

Review of evidence concerning the contamination of wildlife and the environment arising from the use of lead ammunition

A report to Defra



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Executive summary

1. For over 100 years the environmental effects of lead from spent shotgun and rifle ammunition has been the subject of much research, particularly in North America. Lead has no known biological function and in high enough amounts is toxic and sometimes fatal to animals, particularly birds and mammals. Hundreds of tonnes of lead are deposited annually onto the soil by game shooting, target shooting and, before a ban in 1999 in England, wildfowling. Spent lead pellets are often ingested by birds, such as ducks, geese and pheasants, which mistake them for food or grit. Predatory and scavenging birds may ingest shot or bullet fragments embedded in the tissues of prey wounded or killed by hunters. In the acid conditions in the gizzard, the lead is dissolved and absorbed into the blood stream resulting in acute or chronic poisoning. While individual cases of lead poisoning are not disputed, the effect at the population level is uncertain.
2. The degree of contamination from lead ammunition is directly proportional to the intensity of use. Both wildfowling and game shooting are seasonal activities while target shooting (e.g. clay pigeon shooting) occurs throughout the year. Before the lead-shot ban for wildfowling in England, densities of up to 300,000 pellets/ha in the top 15 cm of sediment were found at wetland habitats across Britain. In other countries >2 million/ha have been recorded, but the availability of shot is dependent on the nature of the sediments – if soft, pellets can sink rapidly out of reach of waterfowl, while heavy clay retains shot close to the surface. Game shooting takes place over farmland, moorland and forests with probably less intense hunting pressure (shooters per unit area), but, as an example, a pellet density of 107,639/ha in the top 1 cm of soil was measured after the hunting season ended, a 300% increase over the pre-hunting density. In contrast, on clay pigeon, trap and skeet ranges the discharge of up to 36 g of lead shot per cartridge occurs over a relatively small, well-defined area and pellet densities of up to 3.7 billion/ha in the top 7.5 cm of soil have been recorded. On rifle and pistol ranges, lead from spent bullets tends to accumulate in a very restricted area and >300 g lead/kg dry soil has been measured against a natural background level of 10-30 mg/kg.
3. It has been estimated that when exposed to air and water half of the metallic lead in spent pellets will break down to form lead oxides, carbonates and other compounds in 40-70 years and complete decomposition will take 100-300 years. These lead compounds are soluble under acidic ($\text{pH} < 6.0$) conditions resulting in elevated lead levels in soil, water and vegetation. One lead compound, cerussite, has been shown to bioaccumulate in earthworms and may cause elevated lead levels in the shrews that eat them. However, bioavailability and movement of lead through the food chain are dependent on a number of factors, which may vary locally. Where shot has fallen onto arable fields, oxidation of the pellets over many years has increased the amount of 'plant-available' lead to a point where crop yields have been reduced. In contrast, where shot has fallen in woodland and onto rough pasture, bioavailability has been low with little movement of lead to higher trophic levels. Soils with a high CaCO_3 , Fe, Al and P content can immobilise lead and with adequate amounts of phosphorus, insoluble lead phosphate can be formed.

