Supporting Information for: Direct evidence of poison-driven widespread population decline in a wild vertebrate

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Appendix S1. Summary of the accumulated evidence on the effects of toxicants, such as the organochlorine insecticide DDT (i.e. dichlorodiphenyltrichloroethane), lead ammunition, and the anti-inflammatory drug diclofenac, on wild species. These toxic substances are known to be the cause of large population declines of falcons, condors and vultures, respectively. The description of these paramount cases aims to illustrate how most evidence on the impact of poisoning on wildlife to date is retrieved at individual level, from which the potential effects of toxic compounds at population level are indirectly inferred, e.g. using demographic modelling or deductive reasoning. In contrast, our study moves forward in gathering compelling evidence on the population-level effects of toxicants, by demonstrating a straightforward relationship between toxic-induced individual mortality and population change overlapping in space and time across a large number of locations.

1. DDT and the decline of the peregrine falcon populations in Europe and North America

Organochlorine pesticides, including DDT (i.e. dichlorodiphenyltrichloroethane), are thought to be the most harmful pesticides for wildlife, especially for birds of prey (1, 2). Although synthesized in the XIX century, DDT was not discovered as an insecticide until 1939 (Fig. S1). It was first used to control mosquito outbreaks during the Second World War, and, from 1946 onwards, DDT was widely used against agricultural pests, as well as to kill mosquitoes, in an effort to eradicate malaria (2).

DDT is a fat-soluble, persistent compound that biomagnifies up in the food chain, passing from preys to predators, and thus tends to bioaccumulate, reaching higher concentrations in predators; it is also toxic to fishes (1, 2). Inside the animal's body, DDT biodegrades to DDE

(i.e. dichlorodiphenyldichloroethylene), which is thought to be responsible for eggshell thinning, and subsequent breakage, in birds. Because of their position at the top of the food chain, raptors were the species most affected by DDT (1). Indeed, the populations of bird-eating species such as the peregrine falcon (*Falco peregrinus*) and the sparrowhawk (*Accipiter nisus*) showed the highest declines attributed to DDT (1). In the 1950s and 1960s, peregrine falcon populations plummeted in many parts of western Europe and North America. In the 1960s, the species had already disappeared in the eastern half of the United States; in Great Britain the breeding population in the early 1960s (estimated in 241 occupied territories in 1962) had declined 50 % relative to the population of 1930-1939; in Southern England the decline was of 92 % (3). The Swedish peregrine population crashed from 350 pairs in the 1950s to 15 pairs in 1975 (i.e. >95 % decline) (4), and in West Germany, from about 400 pairs in 1950, to 40-50 pairs in 1973 (5).

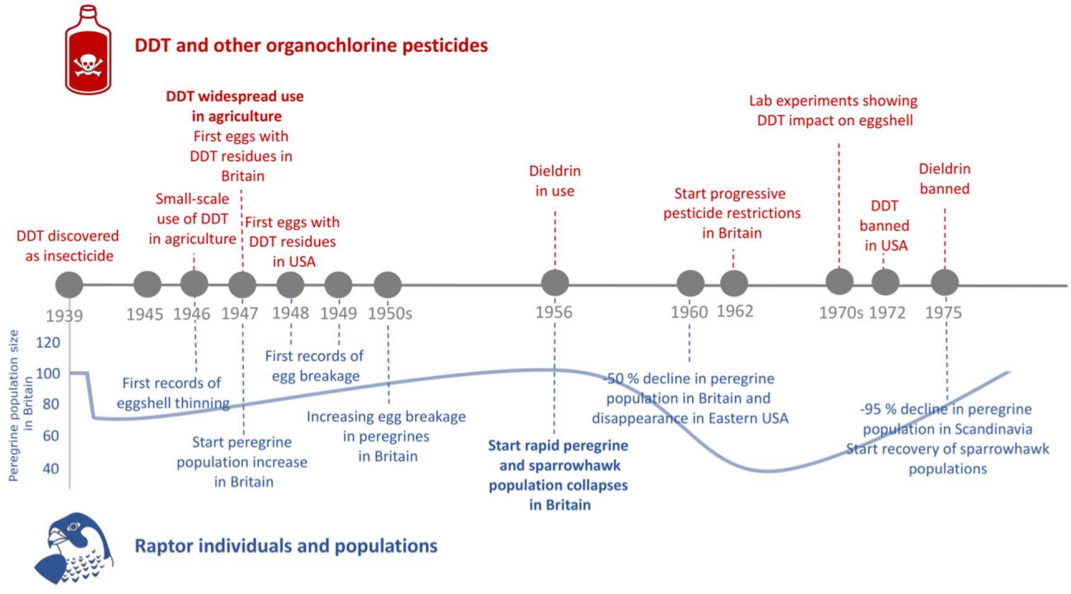

Figure S1. Timeline showing the use of DDT and other pesticides (in red at the top) and their potential impacts on raptor populations (in blue at the bottom): The population trend of the peregrine falcon in Britain is shown according to Ratcliffe (5). Illustrations of Chris Homan and Martina Krasnayová from The Noun Project.

