Shore, Richard F.; Taggart, Mark A. 2019. **Population-level impacts of chemical contaminants on apex avian species.**

Population level impacts of chemical contaminants on apex avian species

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Abstract

We undertook short case studies of how: (i) dieldrin and DDT affected populations of the peregrine falcon (*Falco peregrinus*) and other birds of prey in Britain; (ii) diclofenac impacted vulture populations across in SE Asia. In both cases, high levels of [contaminated-mediated] acute mortality largely drove the population crashes. Impaired, or naturally low, rates of reproduction likely limited recovery rates. The studies illustrate the huge, long-lived impacts that contaminants can have on bird populations. They changed our scientific understanding of the importance of different exposure routes and influenced how we now conduct monitoring and risk assessment. They also demonstrated the value of long-term population monitoring and archived specimens for identifying the causal factors and mechanisms behind the population crashes.

Keywords: organochlorines, pharmaceutical, population crash, monitoring, raptor, vulture

Declaration of interest: None

Introduction

The impacts of chemical contaminants on apex predators, particularly birds, have been well studied over the last 50 years. This is partly because exposure to a diverse range of contaminants, including toxic heavy metals (such as lead), insecticides, biocides and pharmaceuticals, has resulted in lethal poisoning and/or impaired reproduction that, in some cases, has directly led to global population declines [1-6]. Detection of population-level effect is arguably easier in birds than for many other animals. This is because many species are relatively easy to observe and count and there is typically a cadre of professional and amateur ornithologists with an interest in ringing birds, conserving them and undertaking population counts.

In this short paper, we describe two of the clearest case studies in which exposure to chemicals has caused major population declines in apex avian species. The first is the classic case of the impacts that organochlorine (OC) pesticides, particularly cyclodienes (such as dieldrin) and DDT, had on birds of prey, and in particular the peregrine falcon (*Falco peregrinus*). The second is much more recent and catastrophic impact of diclofenac on vultures in SE Asia. We describe the factors that led to population declines in both cases and outline what general lessons have or can be drawn from them, and the role population monitoring played in detecting effects in particular.

Case Study 1: Organochlorine pesticides and the peregrine falcon in Britain

The impacts on wildlife during the late 1940s and 1950s that extensive use of synthetic pesticides were having on the environment, including birds, was highlighted to the world through Rachel Carson's now classic book "Silent Spring" [7]. While Carson was based in America, similar concerns were also being voiced in Britain over the effects of OC pesticides. The realisation that peregrine falcon numbers were declining in Britain was due to a 1961-2 survey, led by Derek Ratcliffe, which measured occupancy and breeding success of peregrines in known territories. This was initiated, somewhat ironically, because of concerns from pigeon fanciers that peregrine numbers were rising and falcons were killing increasing numbers of pigeons. The survey in fact showed that occupation of breeding territories by peregrines was less than half that of the 1930s average and only 21% of occupied territories were producing young; productivity in many areas had become too low to maintain population numbers [3]. There was no evidence of natural mortality factors accounting for this national-scale decline and attention began to focus on the possible toxic effects of cyclodiene pesticides used as seed dressings, and in particular dieldrin (a seed dressing in its own right and a metabolite of another compound, aldrin). These dressings were already reported to have caused the deaths of granivorous birds feeding in treated fields and there were also reports of deaths of predatory birds and mammals [3,8] in and around agricultural fields. Analysis of addled eggs and carcasses showed that peregrines were exposed to dieldrin and other OC pesticides [9] while spatial and temporal analysis demonstrated that the extent of decline in populations was positively correlated with the prevalence of arable farming and with the onset of use of seed dressings [3]. Such associations were also found in other similar species such as the sparrowhawk (*Accipiter nisus*) [2,10-12].

The role of dieldrin in causing population declines in peregrines and other species was complicated by simultaneous exposure of the birds to DDT which was used extensively around the word as an insecticide. DDT causes eggshell thinning, although the extent of thinning can vary markedly between species [13]. Thinning results in structurally weaker eggs and impaired reproductive success. Through measurements of the shell index of failed eggs and eggs held in museum and other collections, Ratcliffe demonstrated that eggshell thinning in peregrines began as far back as 1947 [3], reflecting the post-war surge in use of the compound. There was a similar thinning in sparrowhawk eggs (Figure 1) and other species that began between 1946 and 1952. Chemical analysis also demonstrated that DDE, the main metabolite of DDT, was detectable in peregrine shells from 1947 onwards [14]. The eggshell thinning seen in Britain was also extensively reported in other countries and regions, especially North America [15,16].