4. The principal measure of exposure to lead ammunition in waterfowl and game-birds is the presence of lead pellets in the gizzard. Ingested pellets can be detected by fluoroscopy (X-ray) of the intact gizzard or the gizzard contents, or by visual examination, but it is important to distinguish ingested pellets from pellets fired in, as investigators invariably use hunter-killed birds when determining exposure rates. It has been found that waterfowl in the early stages of lead poisoning are more likely to be taken by hunters than non-poisoned birds. Pellet ingestion rates do not necessarily measure the likelihood of poisoning, as the absorption of lead from the intestine is dependent on the number of pellets ingested and their retention times, the type and volume of food consumed, the type of grit ingested and the physiological condition of the bird. Once in the bloodstream, lead can be deposited in other organs rapidly and remain there for several months. Lead in the bones may accumulate over the lifetime of a bird.
5. As lead is ubiquitous, a diagnosis of lead poisoning from an ammunition source depends on establishing a threshold value to separate background from elevated concentrations in a particular tissue. For blood, a background level established about 30 years ago of $<20 \mu\text{g}/\text{dl}$ is now regarded by some as too high and likely to have been influenced by sample contamination – some investigators now consider $<3.75 \mu\text{g}/\text{dl}$ as the criterion. In the liver, lead concentrations of $\leq 10 \mu\text{g}/\text{g}$ dry weight (dw) or $<6 \mu\text{g}/\text{g}$ dw have been used to indicate background exposure; $38 \mu\text{g}/\text{g}$ dw has been suggested as a defensible criterion for identifying lead-poisoned waterfowl in the absence of pathological observations. Interpretation of elevated lead levels in organs of hunter-killed birds can be difficult because of the likelihood of contamination with minute lead particles from fragmenting pellets. An alternative approach to measuring lead levels in various tissues is to measure the effects on enzyme systems, but it may not always be certain that lead has caused any change that is observed.
6. Ingested lead pellets have been found in a wide range of waterfowl species and the prevalence can vary widely from $<10\%$ for a particular species up to 70% or more. In game-birds (partridge, pheasant, grouse) the prevalence appears to be generally lower and more consistent across species at $<5\%$, but some argue that many birds suffer acute lead poisoning and so are not captured by the sampling method, resulting in an underestimate of ingestion rates. A high ingestion rate of 34% was found in farm-reared pheasants released for shooting onto an island where pellet densities exceeded, locally, 82 million/ha in the top 10 cm of soil. Experimental trials on captive birds, especially mallards, have been carried out to try to establish dose-response relationships in order to estimate how many birds might die from lead poisoning. In some studies, mortality was directly related to the length of pellet retention, but, overall, the effects of diet confound the interpretation of results as one mallard may succumb after ingesting a single pellet, while another seems unaffected after ingesting 30 or more pellets. However, pellets experimentally embedded into the muscle of birds caused no ill-effects, suggesting that wounded birds that recover from the initial injury are not likely to suffer lead poisoning. Although fewer studies have been carried out, game-birds and passerines appear to have a strong ability to void ingested pellets relatively quickly and therefore may have some degree of protection from poisoning.

7. Sources of lead for raptors are unretrieved carcasses, wounded animals that escape and die later, or gut piles left by hunters. Raptors can potentially expel shot or large bullet fragments with the indigestible parts of their prey in the form of regurgitated pellets. In tests, one species of kestrel regurgitated pellets embedded in prey within a day of ingesting it. In contrast, the regurgitation rate in bald eagles was much more variable from 12 h to 48 days and difficult to predict in individual birds. X-rays of carcasses shot with lead bullets have revealed minute particles of lead along with larger bullet fragments scattered among the tissues surrounding the bullet tract. While the larger fragments might be regurgitated by raptors, the dust-size particles would probably not be and their large surface area to volume ratio would facilitate dissolution and absorption of lead. The effect of lead poisoning on raptor populations is unknown, but in general these species are considered to be particularly vulnerable as they tend to be long-lived and slow-breeding. In one study in the USA, the prevalence of lead poisoning in eagles showed no decrease when lead pellets were banned for wildfowling. However, fewer birds were found with acute poisoning after the ban, but significantly more were found with chronic poisoning. It seemed likely that the birds were finding (non-waterfowl) food sources that were still able to be shot legally with lead ammunition. There appears to be little or no information on the risk to mammalian predators and scavengers.
8. Legislation came into force in England in 1999 prohibiting the use of lead shot for wildfowling. In the USA, similar bans had been in place locally since 1977 followed by a nationwide ban in 1991. Canada, Australia, New Zealand and many European countries have now also imposed bans. Several studies in the USA have examined the effect of the ban on waterfowl populations, but none appear to have been carried out in the UK. In the USA, compliance with the ban has been reported as high (up to 99%) and 5-6 years after the nationwide ban, examination of over 15,000 gizzards from mallards shot along the Mississippi Flyway showed that the ingestion rate of lead pellets had been reduced by about two-thirds. The estimated mortality rate from lead poisoning declined from 4% in 1938-54 to $\leq 1.44\%$ in 1996-7 saving 1.4 million ducks out of a population of 90 million from fatal poisoning. However, the slow weathering of lead pellets could maintain lead poisoning as a persistent problem long after hunters have switched to lead-free shot, particularly where clay soils have prevented pellets settling deep enough to be unavailable to ducks and other waterfowl.
9. Humans that eat game animals that have been shot with lead ammunition are at risk of poisoning from the fragments of bullets or pellets that remain embedded in the meat or in other edible tissues; the risk from biologically-incorporated lead in game appears to be low. Radiographs of shot deer have shown that when a lead bullet breaks up on impact some fragments are too small to be visible to the unaided eye and would probably be retained in the meat after butchering. People in communities that depend heavily on hunting for subsistence are likely to be most exposed. Individuals that have accidentally ingested lead shotgun pellets have developed symptoms of poisoning and it has been discovered that pellets can be retained in the appendix for several years, although the dissolution rate appears to be slow in such cases. In assessing the level of risk, embedded lead tends to be distributed heterogeneously in bodies and thus tissue samples can, by chance,