Between 1951 and 1956, Ratcliffe (6) reported for the first time an unusual number of cases (13 out of 59 eyries) of broken eggs in peregrine nests in Britain. Later studies indicated that this phenomenon had increased in Britain from the 1950s, although the first records of egg breakage date back to 1948-1949 (6, 7). The incidence of broken eggs in peregrine nests increased over an order of magnitude, from <2.7 % in the period from 1904-1950 to 28 % in 1950-1966 (8). A similar incidence of egg breakage had also been found in the sparrowhawk in Britain (8). The increase in the egg breakage led researchers to examine any possible change in the thickness of eggshells, which, in the peregrine and the sparrowhawk, decreased significantly since 1946 in both Britain (8) and the United States (9). This synchronic change in eggshell thickness in both regions just after the widespread usage of DDT directly pointed to pesticides in general, and DDT in particular, as primary contributors. Hickey & Anderson (9) studying herring gulls and Cade *et al.*, (10) in Alaskan peregrine falcons found a negative relationship between the shell thickness and DDE residues in eggs. Studies in captive birds demonstrated that a diet containing DDE in environmentally realistic doses caused the thinning of the eggshells in American kestrels (*Falco sparverius*) (11). Accumulated evidence pointed to the DDT as responsible for eggshell thinning and egg breakage in different species, but some authors still raised doubts, arguing that eggshell thinning started too soon, i.e. in 1946 and 1947 in Britain and USA, respectively, just when DDT came into general use (1946-1947) (12). However, these concerns were dismissed when Peakall (13) was able to find DDE residues in the dried membranes of peregrine eggs collected in California between 1948 and 1950, but not in eggs collected before that date; two years later similar studies were conducted in Britain by Peakall *et al.*, (14) who found DDE residues in peregrine eggs from 1946 and 1947.

The mechanism by which DDT affected eggshell was elucidated in the late 1960s and early 1970s using captive non-raptorial birds under laboratory conditions. DDE produces a decrease in calcium content in eggshells thorough inhibiting the carbonic anhydrase and Ca-ATPase activity, enzymes involved in the transport of calcium in the avian shell-forming gland (15-17). This in turn produces eggshell thinning, which occurs only 4 days after the female ingests DDE (40 ppm); the decrease in eggshell thickness reduces both breaking strength and pore density in the shells (18), which leads to egg breakage and breeding failure (19).

Therefore the evidence obtained during decades allowed to elucidate how DDT could be affecting reproduction in birds in the field: i) organochlorine residues are incorporated by parent birds when feeding upon contaminated preys with these pesticides; ii) once in the bird's body, DDT biodegrades to DDE, which alters the calcium transport in the shell-forming gland, iii) this produces a decrease in calcium content in the eggshell, iv) eggshell thinning, v) egg breakage and, finally, vi) breeding failure.

Have DDT-induced effects on eggshell and breeding success caused the collapse of peregrine falcon populations in late 1950s and 1960s?

In Britain, DDT residues in peregrine eggs were present as soon as 1946 and 1947, and the decrease in the thickness of eggshell was detected from 1946 onwards (Fig. S1). However, despite the almost immediate effect of DDT on egg breakage (18), the sharp collapse in the breeding population of peregrine in Britain began long after that, i.e. in 1956 (2, 7). Even more, from the late 1940s to 1955 the British population of peregrine falcon was increasing