The growing evidence that OC pesticides were impacting wildlife led the UK Government Advisory Committee on Pesticides to advise that their agricultural use should be limited. This led to increasing restrictions through the 1960s on the use of cyclodienes as seed dressings, with an eventual ban in Britain in 1975. DDT use was also increasingly restricted although there was not a complete ban o in Britain until the 1980s. Monitoring of pesticide residues in carcasses of birds found dead and in failed eggs demonstrated that exposure levels in peregrines and other species declined following the bans, eventually falling below toxic levels [3,17], although trace amounts of both compounds still remained detectable in eggs and carcasses 20-30 years later [18].

The coupling of dieldrin-induced mortality and DDT-impaired reproduction in peregrine, sparrowhawks [2] and other species was a powerful combination, with two key ecological drivers (survival and recruitment of young) markedly impaired. The consequence was a drastic population crash. This was mainly been attributed to dieldrin-induced mortality and the extent to which DDT-mediated impairment of reproduction may have increased the rate of decline is unclear. However, poor recruitment may well have slowed the recovery of peregrines and other species following the banning of dieldrin. Eggshell thickness in peregrines, although recovering, was still below pre-DDT levels in 1980 and it was only in the late 1980s that population numbers were similar to those seen in the 1930s [19,20]. The Predatory Bird Monitoring Scheme [21] still tracks eggshell thickness in the similarly affected sparrowhawk and eggs only fully recovered to pre-DDT levels in in the late 1990s (Figure 1), slightly lagging behind the recovery in numbers [22]

Case study 2: New threats and pathways: Old World vultures and modern veterinary medicines

In the 1980s, one species (*Gyps bengalensis*) of Old World vulture was considered the most abundant large raptor on Earth [23] – perhaps numbering some 40 million individuals – yet, by 2000, it was being listed by the IUCN as Critically Endangered. Surprisingly, the complete collapse of this and two other South Asian vulture species (*G. indicus* and *G. tenuirostris*) was, in 2004, clearly linked to an entirely new chemical threat and an almost completely overlooked exposure pathway.

Collapsing Old World vulture populations across South Asia were first highlighted in the late 1990s by field conservationists working in India [24] and Pakistan. Geographically extensive and rapid declines in three *Gyps* species were being observed – and the driver behind this was initially very elusive [25,26]. Then in 2004, Oaks presented his team's findings from Pakistan [6], and identified that the wholly unexpected cause of the decline was exposure to a common veterinary pharmaceutical, diclofenac. This non-steroidal anti-inflammatory drug

(NSAID) was widely used as a veterinary drug but had not been considered an environmental toxicant of any note.

Diclofenac had emerged onto the Indian sub-continent as a low-cost off-patent veterinary drug in the mid-1990s, and had quickly ascended to become the NSAID of choice for livestock treatment. Very widely used to treat mastitis, lameness, inflammation, etc., by 2006 diclofenac was detectable as a residue in ~10% of livestock carcasses available to vultures across India [27]. However, it was also extremely nephrotoxic to *Gyps* [6], with an LD_{50} of just 0.098-0.225 mg kg^{-1} bw (in *G. bengalensis*; [28]. Further, very few carcasses (just 0.13–0.75%) had to contain lethal residue levels to drive the mortality rates being observed [29]. From an avian ecotoxicology perspective, a “perfect storm” of sorts had unpredictably occurred (Figure 2).

Since diclofenac was recognised as the principal driver behind vulture declines in South Asia, it has been a race to effectively halt and reverse this trend - and literally “*Save Asia’s Vultures from Extinction*” (www.save-vultures.org). Certain critical actions have occurred, including: (a) several large *Gyps* captive breeding (and ultimately release) programmes have been instigated in South Asia; (b) diclofenac has been effectively banned as a veterinary drug (starting in 2006) across the region; and (c) a veterinary NSAID proven to be “*vulture safe*” (at normal and elevated exposure levels) has been identified – meloxicam [30]. These actions, alongside other intensive efforts, may now [31] be leading to a cessation in declines and/or some small signs of recovery for *G. bengalensis* – but critically, numerous wider questions regarding NSAID safety toward scavenging birds still remain.

