestock dying shortly after treatment with carprofen and flunixin may contain sufficient residues to pose a threat to scavenging birds. One study indicated that a vulture consuming a 1kg meal from an animal that died shortly after a veterinary course of these drugs, could be exposed to doses close to or within, the range of doses that caused mortality of birds after clinical treatment.

Acetaminophen, a NSAID recently introduced to veterinary use for the treatment of domestic animals, did not have toxic effects in a study on chickens.[363] The same study found toxic effects from diclofenac. Therefore, acetaminophen may be an option to further explore for the treatment of livestock without risk of toxic effects on birds.

Meloxicam is of low toxicity to *Gyps* vultures and a wide range of other raptors and scavenging birds. Meloxicam is out of patent, licensed for veterinary use in India, and considered a very effective NSAID to treat a variety of livestock ailments. The treatment of meloxicam on 60 different bird species with a sample size of 739 birds resulted in no mortalities.[364] Meloxicam is now being used in South Asia, but remains more expensive than diclofenac was previously.

One study found exposure to meloxicam at the maximum levels likely to be found in the wild safe for the Oriental white-backed vulture, long-billed vulture and a range of other scavenging birds in India (Egyptian vulture, cattle egret *Bubulcus ibis*, house crow *Corvus splendens*, large-billed crow and common mynah *Acridotheres tristis*).[365]

The results of one study show that certain NSAIDs are toxic to raptors, storks, cranes and owls. Mortality was found following treatment with diclofenac, carprofen, flunixin, ibuprofen and phenylbutazone.[366] Of particular concern is the mortality of a Marabou stork (*Leptoptilos crumeniferus*) following treatment with flunixin. Storks and New World vultures are phylogenetically closely related.[367] Therefore, the veterinary use of NSAIDs in regions of the Americas with New World vulture populations needs further research.

[361] Sharma, P. (2012). Aceclofenac as a Potential Threat to Critically Endangered Vultures in India: A Review. *Journal of Raptor Research*, *46*(3), 314-318.

[362] Cuthbert, R., Parry-Jones, J., Green, R. E., & Pain, D. J. (2007). NSAIDs and scavenging birds: potential impacts beyond Asia's critically endangered vultures. *Biology letters*, *3*(1), 91-94.

[363] Jayakumar, K., Mohan, K., Swamy, H. N., Shridhar, N. B., & Bayer, M. D. (2010). Study of nephrotoxic potential of acetaminophen in birds. *Toxicology international*, *17*(2), 86.

[364] Cuthbert et al., *NSAIDs and scavenging birds: potential impacts beyond Asia's critically endangered vultures*.

[365] Swarup, D., Patra, R. C., et al. (2007). Safety of meloxicam to critically endangered *Gyps* vultures and other scavenging birds in India. *Animal Conservation*, *10*(2), 192-198.

[366] Cuthbert et al., *NSAIDs and scavenging birds: potential impacts beyond Asia's critically endangered vultures*.

[367] Sibley, C. G., Ahlquist, J. E., & Monroe Jr, B. L. (1988). A classification of the living birds of the world based on DNA-DNA hybridization studies. *The Auk*, 409-423.

This could suggest that a common mechanism of toxicity is responsible for NSAID-related mortality across different orders of birds.[368] Although, whether the toxicity of diclofenac to vultures is caused by diclofenac itself or by its metabolites or a combination of both is unknown.

A number of NSAIDs have not yet been tested as safe for vultures. Many are widespread within the range of resident and migratory accipitridae vultures. For example, the toxicity to vultures is unknown for metamizole, phenylbutazone, ibuprofen, and naproxen. To date, the only NSAID that has been tested and proven safe for vultures in meloxicam.[369] Initial research on poultry suggests that nimesulide may be safe for vultures, but it needs further safety testing.

Figure 1: Knowledge gaps in literature on effects of NSAIDs on migratory birds

- How is vulture diet likely to change exposure rates, eg, proportion of diet of domestic ungulate carcasses versus wild animal carcasses?
- Effectiveness and cost of veterinary diclofenac versus alternative NSAIDs?
- Likelihood of exposure to NSAIDs in areas outside India, Nepal and Pakistan?
    - Does the likelihood of exposure vary with the type of method for collection and disposal of domestic ungulate carcasses?
    - How does the volume of NSAIDs used for veterinary treatment of domestic ungulates in South Asia compare to other regions? Is it the overall volume of NSAID use or when/how it is used that increases exposure in birds?
- Are areas with higher numbers of domestic ungulates more likely to result in higher rates of birds exposed to NSAIDs?
- Toxicity of NSAIDs to New World vultures? Other scavenger migratory bird species?
- Toxicity to migratory birds of other veterinary pharmaceuticals (other than NSAIDs) used to treat domestic ungulates?
- Is there a post-mortem metabolism of these compounds? How long are birds at risk of exposure post-cattle death?
- Is there likely to be illegal use of veterinary purchase of human diclofenac? Is there evidence of illegal use?

6. Conclusion

The 2006 ban on veterinary diclofenac in India, Nepal and Pakistan appears to have caused an initial recovery in numbers of the long-billed vulture in Pakistan and there appear to be similar trends for the oriental white-backed vultures in Nepal and long-billed vultures in India.[370] However, further research is needed on whether the bans have been effective and whether there are any other NSAIDs/veterinary pharmaceuticals that could adversely affect scavenging bird species.

Recent surveys in India indicate the ban on veterinary use of diclofenac has markedly reduced its levels in livestock carcasses to almost half of what they were prior to and immediately after the ban. However, levels of diclofenac in carcasses post-ban still remain sufficiently high to continue causing

[368] Naidoo et al., *Toxicity of non-steroidal anti-inflammatory drugs to Gyps vultures: a new threat from ketoprofen*.
[369] Cuthbert et al., *NSAIDs and scavenging birds: potential impacts beyond Asia's critically endangered vultures*.; Naidoo et al., *Toxicity of non-steroidal anti-inflammatory drugs to Gyps vultures: a new threat from ketoprofen*.
[370] Chaudhary et al., *First evidence that populations of the critically endangered Long-billed Vulture Gyps indicus in Pakistan have increased following the ban of the toxic veterinary drug diclofenac in south Asia*; Prakash, V., Bishwakarma, M. C., et al. (2012). The Population Decline of *Gyps* Vultures in India and Nepal Has Slowed since Veterinary Use of Diclofenac was Banned. *PloS one*, *7*(11), e49118.

population declines, estimated at 18 per cent per year for the oriental white-backed vulture in India.[371]

This situation may be confined to areas where (1) domestic ungulates form part of the diet of birds; (2) NSAIDs are used to treat domestic ungulates; and (3) similar exposure scenario to South Asia, eg, same level of treatment of sickly/old cattle with NSAIDs and carcasses left in the open.

The next steps are to evaluate the effect of diclofenac and other NSAIDs on migratory birds in areas outside India, Nepal and Pakistan where used to treat domestic ungulates whose carcasses are likely to be available for scavenging. Safe alternatives also need to be identified.

[371] Cuthbert, R., Taggart, M. A., Prakash, V., Saini, M., Swarup, D., Upreti, S., ... & Green, R. E. (2011). Effectiveness of action in India to reduce exposure of *Gyps* vultures to the toxic veterinary drug Diclofenac. *PloS one*, *6*(5), e19069.

Effects of lead ammunition and fishing weights

1. Introduction

Lead is a metal that is toxic to all vertebrate taxa, acting as a non-specific poison that affects all body systems (Franson and Pain 2011). Although lead is a naturally occurring element, it has a number of anthropogenic uses, some of which may expose wildlife to its toxic effects.

The first reports of lead poisoning in wild birds appeared in the 1870s when it was determined that the ingestion of lead gunshot was responsible for mortality amongst wildfowl in the USA and pheasants in the UK.[372] Particulate lead consumed by birds is broken down and dissolved in the muscular gizzard and/or stomach from where ionic lead is then absorbed into the bloodstream.[373] Today the main sources of lead for birds, and particularly for migratory birds, are lead ammunition and lead fishing weights, which will be the focus of this review.

However, lead from industrial sources including mining and smelting activities, lead in paint, petrol and other products can potentially affect migratory birds wherever there is exposure. Lead released into the environment from these anthropogenic sources and activities can also contaminate soil, and even water to some extent, and become bound in the tissues of plants and invertebrates, and their consumers.[374] This can occasionally pose a risk to migratory birds and may cause localised mortality events in areas of high exposure.