after the population control exerted during the Second World War (7); in 1940-1945 peregrine falcons were legally shot and their nests and eggs destroyed to avoid predation on domestic pigeons used for carrying military messages (5, 7). The decrease of the peregrine population after 1955 was so rapid (i.e. 50 % in less than 10 years) that it was suggested that adult mortality had to be involved (7). This temporal pattern of population decline matched the introduction of other organochlorine pesticides: the seed dressing cyclodienes (7). The cyclodienes aldrin, dieldrin and heptachlor cause (sub)lethal toxicity increasing mortality rates (2). Similar to the peregrine, the sparrowhawk population in Britain suffered a post-1955 collapse too, supporting the hypothesis that DDT was not the ultimate contributor to such a rapid population crash. Indeed, it was rather related to the use of dieldrin (2, 19, 20), as this pesticide was in use from 1956 and once dieldrin was definitively banned in 1975, the population of sparrowhawk rapidly recovered (19). Although some evidence suggested a positive relationship between DDT-induced eggshell thinning and decreasing populations of peregrine (21), the population changes in the peregrine and sparrowhawk observed over time do not seem to support it (at least in Britain, see above). DDT was the first pesticide subjected to environmental regulations; it was banned in the United States in 1972 (21). In Great Britain restrictions for use of DDT started in 1962 and it was completely banned in 1986.

This demonstrates that although the toxicological effects of DDT on eggshell thinning and reproduction are well-established, these effects have not been well reflected at the population level, likely because other density-dependent factors have compensated for the reduction in breeding success driven by DDT (20). Therefore, evidence linking DDT effects at the individual level (i.e. shell thinning and breeding failure) with effects at the population level (i.e. population collapse and declines) is weak or not-well supported (9, 21). Indeed, Ratcliffe

(7) stated that “the respective contributions of adult mortality and breeding failure to the peregrine population decline will perhaps never be known”. Several decades later, Walker *et al.*, (2) highlight the same idea “although it is well established that lethal toxicity [of DDT] and eggshell thinning have occurred in the field in the UK, difficulties arise in quantifying them and relating them to population change”.

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2. Lead ammunition and the extinction of the California condor in the wild

The California condor (*Gimnogyps californianus*) case study represents one of the few in the world in which a toxicant has been recognized to be the most important factor driving the global extinction of a species in the wild.

In the nineteenth century, the California condor was widely distributed along the west coast of North America, from British Columbia in Canada to the mountains of northern Baja California in México (22, 23). During the 19th and 20th centuries, however, the species suffered a drastic and rapid reduction in both range and population until its total extinction in the wild in the 1980s (Fig. S2). Rough estimates suggested a condor population of about 150 birds in the 1940s, which was further reduced to about 100 birds in the early 1960s (23). In the late 1970s, the species only consisted of about 30 birds occupying a small area in the San Joaquin Valley of California. In subsequent years the population continued declining, from 21 birds in 1982, to 5 in 1986 (22, 24). Owing to this rapid and continuing population decline, captive breeding programs began in 1982 with the capture of two condors, a nestling and an immature bird. The last wild condor was captured in 1987, and thus the entire condor population was brought into captivity. In 1987 the captive population consisted of 27 individuals (22) taken from the wild: 7 adult birds, 7 birds that were taken as nestlings or immatures and 13 full-grown birds that had been taken as eggs (23).

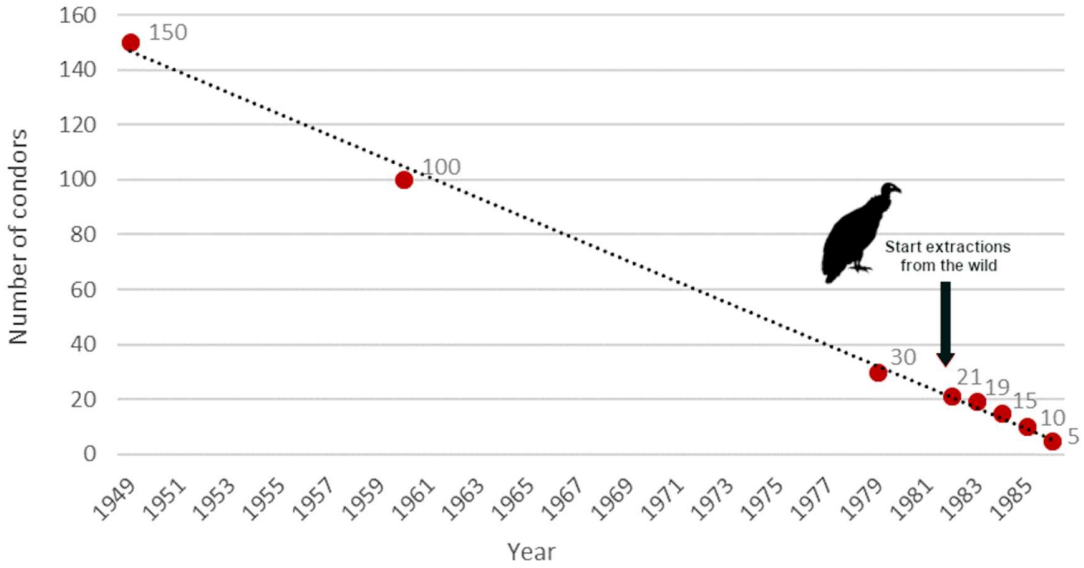

Figure S2. Decrease of California condor population over time: The collapse was so fast that wild birds were captured from 1982 onwards to save the species from extinction. Between 1982 and 1986, 15 free-flying condors died, 6 of them in the winter of 1984-85; the causes of death were only determined for 4 of these birds (i.e. 3 were poisoned by lead and 1 by cyanide) (23).