contain very high or very low lead concentrations, making it difficult to assess the threat to human health with any degree of confidence. However, the potential risk to humans from embedded fragments was demonstrated when samples of contaminated meat were fed to pigs resulting in blood lead levels significantly higher than in pigs fed uncontaminated meat.

10. The EU limit for lead in meat (excluding offal) is currently 0.1 mg/kg wet weight (ww), but this does not apply to game meat. The limit for offal is 0.5 mg/kg ww and in Spain, where the liver of waterfowl is considered a delicacy, examination of a sample of 411 livers revealed that 40.4% exceeded this limit. The FAO/WHO tolerable daily intake (TDI) for lead is 3.57 µg/kg body weight (25 µg/kg body weight/week), although this is no longer considered protective. After measuring lead levels in seabirds eaten by people in Greenland, it was suggested that the TDI would be exceeded by eating 4 or more bird meals per week, but the consumption of birds is seasonal and longer-term monitoring would be needed to establish the risk to consumers. An additional risk may come from traditional recipes used to prepare game meat, especially where vinegar is used in the process. It was found that a lead pellet weighing 0.11 g and cooked in vinegar could release a quantity of lead sufficient to exceed the EU limit for meat and with 4 pellets the level could be 300 times over the limit. It has been argued that because of variation in response among humans to lead, the setting of a threshold concentration below which there are no harmful effects is meaningless. Nevertheless, it is considered prudent to reduce further exposure to anthropogenic lead in the environment.
11. If no lead pellets or bullet fragments are found in animals with elevated lead levels the source of the exposure may not be immediately obvious. Within the last 10 years or so, the use of stable lead isotope ratios has enabled researchers to identify the lead source, or at least to eliminate certain potential sources. Lead has 4 stable isotopes, three of which are radiogenic. Different ores may have different isotopic compositions which are retained in the products which are eventually made from them; the ratio of ^{206}Pb : ^{207}Pb is most commonly used in analytical work. In one study, low levels of lead in the bones of red grouse were linked to background lead in the soil, but it was speculated the long-term deposition of lead pellets onto soils might shift the isotopic signatures away from the natural background source towards those for gunshot. If this happens, the technique will become less useful.
12. Various methods have been developed to mitigate the environmental effects at locations where lead ammunition can still be used and to clean up environments where it had been used in the past. These include lead recovery techniques so that the metal can be recycled, but these are generally more suitable for target shooting ranges where shot-fall zones and bullet catchers are clearly delineated and access for machinery is easy. The initial cost in 2006 of decontaminating 50-60 ranges in Finland was estimated to be €250 million. On wetlands, cultivation (ploughing and disking) of the soil can redistribute shot to deeper layers in the soil, although it can cause temporary reductions in vegetation that may be an important food source for some waterfowl. Other methods applied to shooting grounds include adding lime to the soil to raise the pH, adding phosphorus to produce an insoluble

lead compound, phytostabilisation, and encouraging vegetation and spreading mulches and compost to provide natural complexing agents.