In the following sections of this report each potential source of lead is described separately. Regardless of the source and exposure route, the toxic effects of lead on an individual bird are generally similar. Nevertheless, the likelihood of exposure may vary according to the nature of environmental contamination and the prevailing conditions. Furthermore, differences in lead uptake both within and between taxa may arise as a result of variations in feeding ecology and physiology.

2. Likelihood of exposure to lead ammunition

Lead ammunition, in the form of bullets, air rifle pellets and shot can constitute a source of poisoning for migratory birds. Species that feed in areas where lead ammunition is used for any shooting activities are at particular risk of lead exposure and ingestion. Particulate lead can be consumed directly from the environment. Indirect consumption causing secondary poisoning can

[372] Franson, C.J., and D.J. Pain. 2011. Lead in birds. In *Environmental contaminants in biota: interpreting tissue concentrations*, edited by W. N. Beyer and J. P. Meador: Taylor & Francis Group. Boca Raton, USA; Pokras, M.A., and M.R. Kneeland. 2009. Understanding lead uptake and effects across species lines: a conservation medicine based approach. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA*, edited by R. T. Watson, M. Fuller, M. Pokras and W. G. Hunt. 7-22.

[373] Friend, M., and C.J. Franson. 1999. Field manual of wildlife diseases. General field procedures and diseases of birds. US Geological Survey, Madison, Wisonsin Resources Division.

[374] Bannon, D.I., P.J.Parsons, J.A. Centeno, S. Lal, H. Xu, A.B. Rosencrance, W.E. Dennis, and M.S. Johnson. 2011.Lead and copper in pigeons (*Columbia livia*) Exposed to a Small Arms-Range Soil. Archives of Environmental Contamination and Toxicology 60 (2): 351-360; Vyas, N.B., J.W. Spann, G.H. Heinz, W.N. Beyer, J.A. Jaquette, and J.M. Mengelkoch. 2000. Lead poisoning of passerines at a trap and skeet range. *Environmental Pollution* 107 (1): 159-166.

occur in predators and scavengers that consume the tissues of shot animals, animals with ingested particulate lead within their gastrointestinal tract or lead poisoned animals with elevated tissue lead levels. Alternatively, in some circumstances, lead from spent ammunition in the environment can become available through its accumulation in plants, invertebrates and small vertebrates which in turn may be consumed by other species further up the food chain, including migratory birds.[375]

There is a vast body of literature on the effects of lead ammunition on resident and migratory birds although reporting is mainly from North America and Europe.

2.1. Primary poisoning (direct consumption of lead from the environment)

2.2.1. Availability of lead ammunition in the environment

Each lead shotgun cartridge may contain several 100 lead pellets (depending on shot size) and only a small proportion of the pellets may be retrieved within a killed bird.[376] There have been a number of studies of the density of lead shot occurring in the environment (see Mateo (2009) for a summary for European countries),[377] but, globally, the precise tonnage of lead used for shooting each year is not known. In European wetlands an annual dispersion of 2,400-3,000 tonnes has been estimated, whilst for some countries the overall amount is considered to be several thousands of tonnes (6,000 in Spain and 4,600-10,000 in Italy).[378]

The highest reported densities of lead shot are found next to shooting ranges and clay pigeon shoots. Densities of up to 2400 shot/m^2 in the upper 5cm of shoreline and 257 shot/m^2 in the top 15cm of soil were recorded in the vicinity of two British clay pigeon shooting ranges.[379] Four Danish shooting ranges located near shallow water had shot densities ranging from 44-2045 shot/m^2 and two Dutch clay shooting ranges had 400 and 2195 shot/m^2.[380]

Densities of shot resulting from hunting rather than clay pigeon shooting are not surprisingly greatest where shooting is most concentrated (Stansley, Widjeskog, and Roscoe 1992). For hunting, the method and scale of the activity will determine the density of shot deposited in the local environment.[381] As an example, high densities of shot have been found in southern European wetlands (up to 399 shot/m^2 within the top 30cm) where hunting pressure is high and habitats for quarry species (ie, waterbirds) are relatively restricted. In comparison, northern European wetlands

[375] Bennett, J.R., C.A. Kaufman, I. Koch, J. Sova, and K.J. Reimer. 2007. Ecological risk assessment of lead contamination at rifle and pistol ranges using techniques to account for site characteristics. *Science of The Total Environment* 374 (1): 91-101; Migliorini, M., G. Pigino, N. Bianchi, F. Bernini, and C. Leonzio. 2004. The effects of heavy metal contamination on the soil arthropod community of a shooting range. *Environmental Pollution* 129 (2): 331-340.

[376] Cromie, R.L., A. Loram, L. Hurst, M. O'Brien, J. Newth, M.J. Brown, and J.P. Harradine. 2010. Compliance with the Environmental Protection (Restrictions on Use of Lead Shot)(England) Regulations 1999. Report to Defra. Bristol, UK.

[377] Mateo, R. 2009. Lead poisoning in wild birds in Europe and the regulations adopted by different countries. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA*, edited by R. T. Watson, M. Fuller, M. Pokras and W. G. Hunt. 71-98.

[378] Andreotti and Borghesi 2012; Guitart and Mateo 2006.

[379] O'Halloran, J., A.A. Myers, and P.F. Duggan. 1988. Lead poisoning in swans and sources of contamination in Ireland. *Journal of Zoology* 216 (2): 211-223; Mellor, A., and C. McCartney. 1994. The effects of lead shot deposition on soils and crops at a clay pigeon shooting site in northern England. *Soil Use and Management* 10 (3): 124-129.

[380] Smit, T., T. Bakhuizen, C.P.H. Gassenbeek, and L.G. Morall. 1988. Occurrence of lead pellets around duck blinds and at clay pigeon grounds. *Limosa* 61: 183-186; Peterson, B.D., and H. Meltofte. 1979. Occurrence of lead shot in the wetlands of western Jutland, Denmark, and in the gizzards of Danish ducks. *Dansk. orn. Foren. Tidsskr* 73: 257-64.

[381] Mateo, *Lead poisoning in wild birds in Europe and the regulations adopted by different countries*.

have been found to have lower densities of shot. For example, fewer than 100 shot/m^2 were found within the top 20cm layer, and generally between 10 and 50 shot/m^2 were recorded in UK wetlands in the 1980s.[382]

There have been relatively fewer studies of shot density in non-wetland areas used for hunting. One Spanish estate where red-legged partridge (*Alectoris rufa*) were being shot with up to 16 guns positioned at 40m intervals, reported a shot density of 7.4 shot/m^2 within the top 1cm of soil (shooting occurred over two days per year, for two years, with one shooting-free year in between).[383]

Lead is a relatively stable metal under most conditions and would retain its structural integrity for a considerable period of time, with complete decomposition likely taking tens or hundreds of years.[384] Depending on the substrate and/or local conditions shot may sink over time or be moved by water following inundation, ploughing or other substrate movements.[385] Hence lead ammunition can persist in the environment sometimes becoming less available to birds or being moved and re-exposed by natural or anthropogenic processes. This potential for a "historical legacy" of lead shot in the environment is an important aspect of the epidemiology of lead poisoning of wild birds.

2.1.2. Influence of feeding ecology on exposure

It is well established that spent lead gunshot in the environment may be ingested directly by birds, particularly by those with a muscular gizzard such as Anseriformes, Galliformes and granivorous Columbiformes. Indeed, it is an unfortunate coincidence that lead shot is primarily used for killing the very quarry species which are most likely to inadvertently consume it from the environment thus increasing their potential exposure.

Birds most likely take up lead shot when they mistake it for food items or grit, which is retained in the muscular gizzard to aid mechanical breakdown of food. Poisoning and mortality of wildfowl, other waterbirds and terrestrial birds through this route of lead ingestion from gunshot is well documented globally.[386]

As lead may be consumed by birds as they forage, variations in feeding ecology will affect the likelihood of exposure to lead shot. For example, whilst ducks and swans feeding in shallow sediments may ingest lead, up-ending swans and diving ducks may be exposed to shot which is too deep for dabbling ducks.

[382] Mudge, G.P. 1984. Densities and settlement rates of spent shotgun pellets in British wetland soils. *Environmental Pollution Series B-Chemical and Physical* 8 (4): 299-318.