The remnant condor population in the early 1980s exhibited high mortality rates (26 %) (22) that, according to demographic models, exceeded the levels for self-sustaining populations (i.e. <10 %) (22). Three out of four free-living condors found dead in the early 1980s died by lead poisoning, and the other one by cyanide poisoning (22). While habitat loss and direct persecution (shooting, poisoning) seemed to be the main causes of decline during the 19th and early 20th centuries (25), lead poisoning was attributed as the main cause of decline of the remnant population (22, 25). This evidence was later confirmed when the captive birds were released since 1992. In the 1997-2010 period, 150 condors were released in California. Between 50 % and 88 % of these condors released each year showed blood lead levels >100 ng/mL (about 3 times higher than blood levels of pre-release condors, 30.3 ng/mL). In

addition, a mean of 20 % of the birds per year (range = 0-44 %) reached the level of clinical lead poisoning (i.e. 450 ng/mL), thus needing chelation treatment for recovery (26).

Evidence shows that condors were poisoned by ingestion of lead ammunition fragments in carcasses or gut piles of shot animals (e.g. big game, small mammals, feral hogs, coyotes, livestock) (25, 26). Isotopic composition of blood lead for many of the free-flying condors (63 %, n=110) was consistent with lead-based ammunition; other sources of lead were background environmental and lead-based paint.

The free-ranging condor population currently undergoes intensive management to limit exposure to lead. Blood lead levels are frequently measured for most birds; if lead levels are high (i.e. >450 ng/mL), the birds receive chelation therapy in captivity and are released once the lead levels are deemed nominal. Demographic models showed that, without this intensive management to limit high lead levels among the free-ranging condor population, the population in California would decline from 150 individuals to 22 individuals in 11 years if mortality is assumed at blood lead levels of 1,000 ng/mL, or in 61 years, assuming mortality at 3,000 ng/mL (26).

Overall, evidence of the negative individual-level effects of lead on condors seems to be clear (21). However, how these individual-level effects translate to population-level effects is based on a deterministic, age-based demographic model (26), that indirectly shows the pervasive impact of lead on the California condor population level, but there is not direct evidence linking effects of the toxicant between individual and population levels.

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3. Toxic impact of the anti-inflammatory drug diclofenac on vulture populations in Asia

The rapid collapse of the Asian vulture populations (>90 % of decline in less than 10 years), which led toward the brink of extinction to three formerly common *Gyps* species, namely, the oriental white-rumped vulture (*Gyps bengalensis*), the long-billed vulture (*G. indicus*), and the slender-billed vulture (*Gyps tenuirostris*) (27-29), is frequently highlighted as one of the most dramatic episodes of wildlife poisoning in the past few decades. Investigations revealed the anti-inflammatory diclofenac as the cause of these population declines, whereby vultures were contaminated by consuming livestock carcasses treated with this drug (30, 31).

The first vulture population declines were reported in the Keoladeo National Park in Rajasthan, northern India. Between 1986 and 1999, the resident populations of the oriental white-rumped and the long-billed vultures plummeted by 95 % (from 1,800 to 86 individuals) and by 96 % (from 816 to 25 individuals), respectively (27). The white-rumped vulture disappeared as a breeder in the park (from 204 nests in the 1985-1986 breeding season to none in 1999-2000 and 2000-2001) (28).

Sharp vulture population declines were also reported in other regions of India (28). Between 1991-1993 and 2000, the number of white-rumped vultures counted in road transects across the country declined by 95.7 %, from 20,974 (3.3 birds/km) to 883 (0.14 birds/km). In the same period, the number of long-billed and slender-billed vultures censused by road transects declined by 92.2 % (28). The two species were counted together as they were not differentiated during fieldwork (28).