Whilst Old World *Gyps* vultures are now known to be particularly susceptible to diclofenac poisoning, [32] presented the first indication (from a survey collating data from 870 birds from 79 species) that a much wider suite of NSAIDs (including carprofen, flunixin, ibuprofen and phenylbutazone) may also be nephrotoxic to a range of avian scavengers (globally). Since then, safety trials have now confirmed nephrotoxicity at plausible field exposure levels in *Gyps* due to ketoprofen [33], carprofen [34] and aceclofenac [35] – whilst very worryingly, both nimesulide (in South Asia; [36] and flunixin (in Europe; [37]) have now been clearly linked to wild *Gyps* mortalities on two continents. Further, concern now also exists regarding potential impacts on what some may consider to be more enigmatic species such as eagles, following the death of two steppe eagles (*Aquila nipalensis*) in India in association with diclofenac [38]. In a concerning twist to this story – whilst SE Asia was extremely quick to ban diclofenac across that region to protect vultures – and despite immense efforts in Europe in recent decades to conserve European vulture species, diclofenac was recently permitted to emerge onto the veterinary market in Spain, Portugal and Italy.

Discussion

These case studies demonstrate the massive and long-lasting impact that environmental contaminants can have on bird populations. Commonalties include the facts that acute mortality was a major ecological driver and that population recovery was/is limited by DDT-impaired or naturally low (vultures) rates of reproduction and recruitment. The impact of OCs on wildlife spawned the birth of "ecotoxicology" and provided major insights into the importance of persistence, bioaccumulation and toxicity (PBT) as key characteristics of chemicals associated with environmental risk; PBT remains a cornerstone for chemical risk assessment today. The OC "experience" also led to the development of a specific bird reproduction test (that includes measurement of eggshell thinning) as part of pre-registration pesticide testing regimes, and provided a clear demonstration of how exposure to chemical mixtures can exacerbate impacts on populations. It also led to the widespread practice of chemical and eggshell thickness monitoring in predatory birds [39] , these species being used as environmental sentinels to help detect the emergence of new contaminant risks [40]. The pioneering work of Ratcliffe also demonstrated the value of analysing archived historical samples to understand the mechanisms by which chemicals exert population effects and to provide proof of synchrony between chemical use and such effects. The Asian vulture case has highlighted again just how easily a new environmental contaminant can quickly and unpredictably emerge and have global population level impacts on wild species by causing direct mortality. This occurred despite huge advances in knowledge and experience that have advanced chemical risk assessment in the last 50 years. It has also highlighted the importance of assessing risk across a diverse range of ecological traits and exposure pathways. The risk to scavengers and decomposers feeding on the carcasses and waste of medically-treated animals (including humans) has largely been overlooked, but this is likely one of the most significant pathways by which wildlife are exposed to for pharmaceuticals [41]. The loss of vulture populations has also highlighted how contaminant-induced population declines can have severe knock-on effects, such as widescale loss of invaluable and long-standing ecosystem services. In the case of *Gyps* vultures, a rapid and very efficient carrion disposal system which has helped reduce the occurrence and spread of disease [42] for decades has now been almost completely lost across South Asia.

Would ongoing and/or increased population monitoring have prevented the population crashes described in these case studies? Declines may have been detected more rapidly but there are many other factors that can cause bird populations to fluctuate [43]. Even with ongoing population monitoring in place, the speed of the OC and diclofenac-mediated declines would probably still have resulted in population crashes within the time it would have taken to attribute cause and initiate mitigation. Remarkably, the Asian vulture declines were recognised, causality determined, and government bans implemented all within about a decade, yet Old World *Gyps* populations in South Asia still plummeted from 10s of millions to precariously low levels (just thousands to 10s of thousands) within that timeframe. Clearly, prevention rather than remediation is paramount and requires well-founded risk assessment that, as the vulture case study demonstrates, still evidently requires significant improvement. Ongoing monitoring is however likely to provide early alerts of slower contaminant-induced declines, and there clearly remains high value in linking spatial and temporal population trends to information on contaminant exposure. Detected associations are often correlative and not proof of causality but they add significantly to the overall "weight of evidence" regarding both risk and impact. In all but the most clear-cut circumstances, decisions as to whether to restrict or ban the use of certain chemicals, because of their environmental impact, will typically rely on such weight of evidence.