[383] Ferrandis, P., R. Mateo, F.R. López-Serrano, M.n. Martínez-Haro, and E. Martínez-Duro. 2008. Lead-shot exposure in red-legged partridge (*Alectoris rufa*) on a driven shooting estate. *Environmental science & technology* 42 (16): 6271-6277.

[384] Rooney, C.P., R.G. McLaren, and L.M. Condron. 2007. Control of lead solubility in soil contaminated with lead shot: effect of soil pH. *Environmental Pollution* 149 (2): 149-157; Scheuhammer, A.M., and S.L. Norris. 1996. The ecotoxicology of lead shot and lead fishing weights. *Ecotoxicology* 5 (5): 279-295.

[385] Thomas, C.M., J.G. Mensik, and C.L. Feldheim. 2001. Effects of tillage on lead shot distribution in wetland sediments. *The Journal of Wildlife Management*: 40-46.

[386] Newth, J.L., R.L. Cromie, M.J. Brown, R.J. Delahay, A.A. Meharg, C. Deacon, G.J. Norton, M.F. O'Brien, and D.J. Pain. 2012. Poisoning from lead gunshot: still a threat to wild waterbirds in Britain. *European Journal of Wildlife Research* 59 (2): 195-204; Bingham, R.J., R.T. Larsen, J.A. Bissonette, and J.T. Flinders. 2009. Causes and consequences of ingested lead pellets in Chukars. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA*, edited by R. T. Watson, M. Fuller, M. Pokras and W. G. Hunt. 190-193.

In recognition of the particular risks of exposure of waterbirds to lead shot, restrictions on its use (and use of non-toxic alternatives) have been introduced over wetlands in many countries.[387] However, species such as geese and swans which feed extensively in terrestrial and agricultural habitats may be exposed to lead shot in both wetland and terrestrial environments.

As well as these broader aspects of feeding ecology and shot availability in the environment, consumption of shot may be affected by the availability of alternative grit, the absence of which increases rate of shot ingestion;[388] and seasonal diet of the bird, eg, during periods of abundance of hard food such as seeds birds may increase their grit, and thus lead, ingestion.[389]

2.1.3. Other factors affecting exposure

Several environmental and anthropogenic factors influence the spatio-temporal distribution of lead ammunition in the environment and thus exposure risk, including:

- proximity to hunting or other shooting activities where lead ammunition is being used;
- hunting intensity with lead shot, in general the greater the intensity the greater the potential exposure;
- legislation (and compliance therewith) relating to the use of lead shot. In the USA, the impact of a ban on the use of lead shot for waterfowl shooting shifted to the introduced non-toxic alternatives.[390] Conversely, this was not the case in England where compliance with legislation is low;[391]
- time in relation to hunting seasons, where exposure towards the end of a hunting season will be greater;
- habitat over which lead is used and its attractiveness to birds, eg, wetland type;
- substrate type, water inundation and other local conditions and how these affect sinking/movement of shot over time;
- land management, eg, ploughing in of shot;[392]
- land disruption, eg, temporary inundation of terrestrial shot-over areas may attract dabbling ducks; spates and flooding can erode watercourses and expose historically deposited lead;
- chemical and physical processes in the environment, breaking down the shot over time.

2.1.4. Risk from embedded lead ammunition

A proportion of quarry species may survive with lead ammunition embedded in their tissues (see Section 2.2.1). Although it is known that toxicity from retained lead ammunition can occur in

[387] Avery, D. And R.T. Watson. 2009. Regulations of lead-based ammunition around the world. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA*, edited by R. T. Watson, M. Fuller, M. Pokras and W. G. Hunt. 161-168.

[388] Mateo, R., A.J. Green, H. Lefranc, R. Baos, and J. Figuerola. 2007. Lead poisoning in wild birds from southern Spain: a comparative study of wetland areas and species affected, and trends over time. *Ecotoxicology and Environmental Safety* 66 (1): 119-126.

[389] Rocke, T.E., C.J. Brand, and J.G. Mensik. 1997. Site-specific lead exposure from lead pellet ingestion in sentinel mallards. *Journal of Wildlife Management* 61 (1): 228-234.

[390] Anderson, William L., Stephen P. Havera, and Bradley W. Zercher. "Ingestion of lead and nontoxic shotgun pellets by ducks in the Mississippi flyway." *The Journal of wildlife management* (2000): 848-857.

[391] Cromie, et al., *Compliance with the Environmental Protection (Restrictions on Use of Lead Shot)(England) Regulations 1999*.

[392] Thomas, et al., *Effects of tillage on lead shot distribution in wetland sediments*.

humans,[393] it has been suggested that toxic effects within wounded but surviving wildlife should not necessarily arise from ammunition embedded subcutaneously or intramuscularly as the pH conditions in these tissues may not dissolve lead.[394] Tavecchia *et al*. (2001) reported a 19 per cent relative decrease in survival of adult of mallard in the Camargue, France, with tissue-embedded pellets;[395] however, it is difficult to determine whether this is due to the trauma of being shot and its sequelae or due to lead toxicity or a combination of both.

2. 2. Secondary poisoning (consumption of lead-containing food items)

2.2.1. Availability of lead ammunition within food items

Lead ammunition within the alimentary tract and/or embedded in either live prey or carrion may provide a potential route of secondary lead poisoning for predatory and scavenging species. For example, 13 per cent of living whooper swans (*Cygnus cygnus*) and 23 per cent of Bewick's swans (*Cygnus columbianus bewickii*) were found to carry shot within their tissues (Newth, Brown, and Rees 2011). Embedded shot prevalence in first winter and adult pink-footed geese (*Anser brachyrhynchus*) are between 7 per cent and 36 per cent respectively.[396] In an extensive study of some 40,000 common teal (*Anas crecca*) trapped in France, Guillemain *et al*. (2007) found some 9.6 per cent and 7.5 per cent of adult males and females respectively carried embedded shot.

Embedded shot found in North American wildfowl quarry species ranges between 10 and 42 per cent.[397] In Greenland, embedded shot rates of up to 20 per cent and 29 per cent were found respectively in king eiders (*Somateria spectabilis*) and common eiders (*S. mollissima*).[398] However, similar data are not available for a wide range of species over broad geographical regions and importantly, impacts of changes in legislation regarding the use of lead (and legislative compliance levels) will affect prevalence of toxic lead shot.

Although most studies relating to lead poisoning have focused on lead shot, there is a growing body of literature relating to poisoning caused by bullets. Bullets, like shot, fragment upon impact and hence particles of bullets may be found some distance from the wound canal[399] providing contamination of a greater proportion of the tissues of potentially consumed animals than previously thought.

[393] McQuirter, J. L., S. J. Rothenberg, G.A. Dinkins, V. Kondrashov, M. Manalo, and A.C. Todd. 2004. Change in blood lead concentration up to 1 year after a gunshot wound with a retained bullet. *American Journal of Epidemiology* 159(7): 683-692.

[394] De Francisco, N., J.D.R. Troya, and E.I. Aguera. 2003. Lead and lead toxicity in domestic and free living birds. *Avian Pathology* 32 (1): 3-13.

[395] Tavecchia, G., R. Pradel, J.-D. Lebreton, A.R. Johnson, and J.-Y. Mondain Monval. 2001. The effect of lead exposure on survival of adult mallards in the Camargue, southern France. *Journal of Applied Ecology* 38 (6): 1197-1207.

[396] Noer, H., J. Madsen, and P. Hartmann. 2007. Reducing wounding of game by shotgun hunting: effects of a Danish action plan on pink-footed geese. *Journal of Applied Ecology* 44 (3): 653-662; Noer, H., and J. Madsen. 1996. Shotgun pellet loads and infliction rates in pink-footed geese *Anser brachyrhynchus*. *Wildlife Biology* 2 (2): 65-73.

[397] Scheuhammer and Norris,*The ecotoxicology of lead shot and lead fishing weights*.

[398] Falk, K., F. Merkel, K. Kampp, and S.E. Jamieson. 2006. Embedded lead shot and infliction rates in common eiders *Somateria mollissima* and king eiders *S. spectabilis* wintering in southwest Greenland. *Wildlife Biology* 12(3): 313-321.

[399] Knott, J., J. Gilbert, D.G. Hoccom, and R.E. Green. 2010. Implications for wildlife and humans of dietary exposure to lead from fragments of lead rifle bullets in deer shot in the UK. *Science of The Total Environment* 409 (1): 95-99; Pain, D.J., I.J. Fisher, and V.G. Thomas. 2009. A global update of lead poisoning in terrestrial birds from ammunition sources. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA*, edited by R. T. Watson, M. Fuller, M. Pokras and W. G. Hunt. 99-118.