In 2001, high annual mortality rates of 11-18 % were reported for oriental white-rumped vultures in the Punjab province, Pakistan (29). Here, a total of 668 sick or dead vultures were collected between December 2000 to June 2001 (29). Post-morten analyses were conducted in 259 of these individuals, 219 (85%) of which had urate deposits compatible with visceral gout disease causing kidney failure, hyperuricaemia and uric acid deposits (30). Detailed necropsies and analyses (i.e. liquid chromatography and mass spectroscopy) performed in a subsample of these vultures detected diclofenac residues in 25 out of 25 (100 %) vultures that died of kidney failure, and in 0 out of 13 (0 %) vultures that died from other causes. Experiments with captive vultures were carried out to further demonstrate that diclofenac caused death and renal failure in *Gyps* vultures. Firstly, oral doses of diclofenac were administered to four captive juvenile vultures; three of them died within three days after the diclofenac administration, showing hyperuricaemia. Secondly, 20 captive vultures were experimentally fed with meat of livestock treated with diclofenac a few hours before death; 13 of these vultures (65 %) died and their necropsies revealed visceral gout and renal lesions as those observed in the vultures collected in the field.

Sharp population declines of oriental white-rumped and long-billed vultures continued between 2000 to 2004 in India and Pakistan (31, 32), and a high proportion (72%) of individuals found dead in India and Nepal had residues of diclofenac and visceral gout. A demographic simulation model showed that the observed rates of population decline in 2000-2004 could result from a small proportion of ungulate carcasses (0.277 %, i.e. 1 of every 360) contaminated with a lethal dose of diclofenac (31).

Overall, there is strong evidence of diclofenac causing mortality in individuals of the oriental white-rumped vulture and other *Gyps* species (30, 32), but the link of the evidence gathered at individual level with the widespread population declines of vultures is merely suspected or inferred. Thus, Shultz *et al.*, (32) concluded that "*Taken together, our study and that of Oaks et al., (30) have found diclofenac residues and gout in vulture carcasses collected across most of the geographical extent of the documented declines in vulture populations. The high proportion of dead vultures with signs of diclofenac poisoning make it probable that this is the major cause of the rapid population declines reported to have occurred across the subcontinent*".

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Appendix S2. Why can raw poisoned dogs be a good proxy of wildlife poisoning.

Raw number of poisoned dogs can be a good proxy for real figures of wildlife poisoning, as has been already highlighted in Spain (1, 2). Several reasons support this statement:

1) Hunting and shepherd dogs are highly exposed to a wide range of toxicants. These dogs are widespread in the countryside, especially in human-dominated rural landscapes where toxic compounds can be present (e.g. grazing areas with top predators, hunting preserves, farmlands) (3, 4). As dogs have managed to adapt to survive on the meat and non-meat scraps and leftovers of human existence and thrive on a variety of foods (5), they are very good at locating almost any kind of edible remains; not in vein they are even trained to actively look for people, dead animals and even toxic substances, including poison-laced baits (6). Dogs are therefore exposed to a wide range of toxicants, from misuse or abuse of these compounds (e.g. for plague control) to deliberately poisoned meat baits against species harmful for human activities, and hunting disposals (2, 7). Indeed, as reported in (2): "*Domestic animals* [82 % were dogs] *are potential sentinels of the use of poison to kill wildlife because the same compounds have been found in both animal groups".*

2) Secondly, poisoned dogs have a higher detectability than wildlife, since they have owners who actively look for their missing animals and take them to the veterinarian when ill (1, 2, 7). Furthermore, this prompt detection facilitates that both the necropsy and the analyses for the identification of toxicants could be done earlier as well (2). Higher detectability of poisoned dogs seems to occur elsewhere, e.g. >1,000 poisoned dogs were reported in Italy in 2005-2008, compared to < 80 red foxes (*Vulpes vulpes*), the wild species most affected by poisoning during the same period (8). Similarly, dogs

were the species more frequently reported as poisoned in Greece, with 867 individuals in 2000-2016, far more than the 287 red foxes registered as the most affected wild species (9).

3) The lack of correlation of the surface of urban areas (an indicator of human population) and both confirmed nor for suspected poisoned dogs per 10x10-km square (Pearson correlation, r_p= -0.04, P= 0.524; r_p= 0.10, P= 0.102, respectively) would further support our hypothesis that dogs exposed to poison are mostly from rural areas (e.g. shepherd and hunting dogs). According to this, the number of poisoned dogs would not depend on the total dog population, which, at least in Spain, seems to be concentrated in large urban areas, i.e. cities (10).