Acknowledgement

This work was supported by the Natural Environment Research Council [NERC grant reference number NEC06550]

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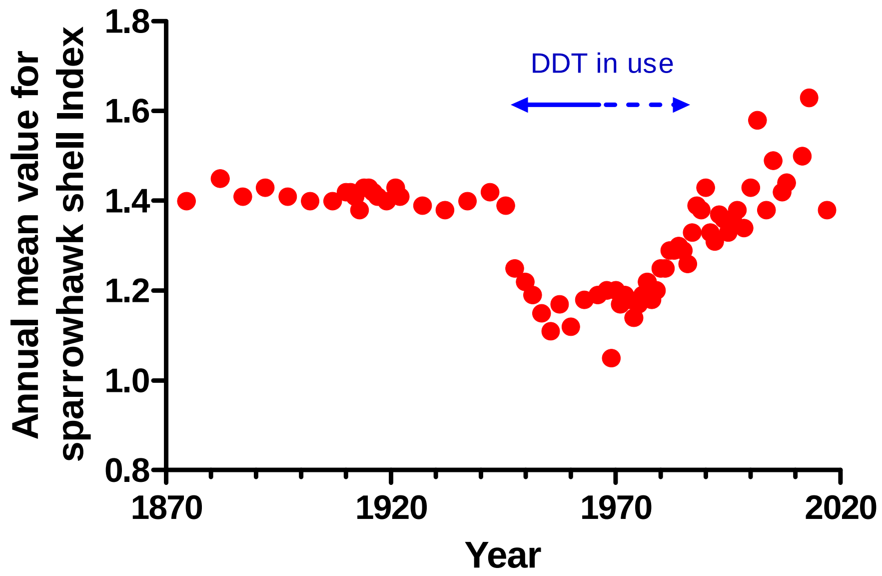

Figure 1. Mean annual eggshell index for sparrowhawks in Britain. Data are updated (and statistically summarised) from those published by Newton ([2,44]

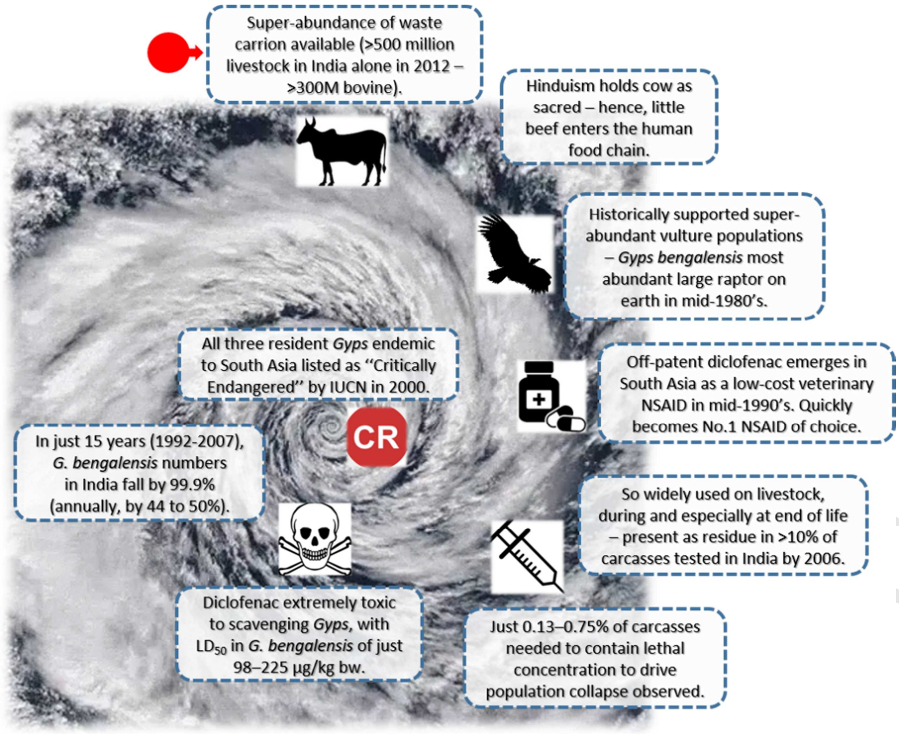

Figure 2. Some key factors (clockwise from the top) relevant to the collapse of scavenging *Gyps* vulture populations in South Asia due to diclofenac.

Highlights

- Contaminants can exert massive adverse effects on bird populations
- We illustrate this by reviewing the effects of cyclodiene and other organochlorine (OC) pesticides on birds of prey and the effect of diclofenac on Asian vulture populations
- In both cases, acute mortality drove population crashes and with slow (multi-decadal) rates of recovery
- Long-term population monitoring and archived specimens were crucial for identifying the causal factors and mechanisms behind the population crashes
- Risk assessment and monitoring methods improved following the OC “experience” in the 1950/60s but were not sufficient to pre-empt diclofenac –mediated effects in the 1990s
- Diclofenac demonstrated how contaminants can impact wildlife populations and the ecosystem services they provide