Animals suffering from lead poisoning *e.g.* with high soft tissue levels of lead (see Section 4.1) may also prove a source of lead for predatory and scavenging birds.

2.2.2. Influence of feeding ecology on exposure

Predatory or scavenging species are exposed to metallic lead whenever they consume prey containing embedded shot or bullet fragments. Lead bullets, or large fragments thereof, are more likely to be consumed by large raptors, which account for cases of lead poisoning in species such as white-tailed eagles (*Haliaeetus albicilla*),[400] Steller's sea eagles (*H. pelagicus*),[401] golden eagles (*Aquila chrysaetos*)[402] and California condors (*Gymnogyps californianus*).[403]

2.2.3. Other factors affecting exposure

As well as taxon and feeding ecology, there are other factors which will affect exposure to lead including:

- scale of hunting with lead which will be directly related to exposure to contaminated prey;
- hunting behaviour will also affect exposure *e.g.* wherever there is a tradition of leaving viscera from hunted game, this can in effect create a ready source of particulate lead;[404]
- hunter competence and adherence to hunting codes of conduct will also affect availability of lead to scavengers and predators *e.g.* reducing wounding rates and/or burying viscera;
- legislation: where regulations exist to prevent use of lead shot or bullets and there is good compliance, exposure will be reduced;
- degree of debilitation of prey: predation risks are higher for injured (potentially shot with lead) and sick (potentially lead poisoned and still carrying metallic lead) individuals and thus there is an increased risk of these forming a disproportionately larger part of the diet of predators and scavengers.

2.2.4. Risk from bioaccumulation and contamination of the wider environment

Several studies have highlighted how environmental contamination with lead ammunition can lead to bioaccumulation of lead within vegetation, invertebrates and other edaphic organisms.[405] Such accumulation can create another source of lead exposure for birds and other wildlife.[406]

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[400] Krone, O., N. Kenntner, A. Trinogga, M. Nadjafzadeh, F. Scholz, J. Sulawa, K. Totschek, P. Schuck-Wersig and R. Zieschank. 2009. *Lead poisoning in whitetailed sea eagles: causes and approaches to solutions in Germany*. In: *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA*, edited by R. T. Watson, M. Fuller, M. Pokras and W. G. Hunt. 289-301.

[401] Saito, K. 2009. Lead poisoning of Steller's sea-eagle (*Haliaeetus pelagicus*) and whitetailed eagle (*Haliaeetus albicilla*) caused by the ingestion of lead bullets and slugs. *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans, The Peregrine Fund, Boise, Idaho*: 302-309.

[402] Craig, T.H., J.W. Connelly, E.H. Craig and T.L. Parker. 1990. Lead concentrations in golden and bald eagles. *The Wilson Bulletin:* 130-133.

[403] Finkelstein, M.E., D.F. Doak, D. George, J. Burnett, J. Brandt, M. Church, J. Grantham, and D.R. Smith. 2012. Lead poisoning and the deceptive recovery of the critically endangered California condor. *Proceedings of the National Academy of Sciences*, 109 (28): 11449-11454.

[404] Pain, D. J., C. Bavoux, and G. Burneleau. 1997. Seasonal blood lead concentrations in Marsh Harriers (*Circus aeruginosus*) from Charente- Maritime, France: relationship with hunting season. *Biological Conservation* 81:1–7.

[405] Bennett, et al., *Ecological risk assessment of lead contamination at rifle and pistol ranges using techniques to account for site characteristics*; Migliorini, et al., *The effects of heavy metal contamination on the soil arthropod community of a shooting range*.

[406] Mellor, A., and C. McCartney. 1994. The effects of lead shot deposition on soils and crops at a clay pigeon shooting site in northern England. *Soil Use and Management* 10 (3): 124-129.

3. Likelihood of exposure to lead fishing weights

Fishing weights, also known as 'sinkers' or 'leads' are often made of lead and vary in shape and size. Lead can also be found in other equipment used for angling and commercial fishing such as fishing lines, downriggers, lures (or jigs) and seine ropes. Lead fishing weights can be introduced into the aquatic environment by commercial and recreational anglers when they are lost through accidental or intentional line breakage or when they are otherwise discarded.[407]

Lead fishing weights become available to birds when they are lost or discarded into the aquatic environment. Birds may ingest these lead items when feeding in waterbodies or near-shore sediments. As with lead ammunition, there is also a risk of secondary poisoning when predators or scavengers ingest lead fishing weights lodged in their prey, although, the extent of this phenomenon is currently unknown.[408]

3.1. Availability of lead fishing weights in the aquatic environment

Lost lead fishing weights are likely to be relatively stable in the environment, remaining intact and persisting in water for decades to centuries.[409] The quantity of lead fishing tackle that enters the aquatic environment and becomes available for ingestion by birds is not accurately known, but some estimates are available for certain locations. The mass of lead sold as fishing weights every year is estimated at 3,977 tonnes in the USA and up to 559 tonnes in Canada.[410] Most of these new fishing weights are believed to be purchased to replace lost weights and thus it is suggested that these estimates roughly equate to the mass of those lost and discarded.[411] In 2004, the European Commission reported that the total consumption of lead for fishing weights used in non-commercial angling was 2000 to 6000 tonnes a year in 25 EU member states.[412] Another study estimated that approximately one metric tonne of lead fishing weights is lost annually across five fished waterbodies in Minnesota, USA.[413]

3.2. Influence of feeding ecology on exposure

Birds that consume grain, vegetation and insects, have a muscular gizzard to break down food items prior to digestion. As with lead shot, waterbirds are particularly prone to ingesting small lead items such as fishing weights, as they mistake them for food particles or for the grit they require to aid the breakdown of food within the gizzard.

Species that feed in near-shore soils and sediments and in waterbodies that are, or have been heavily fished, are particularly at risk of lead poisoning from the inadvertent consumption of lost or

[407] Goddard, C.I., N.J. Leonard, D.L. Stang, P.J. Wingate, B.A. Rattner, J.C. Franson, and S.R. Sheffield. 2008. Management concerns about known and potential impacts of lead use in shooting and in fishing activities. *Fisheries* 33 (5): 228-236.

[408] Ibid and Pain, et al., *A global update of lead poisoning in terrestrial birds from ammunition sources.*

[409] Sporting Arms and Ammunition Manufacturers' Institute Inc. 1996. Lead mobility at shooting ranges. Newtown, Connecticut.

[410] AEWA. 2012. Literature review: effects of the use of lead fishing weights on waterbirds and wetlands. *Doc StC Inf. 7.6*: 1-20; Scheuhammer, A.M., S.L. Money, D.A. Kirk, and G. Donaldson. 2003. *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife*. Vol. 108: Canadian Wildlife Service Ottawa, Canada.

[411] Scheuhammer, et al., *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife*.

[412] European Commission. *Advantages and drawbacks of restricting the marketing and use of lead in ammunition, fishing sinkers and candle wicks* 2004. Available from http://ec.europa.eu/enterprise/sectors/chemicals/files/studies/ehn_lead_final_report_en.pdf.

[413] Radomski, P., T. Heinrich, T.S. Jones, P. Rivers, and P. Talmage. 2006. Estimates of tackle loss for five Minnesota walleye fisheries. *North American Journal of Fisheries Management* 26 (1): 206-212.

discarded lead fishing weights.[414] Waterbirds are most likely to ingest small fishing weights, exclusively used for recreational angling, that weigh less than 50 g and are smaller than 2 cm in any dimension.[415]

Larger waterbirds such as the common loon (*Gavia immer*) are also able to ingest larger and heavier weights. There is evidence to suggest that some piscivorous species, including the common loon, may ingest lead fishing weights as a by-product of consuming lost bait fish attached to the fishing tackle.[416] Predators and scavengers that feed on waterbirds poisoned by fishing weights may also be at risk of secondary poisoning.[417]

3.3. Other factors affecting exposure

The amounts of lead fishing tackle dispersed in wetlands and their subsequent exposure to waterbirds may vary according to the intensity of angling pressure, the location of angling activity such as distance from the shoreline or boat, the influence of the aquatic habitat on the likelihood of equipment breakage and loss, and angler skill.[418] For instance, it is estimated that there were fewer than 0.01 weights/m^2 in areas of low angling pressure and up to 0.47 weights/m^2 in areas of high angling pressure.[419] Lead weights may persist for tens or hundreds of years, accumulating in sediments.[420] Therefore, lead weights may also remain available to birds inhabiting areas that were historically fished even if not currently fished. However, lost lead items may settle deeper into the sediment as time passes[421] and may thus become increasingly unavailable for ingestion by birds.[422]

4. Toxicity of lead to migratory birds and its impacts

4.2. Impacts of lead ammunition

4.2.1. Individual impacts

Both acute and chronic lead poisoning can cause mortality of birds (see Table 1). Ingestion of a single lead pellet (representing up to several grams of lead) may be sufficient to kill a small bird, such as a

[414] Goddard, et al., *Management concerns about known and potential impacts of lead use in shooting and in fishing activities*.