Considering the arguments exposed above, dividing the raw number of poisoned dogs by the total number of dogs in a site could not always accurately reflect the real incidence of poisoning events since not all the dogs registered are equally exposed to poisons. Additionally, given the high exposure to poisoning expected for certain dogs (i.e. hunting and shepherds) as well as the high detectability of poisoned dogs, even with accurate dog censuses (which do not seem to be currently available in Spain) (10, 11), the number of poisoned dogs could depend on dog population size until a certain threshold above which this relationship would disappear (Fig. S3). This could occur in sites with a high number of dogs relative to poisoning events in the field. In these sites, considering the number of poisoned dogs divided by the dog population size could underestimate the real incidence of poisoning, thus reflecting the true incidence of poisoning worse than the raw number of poisoned dogs (Fig. S3).

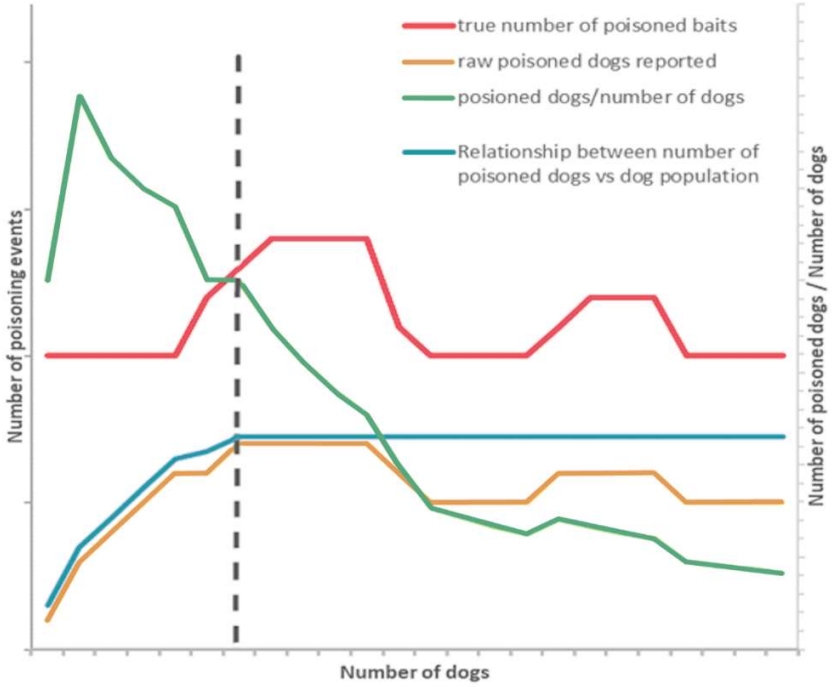

Fig. S3. The number of detected poisoned dogs corrected for population size (green line) reflects worse the true incidence of poisoning baits (red line) than the raw number of poisoned dogs (yellow line). Assuming a limited number of poisoned baits in the field, there is a dog population size (dashed vertical line) above which no more dogs can be poisoned (and therefore detected as poisoned) even with an increasing dog population (blue line). See main text for details on the simulation.

In this simulation (Fig. S3), we estimated the number of poisoned dogs (yellow line) as a function of the dog population. The number of poisoned dogs reported would also depend on the true number of poisoned baits available in the field (red line) and on the detectability of a poisoned dog, that we have randomly set at $P = 0.5$ for this representation (any other P value would not change the shape of the yellow line but just would depict it closer or further away from the red line). When the estimated number of poisoned dogs reported (yellow line) is divided by the dog population, we obtained the variation in the number of poisoned dogs per capita (green line). This number of poisoned dogs corrected for population size reflects worse the true incidence of poisoning baits (red line) than the raw number of poisoned dogs (yellow

line). Additionally, assuming a limited number of poisoned baits in the field, there is a dog population size (dashed vertical line) above which no more dogs can be poisoned (and therefore detected as poisoned) even though the total number of dogs would increase (blue line).

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Table S1. Response and explanatory variables considered to assess the impact of wildlife poisoning on the abundance and distribution of the red kite breeding population in mainland Spain between 1994 and 2014.