[415] Scheuhammer, et al., *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife*.

[416] Franson, C.J., S.P. Hansen, T.E. Creekmore, C.J. Brand, D.C. Evers, A.E. Duerr, and S. DeStefano. 2003. Lead fishing weights and other fishing tackle in selected waterbirds. *Waterbirds* 26 (3): 345-352; Stone, W.B., and J.C. Okoniewski. 2001. Necropsy findings and environmental contaminants in common loons from New York. *Journal of Wildlife Diseases* 37 (1): 178-184.

[417] Goddard, et al., *Management concerns about known and potential impacts of lead use in shooting and in fishing activities*; and Pain, et al., *A global update of lead poisoning in terrestrial birds from ammunition sources.*

[418] Goddard, et al., *Management concerns about known and potential impacts of lead use in shooting and in fishing activities*; Scheuhammer, et al., *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife;* Rattner, B.A., J.C. Franson, S.R. Sheffield, C.I. Goddard, N.J. Leonard, D. Stang, and P.J. Wingate. 2008. Technical review of the sources and implications of lead ammunition and fishing tackle on natural resources. *The Wildlife Society and American Fisheries Society Technical Review*: 08-01; Sears, J. 1989. A review of lead poisoning among the River Thames Mute Swan *Cygnus olor* population. *Wildfowl* 40: 151-152; Simpson, V.R., A.E. Hunt, and M.C. French. 1979. Chronic lead-poisoning in a herd of mute swans. *Environmental Pollution* 18 (3): 187-202.

[419] Duerr, A.E., and S. DeStefano. 1999. Using a metal detector to determine lead sinker abundance in waterbird habitat. *Wildlife Society Bulletin* 27 (4): 952-958.

[420] Rooney, C.P., R.G. McLaren, and L.M. Condron. 2007. Control of lead solubility in soil contaminated with lead shot: effect of soil pH. *Environmental Pollution* 149 (2): 149-157.

[421] Birkhead, M. 1983. Lead levels in the blood of mute swans *Cygnus olor* on the River thames. *Journal of Zoology* 199 (January): 59-73; Sears, *A review of lead poisoning among the River Thames Mute Swan Cygnus olor population*.

[422] Scheuhammer, et al., *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife.*

common teal.[423] Clinical signs of poisoning may be seen within a few days of ingestion of lead, with death occurring within two or three weeks, or months depending on dose and exposure period.

Lead toxicosis can result in a range of clinical conditions such as: anaemia, lethargy, anorexia, paralysis of the upper alimentary canal leading to food impaction and vomiting; weight loss and emaciation; a range of central nervous signs including muscular in-coordination, paralysis of the legs and/or wings (birds which lose their ability to walk may drag themselves about causing abrasions to their wings); convulsions and diarrhoea.[424]

Sub-lethal doses can result in mortality through the effects of lead on immunocompetence and birds with clinical or sub-clinical lead poisoning would be expected to succumb more readily to other causes of death involving infectious agents or autoimmune conditions.[425]

Sub-lethal effects causing immunosuppression, loss of coordination and/or partial paralyses may subsequently contribute to premature death from other causes such as disease, starvation, predation and flying accidents.[426] Reduced growth rate has been shown in American kestrel nestlings (*Falco sparverius*) from chronic lead exposure in adults. [427]

However, it is difficult to relate lead tissue concentrations to toxic effects as the level and duration of lead exposure, previous history and response to exposure, the overall health of the bird, the extent of existing damage and the potential interaction between lead and other disease agents may all be influential factors. There are also variations in sensitivity to lead poisoning within and between taxa.[428]

The absorption of lead and its delivery to body tissues may also be affected by a number of factors such as age, gender, [429] and stomach type. [430]

Table 1: Suggested interpretations of tissue lead concentrations in three orders of birds (adapted from (Franson and Pain 2011)). Birds with severe clinical poisoning are at risk of mortality, whereas those with subclinical and clinical may only show sub-lethal effects.

Order	Blood µg/dL	Liver mg/kg ww	Kidney mg/kg ww	Sources

[423] Mateo, *Lead poisoning in wild birds in Europe and the regulations adopted by different countries*; Guillemain, M., O. Devineau, J.-D. Lebreton, J.-Y. Mondain-Monval, A.R. Johnson, and G. Simon. 2007. Lead shot and teal (*Anas crecca*) in the Camargue, southern France: effects of embedded and ingested pellets on survival. *Biological Conservation* 137 (4): 567-576; Tavecchia, G., R. Pradel, J.-D. Lebreton, A.R. Johnson, and J.-Y. Mondain Monval. 2001. The effect of lead exposure on survival of adult mallards in the Camargue, southern France. *Journal of Applied Ecology* 38 (6): 1197-1207.

[424] Friend and Franson, Field manual of wildlife diseases; Sainsbury, A.W., P.M. Bennett, and J.K. Kirkwood. 1995. The welfare of free-living wild animals in Europe: harm caused by human activities. *Animal Welfare* 4 (3): 183-206.

[425] Grasman, K.A., and P.F. Scanlon. 1995. Effects of acute lead ingestion and diet on antibody and T-cell-mediated immunity in Japanese quail. *Archives of Environmental Contamination and Toxicology* 28 (2): 161-167; Trust, K.A., M.W. Miller, J.K. Ringelman, and I. Orme. 1990. Effects of ingested lead on antibody production in mallards (Anas platyrhynchos). *Journal of Wildlife Diseases* 26 (3): 316-322.

[426] Kelly, A., and S. Kelly. 2005. Are mute swans with elevated blood lead levels more likely to collide with overhead power lines? *Waterbirds* 28 (3): 331-334; Tavecchia, et al., *The effect of lead exposure on survival of adult mallards in the Camargue, southern France*.

[427] Scheuhammer, A.M. 1987. The chronic toxicity of aluminium, cadmium, mercury and lead in birds: a review. *Environmental Pollution* 46 (4): 263-295.

[428] Carpenter, J.W., O.H. Pattee, S.H. Fritts, B.A. Rattner, S.N. Wiemeyer, J.A. Royle, and M.R. Smith. 2003. Experimental lead poisoning in turkey vultures (*Cathartes aura*). *Journal of Wildlife Diseases* 39 (1): 96-104.

[429] Scheuhammer and Norris, *The ecotoxicology of lead shot and lead fishing weights*.

[430] Pain, et al., *A global update of lead poisoning in terrestrial birds from ammunition sources*.

Anseriformes				
Subclinical	20<50	2<6	2<6	Dieter and Finley (1979), Degerness (1991)
Clinical	50-100	6-10	6-15	Longcore et al. (1974), Beyer et al. (2000)
Severe	>100	>10	>15	Nakade et al. (2005), Degerness et al. (2006)
Falconiformes				
Subclinical	20<50	2<6	2<4	Custer et al. (1984), Henny et al. (1991)
Clinical	50-100	6-10	4-6	Kramer and Redig (1997), Lumeij et al. (1985)
Severe	>100	>10	>6	Pattee et al. (2006), Langelier et al. (1991)
Columbiformes				
Subclinical	20<200	2<6	2<15	DeMent et al. (1987), Scheuhammer and Wilson (1990)
Clinical	200-300	6-15	15-30	Anders et al. (1982), Boyer et al. (1985)
Severe	>300	>15	>30	Barthalmus et al. (1977), Schulz et al. (2006)

4.2.2. Population impacts

Lead, as a toxin that causes mortality, both direct and indirectly, and additionally has possible impacts on productivity has the potential for population level impacts.

Whilst large-scale mortality events occasionally occur, mortality from lead poisoning is usually less conspicuous, which may result in frequent and largely invisible losses of small numbers of birds remaining undetected.[431] Moribund birds often become increasingly reclusive and dead birds may be scavenged before being detected.[432] Some birds may die from lead poisoning without exhibiting typical pathology and thus their death may be subsequently attributed to another cause.[433]

Determining impacts at a population level is not straightforward. Robust surveillance data, current ingestion rates, an understanding of population dynamics, dispersal, site fidelity, long-term reproductive success and mortality rates are required for survival analyses and an assessment of population-level effects. Collection of such data is challenging and may require commitment to long-term studies so for most species, an accurate assessment of the extent of mortality from lead ingestion is not currently possible.

In Europe, it has been estimated that approximately 8.6 per cent of wildfowl (representing 17 different species), may die every winter from lead poisoning caused by ingestion of lead gunshot.[434] Whilst some of the information on which this estimate was based is old and shot ingestion rates may now be different in some species, overall mortality is nonetheless likely to remain high. Concern has been expressed over the potential for lead poisoning to be contributing to declines in some species

[431] Newth, et al., *Poisoning from lead gunshot: still a threat to wild waterbirds in Britain*.
[432] Ibid and Pain, D.J. 1991b. Why are lead-poisoned waterfowl rarely seen? The disappearance of waterfowl carcasses in the Camargue, France. *Wildfowl* 42: 118-122.
[433] Beyer, W.N., J.C. Franson, et al. 1998. Retrospective study of the diagnostic criteria in a lead-poisoning survey of waterfowl. *Archives of Environmental Contamination and Toxicology* 35 (3): 506-512.
[434] Mateo, *Lead poisoning in wild birds in Europe and the regulations adopted by different countries*.

of common wildfowl, such as pochard (*Aythya ferina*) and northern pintail (*Anas acuta*).[435] In addition, lead has been identified as a threat to certain globally threatened European wildfowl such as the white-headed duck (*Oxyura leucocephala*) and marbled teal (*Marmaronetta angustirostris*).[436] Sub-lethal effects of lead ingestion are likely to affect many more birds and in some cases are likely to have contributed to death from other causes.

Prior to the ban on lead shot for waterfowl hunting in the USA in 1991 it was estimated 1.6 - 2.4 million of some 100 million wildfowl died from lead poisoning annually.[437] Losses still occur due to historically deposited lead, hunting other species with lead, non-compliance with regulations, and areas contaminated from target shooting activities.[438]

In recent times a body of evidence has accumulated describing lead poisoning in terrestrial birds. These include quarry species and their relatives such as mourning doves (*Zenaida macroura*) and grey partridge (*Perdix perdix*), which ingest spent lead shot whilst feeding.[439] Also, further evidence has emerged of lead poisoning in raptors from lead ammunition embedded in the tissues of prey and carrion.[440]

Long-lived species with relatively low rates of reproduction, such as eagles and condors are particularly susceptible to the effects of mortality caused by lead poisoning. The threatened population of the Spanish imperial eagle (*Aquila adalberti*), cannot sustain high adult mortality and is thus particularly at risk.[441] Perhaps the best studied impact of lead ammunition-related toxicosis is represented by the case of the endangered California condor (*Gymnogyps californianus*). Declining in part due to lead poisoning, toxicosis is currently the principle cause of mortality despite various measures to reduce exposure to lead ammunition including restrictions on its use throughout the birds' range.[442]

A ban on use of lead bullets in Hokkaido, Japan, has been introduced, where population modelling of the effects of lead poisoning on Steller's sea eagles indicated that a decline in the population would have been expected without such an intervention.[443] White-tailed sea eagles suffer high levels of mortality due to lead poisoning in a number of countries such as Finland, Germany and Austria.[444] In Germany, population modelling has shown that their population trend would increase more rapidly

[435] Ibid.
[436] Ibid.
[437] Friend and Franson, *Field manual of wildlife diseases*.
[438] Ibid.
[439] Franson, C.J., S.P. Hansen, and J.H. Schulz. 2009. Ingested shot and tissue lead concentrations in Mourning Doves; Potts, G.R. 2005. Incidence of ingested lead gunshot in wild grey partridges (*Perdix perdix*) from the UK. *European Journal of Wildlife Research* 51 (1): 31-34.
[440] Pain, et al., *A global update of lead poisoning in terrestrial birds from ammunition sources*.
[441] Sánchez, B., L. González, and B. Barov. 2008. Action plan for the Spanish imperial eagle *Aquila adalberti* in the European Union. BirdLife International for the European Commission.
[442] Cade, T.J. 2007. Exposure of California condors to lead from spent ammunition. *The Journal of Wildlife Management* 71 (7): 2125-2133.
[443] Ueta, M., and V. Masterov. 2000. Estimation by a computer simulation of population trend of Steller's sea eagles. In *First symposium on stellar's and white-tailed sea eagles in East Asia. Wild Bird Society of Japan, Tokyo*, edited by M. Ueta and M. J. McGrady.
[444] Krone, O., T. Stjernberg, et al. 2006. Mortality factors, helminth burden, and contaminant residues in white-tailed sea eagles (*Haliaeetus albicilla*) from Finland. *AMBIO: A Journal of the Human Environment, 35*(3), 98-104.

if lead poisoning was prevented.[445] Whilst there is a range of non-toxic alternatives to lead shot, the majority of bullets are still manufactured from lead despite advances in technology.[446]

[445] Kenntner, N., F. Tataruch and O. Krone. 2001. Heavy metals in soft tissue of white-tailed eagles found dead or moribund in Germany and Austria from 1993 to 2000. *Environmental Toxicology and Chemistry, 20*(8), 1831-1837.

[446] Thomas, V.G. 2013. Lead-free hunting rifle ammunition: product availability, price, effectiveness, and role in global wildlife conservation. *Ambio* 42 (6): 1-9.

Table 2: Migratory bird taxa known* to be affected by lead poisoning, including species of Annexes I & II of the Convention of Migratory Species.

Order	Family	Common name	Scientific name	CMS Annex	Lead source				
					Ammunition	Fishing weights	Industrial	Paint	Other
SPHENISCIFORMES						✓			
PROCELLARIIFORMES								✓	
	Diomedeidae	Laysan albatross	*Phoebastria immutabilis*	II				✓†	
GAVIIFORMES						✓†			
	Gavidae	Common loon (Great northern diver)	*Gavia immer immer (NW European population)*	II		✓†			
PODICIPEDIFORMES					✓	✓			
PELECANIFORMES					✓	✓			
CICONIIFORMES					✓	✓			
PHOENICOPTERIFORMES					✓				
ANSERIFORMES					✓†	✓†	✓		
	Anatidae	Marbled Teal	*Marmaronetta angustirostris*	I	✓†				
	Anatidae	Ferruginous Duck	*Aythya nyroca*	I	✓				
	Anatidae	White-headed Duck	*Oxyura leucocephala*	I	✓†				
FALCONIFORMES					✓†		✓	✓	
	Cathartidae	New world vultures & condors	*Cathartidae. spp.*	II	✓†			✓	
	Pandionidae	Osprey	*Pandion haliaetus*	II			✓		
	Accipitridae	White-tailed eagle	*Haliaeetus albicilla*	I	✓†				
	Accipitridae	Steller's Sea eagle	*Haliaeetus pelagicus*	I	✓†				
	Accipitridae	Spanish Imperial eagle	*Aquila adalberti*	I	✓				
	Accipitridae	Hawks and eagles	*Accipitridae. spp.*	II	✓†				
GRUIFORMES					✓	✓	✓		
	Rallidae	Eurasian coot	*Fulica atra atra (Mediterranean & Black Sea population)*	II	✓				

GALLIFORMES					✓†				
CHARADRIIFORMES					✓		✓		
	Charadriidae	Plovers, dotterels and lapwings	*C. spp.*	II	✓				
	Scolopacidae	Sandpipers and snipes	*S. spp.*	II	✓		✓		
COLUMBIFORMES				II	✓				
	Columbidae	European turtle dove	*Streptopelia turtur turtur*	II	✓				
PSITTACIFORMES				I					✓
CORACIIFORMES				II			✓		
PASSERIFORMES				I	✓		✓		

* reports known to authors – *this will not be comprehensive*

✓ morbidity and/or mortality reported

† identified as causing significant morbidity and/or mortality in one or more geographic locations - *noting this categorisation is illustrative rather than definitive*.