Variable	Description	Data source
Response variables		
Red kite presence	Presence (1) of breeding red kites in 10x10-km UTM squares in the two considered censuses (1994 and 2014). Absence (0) indicates the species disappearance as breeder, i.e. present in 1994 but absent in 2014. The final dataset consisted of 167 presences and 107 absences.	(1, 2)
Red kite abundance	Changes in the abundance of red kite breeding pairs per 10x10-km UTM square between the two considered censuses (1994 and 2014). The dataset consisted of 274 squares.	
Explanatory variables		
Wildlife poisoning		
Poisoned red kites **Poisoned kites** **Poisoned raptors** **Poisoned dogs** **Poisoned animals**	Mean number of individuals per specie or group registered as poisoned in the Spanish database per km^2 at municipality level and per year in 1995-2013. All the variables were calculated by considering all the suspected episodes (subscript $_S$), and only the episodes for which a toxic compound was identified by toxicological analyses (subscript $_C$).	
Poisoned red kites per breeding pairs in 1994 **Poisoned red kites per mean breeding pairs**	Number of red kites registered as poisoned per km^2 and year in each 10x10-km square in 1995-2013, divided by population size of red kite. Two population sizes were considered: the number of breeding pairs in 1994, and geometric mean of the number of breeding pairs in 1994 and 2014.	WWF Spain and SEO/BirdLife database (3)
Poisoned baits	Number of poisoned baits calculated as the previous variables (per km^2 and year and considering all and only the confirmed episodes).	
Habitat characteristics		
Pastures **Woody crops** **Dry crops** **Irrigated crops** **Forests** **Urban**	Percentage (%) of change in surface of the considered land use per 10x10-km UTM square in 1994-2014. Negative values indicated loss and positive values increase of each considered land use.	(4)

Table S1 (cont.)

Population size		
Breeding pairs 1994 **Breeding pairs mean**	Number of red kite breeding pairs estimated per 10x10-km UTM square in 1994, standardized by subtracting the mean and dividing by the standard deviation. The geometric mean of breeding pairs in 1994 and 2014 was also calculated to get a better picture of the red kite breeding population during this period.	(1)

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Table S2. A total of 657 red kites were confirmed as poisoned in Spain in 1995-2013 by toxicological analyses, i.e. 61.1 % out of a total of 1,075 dead individuals recorded as suspected to be poisoned in the same period. Major families of toxicants are shown together with the probable cause of poisoning, i.e. intentional, when used to deliberately kill fauna, or accidental, when red kite poisoning is a collateral impact derived from other use of the chemical such as, for example, pest control (1). The approved uses of chemicals (e.g. to control insects or rodents) and the registration status as plant protection products (PPT) and biocides in the European Union (2, 3) are also provided.

	Dead kites	%	Probable cause	EU Registration status as PPT/Biocides
INSECTICIDES[a]				
Carbamates				
Aldicarb	316	48.10	Intentional	Withdrawn in 2003 / NA
Benfuracarb	1	0.15	Intentional	Withdrawn in 2007 / NA
Carbaryl	1	0.15	Intentional	Withdrawn in 2007 / NA
Carbofuran	228	34.70	Intentional/Accidental	Withdrawn in 2007 / NA
Propoxur	2	0.30	Intentional	Withdrawn in 2002 / NA
Organophosphates				
Chlorfenvinphos	3	0.46	Intentional	Withdrawn in 2002 / NA
Chlorpyrifos	7	1.07	Intentional	Approved / NA
Demeton-S-methyl	8	1.22	Intentional	Withdrawn in 2002 / NA
Diazinon	2	0.30	Intentional	Withdrawn in 2007 / NA
Dimethoate	2	0.30	Intentional	Withdrawn in 2019 / NA
Fenamiphos	4	0.61	Intentional	Approved / NA
Fenitrothion	1	0.15	Intentional	Withdrawn in 2007 / NA
Fenthion	15	2.28	Intentional	Withdrawn in 2003 / NA
Malathion	8	1.22	Intentional	Approved / NA
Methamidophos	7	1.07	Intentional	Withdrawn in 2006 / NA
Methomyl	6	0.91	Intentional	Withdrawn in 2019 / NA
Monocrotophos	6	0.91	Intentional	Withdrawn in 2002 / NA
Parathion	3	0.46	Intentional	Withdrawn in 2001/NA
Other organophosphates	2	0.30	Intentional	
Unknown	10	1.52	Intentional	
Organochlorines				
Endosulfan	3	0.46	Intentional	Withdrawn in 2005 / NA
RODENTICIDES				
Non-anticoagulant rodenticides				
Strychnine	4	0.61	Intentional	Withdrawn in 2004 / NA
Anticoagulant rodenticides (ARs)				
Brodifacum	1	0.15	Accidental	Withdrawn in 2007 /
Bromadiolone	4	0.61	Accidental/Intentional	Approved / Approved
Difenacoum	2	0.30	Accidental	Approved / Approved
Flocoumafen	4	0.61	Accidental	Withdrawn in 2004 /
Other ARs	**7**	1.07	Accidental	--
TOTAL	**657**	**100**		

[a]Several insecticides are/were also acaricides and nematicides. [b]At least one case of insecticide poisoning was caused by the ingestion of passerines accidentally poisoned with microgranulated carbofuran (secondary poisoning). [c]Anticoagulant rodenticides usually appeared in accidental secondary poisonings, but in some cases egg baits prepared with bromadiolone against predators have been detected in Spain. NA: not apply.

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Table S3. Explanatory variables retained by the best final models explaining changes in occupancy and abundance of breeding red kites per 10x10-km square between 1994 and 2014. The importance of each variable (ω_i) and its relationships with the response variable (sign of Z value) are shown. *P* and Z values of the Wald test were calculated for a full model including all the significant variables (*P* <0.05) retained within the set of best models ($\sum\omega_m$ = 0.95) used to obtain the final best model after filtering. Models were trained with 70 % of the data (i.e. Training) and tested with the remaining 30 % (i.e. Test). Results using all data in the models are additionally displayed (i.e. Total). Pearson correlations show the robustness of the relationship between model predictions and the observed changes in the abundance of red kite breeding pairs per 10x10-km square between 1994 and 2014. AICc shows the range of AICc values of the best models (i.e. ($\sum\omega_m$ = 0.95). Subscript "C": only the poisoning episodes for which a toxic compound was identified by toxicological analyses; subscript "S": suspected poisoning episodes (see Table S2). **P* <0.05 for significant correlations. Sens.: sensitivity, Spec.: specificity. See main text for further details.

Variable	Coefficient	Importance (ω_i)	*Z*	*P*
Occupancy best model	**D^2: 9.2 %**			
Intercept	0.463	--	2.613	0.009
Poisoned $dogs_C$	-0.993	0.94	-2.238	0.025
Breeding pairs 1994	0.344	0.82	1.895	0.058
Poisoned red $kites_S$	0.577	0.76	1.783	0.075
Irrigated crops	0.481	0.72	1.641	0.101

AICc	*Training*			*Test*			*Total*	
	AUC	*Sens.*	*Spec.*	*AUC*	*Sens.*	*Spec.*	*Sens.*	*Spec.*
245.48-251.87	0.71	70.2	65.4	0.60	52.8	75.9	61.5	70.7

Variable	Coefficient	Importance (ω_i)	*Z*	*P*
Abundance best model	**D^2: 11.2 %**			
Intercept	0.088	--	1.507	0.132
Poisoned red $kites_S$	-0.170	1.00	-4.001	<0.001
Forests	-0.537	1.00	-10.920	<0.001
Breeding pairs mean	0.093	0.97	2.763	0.006
Poisoned $dogs_C$	-0.206	0.97	-2.404	0.009
Urban	-0.092	0.60	-2.693	0.098

AICc	*Training*	*Test*	*Total*
	Pearson correlation		
1177.12-1182.21	0.46*	0.12	0.36*

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Table S4. Animal species and species of raptors and carnivores for which the IUCN Red List includes poisoning (i.e. threat categories: 5.1.2. Unintentional effects (species is not the target), 5.1.3. Persecution/control and 9.3.3. Herbicides and pesticides) among the main threats for conservation of their global populations (1). The percentages represented by the species threatened by poisoning from the total and total threatened in the wild (i.e. included in the IUCN categories of "Vulnerable", "Endangered", "Critically Endangered") within each considered group are also provided at the bottom (e.g. poisoning is considered among the threats for almost half of the raptors and carnivores listed within IUCN threatened categories). [a]Raptors include species of orders Accipitriformes, Cathartiformes, Falconiformes and Strigiformes. [b]Order Carnivora.

	Number of species threatened by poisoning		
IUCN Category	**Animals**	**Vertebrates**	**Raptors[a] and carnivores[b]**
Extinct (EX)	**16**	**13**	**4**
Extinct In The Wild (EW)	**4**	**4**	**0**
Critically Endangered (CR)	**246**	**163**	**16**
Endangered (EN)	**455**	**323**	**32**
Vulnerable (VU)	**434**	**270**	**45**
Near Threatened (NT or LR/nt)	**283**	**181**	**40**
Least Concern (LC or LR/lc)	**928**	**693**	**109**
Data Deficient (DD)	**236**	**132**	**0**
TOTAL	**2,602**	**1,779**	**246**
Percentage (%) of species threatened by poisoning (N total)			
TOTAL Threatened (CR+EN+VU)	7.97 % (N = 14,234)	8.39 % (N = 9,013)	49.73 % (N = 186)
TOTAL	3.50 % (N = 73,488)	3.50 % (N = 50,816)	28.51 % (N = 861)

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