4.3. Impacts of lead fishing weights

4.3.1. Individual impacts and species affected

Poisoning from lead fishing weights has the greatest impact on waterbirds that feed in or near to waterbodies, particularly in environments that have been heavily fished, and where there is a greater availability of lost or discarded lead fishing items (see Section 3.1).[447] Ingestion of a single lead weight or lead-headed jig (up to several grams of lead) is sufficient to provide a bird with a lethal dose of lead.[448]

Birds that select grit to aid mechanical digestion of food within a muscular gizzard (*e.g.* members of the *Anatidae*) are also more likely to ingest lead, as are piscivorous birds that may also ingest lead weights when consuming fishing bait with the fishing line and weight still attached (see Section 3.3).

To date, lead poisoning from the ingestion of lead angling weights has been reported in several bird species within Annexes I & II of the Convention of Migratory Species (Table 2). The most widely reported species known to be affected by ingesting lead fishing weights are the piscivorous common loon (most important cause of death in North America)[449] and the mute swan (*Cygnus olor*) (major cause of mortality in the UK during the 1970s-1980s).[450] Both species inhabit fished waterbodies and mute swans also take grit to aid digestion, increasing their susceptibility to lead poisoning. Rates of lead poisoning in mute swans are related to the abundance of lead weights in river sediments rather than the abundance of weights on river shores, indicating that swans mostly ingest weights whilst foraging in sediments.[451]

Lead fishing weights have also contributed to lead poisoning mortality in numerous other species of swans, ducks, geese, cranes, pelicans and cormorants (see Table 2).[452]

4.3.2. Population impacts

Whilst there is no doubt that lead poisoning from ingested fishing weights can cause direct mortality and sub-lethal impacts, relatively little is known about its effects on bird populations.[453] Some evidence

[447] Scheuhammer, et al., *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife*; Sears, J. 1988. Regional and seasonal variations in lead poisoning in the mute swan *Cygnus olo*r in relation to the distribution of lead and lead weights, in the Thames area, England. *Biological Conservation* 46 (2): 115-134.

[448] Ibid; and Pokras and Chafel, *Lead toxicosis from ingested fishing sinkers in adult common loons (Gavia immer) in New England*.

[449] Scheuhammer, et al., *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife;* Stone, W.B., and J.C. Okoniewski. 2001. Necropsy findings and environmental contaminants in common loons from New York. *Journal of Wildlife Diseases* 37 (1): 178-184.

[450] Birkhead, M., and C. Perrins. 1985. The breeding biology of the mute swan Cygnus olor on the River Thames with special reference to lead poisoning. *Biological Conservation* 32 (1): 1-11.

[451] Sears, *Regional and seasonal variations in lead poisoning in the mute swan Cygnus olor in relation to the distribution of lead and lead weights, in the Thames area, England*.

[452] See, for example, Franson, C.J., S.P. Hansen, T.E. Creekmore, C.J. Brand, D.C. Evers, A.E. Duerr, and S. DeStefano. 2003. Lead fishing weights and other fishing tackle in selected waterbirds. *Waterbirds* 26 (3): 345-352; Franson, C.J., and M.R. Smith, 1999. Poisoning of wild birds from exposure to anticholinesterase compounds and lead: diagnostic methods and selected cases. Paper read at Seminars in Avian and Exotic Pet Medicine; US Environmental Protection Agency. 1994. Lead fishing sinkers: response to citizens' petition and proposed ban. *Federal Register* 59: 11122-11143; Goode, D.A. 1981. Lead poisoning in swans. In *Report of the Nature Conservancy Council's Working Group, Huntingdon, England*.

[453] Scheuhammer, et al., *Lead fishing sinkers and jigs in Canada: Review of their use patterns and toxic impacts on wildlife.*

does, however, exist: for instance, a ban on lead weights between 0.06g and 28.36g in the UK in 1986 was thought to have caused a reduction in mute swan mortality and a subsequent increase in numbers.[454]

Long-term studies indicate that 22-53 per cent of reported adult common loon mortality in North America is attributable to lead weight and jig ingestion and adult mortality from lead poisoning is suspected to be a potential contributing factor limiting population growth in New England.[455] Suspected cases of lead poisoning from fishing weights have also been reported in threatened species such as whooping cranes (*Grus americana*).[456]

Furthermore, it is likely that the true impacts of lead poisoning on birds are greatly underestimated for a number of reasons. For example, most reports of lead weight poisoned birds have stemmed from their opportunistic discovery rather than through active surveillance and it is therefore likely that many poisoned birds are missed.[457]

Figure 1: Gaps in literature on effects of lead on migratory birds

- Mapping of lead ammunition and fishing weight use and its intensity of use, in relation with at risk species would allow predictions of exposure and poisoning even where surveillance data are lacking. Which species are most likely to be affected by lead?
- It would be valuable to understand hunting/shooting and fishing patterns/practices, plus ammunition usage, globally in relation to migratory bird habitats.
- Assess level of compliance with legislative and voluntary processes related to lead ammunition and lead fishing weights for risk evaluation. How widespread is monitoring of efficacy of legislative and voluntary processes?
- Better surveillance and updated knowledge of ingestion rates and prevalence of embedded shot, plus further research on sub-lethal effects, would help quantify population level impacts.
- What is the current status of activities using lead fishing weights globally? Is there increasing or decreasing use? Are there areas where this is likely to increase?

5. Conclusion

Lead is highly toxic to birds, causing mortality at higher concentrations, and a range of sub-lethal impacts at lower levels. Wherever there is anthropogenic use of lead which is available to migratory birds, poisoning can potentially occur. Thus, it should be noted that although surveillance and research reports on lead poisoning from most sources are mainly from North America and Europe, this is unlikely

[454] Perrins, C.M., G. Cousquer, and J. Waine. 2003. A survey of blood lead levels in mute swans *Cygnus olor*. *Avian Pathology* 32 (2): 205-212; Kirby, J., S. Delany, and J. Quinn. 1994. Mute swans in Great Britain: a review, current status and long-term trends. In *Aquatic Birds in the Trophic Web of Lakes*: Springer.

[455] D. Major, USFWS, pers comm. in: Scheuhammer, *et al*., 2003.

[456] Snyder, S.B., M.J. Richard, R.C. Drewien, N. Thomas, and J.P. Thilsted. 1991. Diseases of whooping cranes seen during annual migration of the Rocky Mountain flock. In *Proceedings American Association of Zoo Veterinarians*, edited by E. Junge. Media, Pennsylvania.

[457] AEWA, *Literature review: effects of the use of lead fishing weights on waterbirds and wetlands*.

to reflect distribution of the problem. Significant morbidity or mortality of threatened species from lead poisoning (whatever the source) is always a cause for conservation concern.

Lead poisoning, whether primary or secondary, through ingestion of shot and bullets has been recorded in at least 20 countries with most reports coming from North America and Europe. However, lead poisoning in migratory birds can be expected to occur wherever lead ammunition is used for shooting (whether hunting, target shooting or for military purposes). It follows that wherever lead shot is used, it will accumulate within the environment and the degree of contamination will be proportional to the intensity of use.

Accepting possible reporting biases, wildfowl and raptors appear more substantially affected by poisoning from lead ammunition than other groups of birds and losses can be high. Population level effects are difficult to quantify for a number of reasons, including lack of robust surveillance data and gaps in our knowledge regarding ingestion rates and subsequent survival. Sub-lethal impacts, such as those on breeding performance and predation avoidance are particularly difficult to quantify. For most countries there are also gaps in our knowledge regarding the effectiveness, or otherwise, of restrictive regulations and use of the non-toxic alternatives.

The effects of lead poisoning from fishing weights on migratory birds are restricted to certain susceptible species and to certain geographical areas where discarded and lost weights are available. In principle, most birds feeding in currently or historically fished waterbodies, or near-shore soils and sediments are at risk of being exposed to and ingesting lead, including a number of migratory species. Species with a muscular gizzard which are likely to feed in areas exposed to lead fishing weights are therefore most at risk of suffering from lead ingestion and poisoning. For these reasons, lead poisoning arising from fishing weights has been widely reported in waterbirds. Although it is difficult to assess the population-level effects of such poisoning, there is some evidence for effects in species that are particularly susceptible to the availability of fishing weights such as the mute swan and the common loon.

Appendix I: Potential toxins to migratory birds (priority given to lowest score)

Toxin	Criteria				Score
	Seriousness of effects 1 = direct population level effect 2 = direct effects, but unlikely to have population level effect 3 = indirect effects	**Breadth of effects** 1= widespread impact on wide variety of species 2 = impact over some regions, but not global and/or impact on narrow range of species over wider areas or significant range of species in limited geographic area 3 = local impact on few species	**Tractability of the problem** 1 = within next 5 years 2 = medium term 3 = only in long-term with significant investment	**Certainty of evidence** 1 = certain 2 = some certainty, but lack of clarity in some areas 3 = significant uncertainty	**lowest score= highest priority**
Insecticides					
Organochlorines	1	2	1	1	5
Organophosphates	1	1	1	1	4
Carbamates	1	1	1	1	4
Neonicotinoids	3	1	3	3	10
Pyrethroids	3	3	3	3	12
Rodenticides					
First generation anticoagulants	2	1	1	2	6
Second generation anticoagulants	1	1	1	1	4
Strychnine	2	2/3	2	2	8
Non-steroidal anti-inflammatory drugs/veterinary pharmaceuticals					
Diclofenac	1	2	1	1	5
Ketoprofen	1	2	1	1	5
Heavy metals					
Lead	1	1	2	1	5
Arsenic, Mercury, Cadmium, Cyanide	2	2	2	1	7
Diffuse pollutants					
PCBs	1	1	2	1	5
Dioxins and dibenzofurons	2	3	2	3	10
PBDEs	2	2	2	2	8
PFCs and PFAs	1	3	3	3	10
PCNs	unknown	unknown	3	3	N/A

Appendix II: Other lead sources of risk to migratory birds

There are a number of other less common sources of lead which can, in some situations, affect migratory birds.

Industrial sources

In some areas lead pollution from industrial sources, such as mining and smelting activities, is the primary source of lead intoxication for migratory birds. When feeding in industrially-contaminated aquatic environments birds ingesting sediments and vegetation are exposed to the highest levels of lead and are at the greatest risk. As a consequence, wildfowl are especially vulnerable.[458] Examples include heavy contamination from long-term mining and smelting in Idaho, USA, which has led to highly elevated blood lead levels and high mortality rates in wildfowl species.[459] Similar poisoning has been reported in the mining regions of Oklahoma, Kansas and Missouri, USA, where mallards have been found with elevated tissue lead concentrations.[460] Although sediment may make up as little as 2 per cent of the wildfowl "diet" (*eg,* wood duck), this represents a principal route for exposure to an environmental contaminant that might not be sequestered by plants or invertebrates.[461]

In areas of industrial contamination, those birds feeding on invertebrates and vertebrates have a lower risk than those feeding on vegetation and in sediments. Nonetheless, industrial lead pollution can still cause mass mortality events via ingestion of lead contaminated invertebrates. In the UK, alkyl lead compounds have been known to be discharged into nearby waterways, sequestered by invertebrates then consumed by waterbirds leading to mass mortality events.[462]

Vertebrate prey cause the lowest risk because nearly all their lead burden is bound up in bone and is therefore relatively unavailable following ingestion.[463]

As well as large impact mass mortality events, industrial lead pollution and increased tissue lead concentrations can cause sub-lethal effects and impair biological functions. Cases of industrial lead pollution causing elevated blood lead levels have been recorded in: osprey (*Pandion haliaetus*,[464]

[458] Beyer, W.N., D. Day, M.J. Melancon, and L. Sileo. 2000. Toxicity of Anacostia River, Washington, DC, USA, sediment fed to mute swans (*Cygnus olor*). *Environmental toxicology and chemistry* 19 (3): 731-735.

[459] Spears, B.L., J.A. Hansen, and D.J. Audet. 2007. Blood lead concentrations in waterfowl utilizing Lake Coeur d'Alene, Idaho. *Archives of Environmental Contamination and Toxicology* 52 (1): 121-128; Sileo, L., L.H. Creekmore, D.J. Audet, M.R. Snyder, C.U. Meteyer, J.C. Franson, L.N. Locke, M.R. Smith, and D.L. Finley. 2001. Lead poisoning of waterfowl by contaminated sediment in the Coeur d'Alene River. *Archives of Environmental Contamination and Toxicology* 41 (3): 364-368; Blus, L.J., C.J. Henny, D.J. Hoffman, L. Sileo, and D.J. Audet. 1999. Persistence of high lead concentrations and associated effects in tundra swans captured near a mining and smelting complex in northern Idaho. *Ecotoxicology* 8 (2): 125-132.

[460] Beyer, W.N., J. Dalgarn, S. Dudding, J.B. French, R. Mateo, J. Miesner, L. Sileo, and J. Spann. 2004. Zinc and lead poisoning in wild birds in the Tri-State Mining District (Oklahoma, Kansas, and Missouri). *Archives of Environmental Contamination and Toxicology* 48 (1): 108-117.

[461] Beyer, W.N., L.J. Blus, C.J. Henny, and D. Audet. 1997. The role of sediment ingestion in exposing wood ducks to lead. *Ecotoxicology* 6 (3): 181-186.

[462] Osborn, D., W.J. Every, and K.R. Bull. 1983. The toxicity of trialkyl lead compounds to birds. *Environmental Pollution Series A, Ecological and Biological* 31 (4): 261-275.

[463] Henny, C.J., L.J. Blus, D.J. Hoffman, R.A. Grove, and J.S. Hatfield. 1991. Lead accumulation and osprey production near a mining site on the Coeur d'Alene River, Idaho. *Archives of Environmental Contamination and Toxicology* 21 (3): 415-424.

[464] Ibid.

American woodcock (*Scolopax minor*),[465] tree swallows (*Tachycineta bicolor)*,[466] American robins (*Turdus migratorius*) and northern cardinals (*Cardinalis cardinalis*).[467]

Paint

Lead has been used extensively historically in paints as both a pigment but also to facilitate quicker drying in oil-based paints. Historically, white lead (basic lead carbonate) was the most common form to be used but other colours were produced using other lead compounds (*e.g.* chromates or oxides). Human health concerns have reduced or eliminated the production and use of lead paint in many parts of the world.

As leaded paints have been mainly used in domestic and industrial environments there is a low risk to migratory birds, indeed few species within Annexes I and II of the Convention of Migratory Species (Table 2) have been reported as poisoned from lead paint. Nonetheless, there are reports of poisoning from this source in a group of California condors roosting on, or in the vicinity of, an inactive fire lookout tower with deteriorating lead-based paint.[468] Also, this source has caused serious conservation concerns, for bird populations at the Midway Atoll in the Central Pacific, where old buildings have been a source of substantial lethal and sub-lethal poisoning in albatross chicks, accounting for 12 per cent of chick deaths between 1993 and 1995.[469] By contrast the adults, with greater feeding experience, feed at sea and have minimal to no lead exposure.[470] Although it is well established that leaded paint is the main cause of lead poisoning of birdlife at the Midway Atoll, the impact on population levels is less clear.

[465] Scheuhammer, A.M., C.A. Rogers, and D. Bond. 1999. Elevated lead exposure in American woodcock (Scolopax minor) in eastern Canada. *Archives of Environmental Contamination and Toxicology* 36 (3): 334-340.

[466] Blus, L.J., C.J. Henny, D.J. Hoffman, and R.A. Grove. 1995. Accumulation in and effects of lead and cadmium on waterfowl and passerines in northern Idaho. *Environmental Pollution* 89 (3): 311-318.

[467] Beyer, et al., *Zinc and lead poisoning in wild birds in the Tri-State Mining District (Oklahoma, Kansas, and Missouri)*.

[468] Finkelstein, M.E., D.F. Doak, D. George, J. Burnett, J. Brandt, M. Church, J. Grantham, and D.R. Smith. 2012. Lead poisoning and the deceptive recovery of the critically endangered California condor. *Proceedings of the National Academy of Sciences*, 109 (28): 11449-11454.

[469] Work, T.M., M.R. Smith, and R. Duncan. 1998. Necrotizing enteritis as a cause of mortality in Laysan albatross, *Diomedea immutabilis*, chicks on Midway Atoll, Hawaii. *Avian Diseases* 42 (1): 1-5; Sileo, L., and S.I. Fefer. 1987. Paint chip poisoning of Laysan albatross at Midway Atoll. *Journal of Wildlife Diseases* 23 (3): 432-437.

[470] Finkelstein, M.E., R.H. Gwiazda, and D.R. Smith. 2003. Lead poisoning of seabirds: Environmental risks from leaded paint at a decommissioned military base. *Environmental science & technology* 37 (15): 3256-3260.