13. It is strongly argued that the most effective way to reduce the risk of lead poisoning in both wildlife and humans is to encourage or compel hunters to switch to non-lead (so-called 'non-toxic') ammunition, which has been available for many years but is generally more expensive. Alternatives to lead shot include tungsten composites, bismuth and steel; non-lead bullets have been developed for some calibres, particularly those used to hunt deer. So far, few Governments have opted to ban lead ammunition entirely and most still allow its use to hunt non-waterfowl birds and mammals. This is partly because there is a lack of scientific certainty about the extent of the problem, but some stakeholders suggest that The Precautionary Principle should take precedence.
14. Those stakeholders opposing a total ban perceive that, as very few dead birds are found and the problem has been present for decades, then the impact must be small. For others, the size of the problem is significant, but concealed to some extent, because dead or dying birds are often removed quickly by predators and scavengers – sudden mass die-offs are rare. A nationwide lead-shot ban in North America, where compliance appears to be high, has probably prevented lead poisoning in up to 4 million waterfowl annually (4% of total population), while in the UK, compliance has apparently been low and the effect on waterfowl populations is unknown. However, it has been postulated (mainly on theoretical grounds) that reducing mortality from lead poisoning might not reduce total mortality in quarry species as natural mortality rates could increase to offset those gains. Nevertheless, lead poisoning causes severe pain and distress in birds and switching to non-toxic ammunition entirely would improve animal welfare. If a ban on lead shot for wildfowling has helped conserve populations of endangered raptors, then it can be argued that a total ban on lead ammunition for all kinds of hunting would be needed to protect other rare species, especially those that prey on or scavenge a wide range of mammalian and avian prey. The risk to humans from consuming contaminated game is unclear as results from different studies are inconsistent and current opinion is that there is no 'safe' level of lead.

Introduction

Lead (Pb) has no known biological function and in high enough amounts is probably toxic to all forms of life. It is found naturally as a component of various minerals, such as galena (PbS), cerussite (PbCO₃) and anglesite (PbSO₄). Humans have mined it for hundreds of years and its low melting point, malleability and low cost have resulted in its use in a wide range of applications. When products are discarded in the environment, any lead they contain follows natural pathways that end in a permanent sink in the soil or ocean sediments. These pathways have dispersed lead so widely that it is doubtful whether any part of the Earth's surface or any living organism remains uncontaminated with anthropogenic lead [131].

One application of lead is the production of projectiles for firearms and it is the only use that involves the deliberate release of the metal into the environment [143]. The lead content in other products, such as paint, water pipes and petrol, has been restricted since the risks of poisoning became better understood. The amount of lead actually dispersed into the environment from ammunition sources is difficult to calculate, but many investigators have provided rough estimates. In 1991, about 6000 tonnes were deposited annually onto the soil surface at clay pigeon ranges in the UK, with the total deposition from all forms of shotgun shooting likely to have been twice that amount [89]. In Denmark in 1984, 800 tons of lead in shotgun ammunition were used annually, compared with a total emission of lead in fuel additives of 250 tons annually [69]; the use of lead shot was banned in 1996 [84]. In Sweden in 1995, 500-600 tons were used annually in shotgun ammunition [78], but in 2002 the government introduced a ban on lead in all types of ammunition with full implementation by 2008 [84]. In Canada between 1988 and 1993, the total deposition into the environment by hunters and clay target shooters averaged 2,149 t/yr [122]. In the US in 1999, the amount entering the environment through the discharge of firearms for hunting and recreational purposes was increasing at the rate of 60,000 tonnes per year [27]. Using data from 1968 [77], 5,600-9,200 t /yr of lead were introduced into the terrestrial environment in the US just for harvesting mourning doves – including other game species would have increased that amount by much more [71].

It has often been assumed that lead from spent ammunition is environmentally inert and thus unlikely to be a source of contamination in the environment. It is now widely accepted that eventually all metallic lead in spent projectiles (bullets or shot) will be mobilised and dispersed into the environment contaminating soils and ground water and then transferring to higher trophic levels. A more long-standing concern has been the poisoning of wildlife from spent ammunition, particularly shotgun pellets. Such poisoning was first identified at the end of the 19th century in the USA (papers cited by [12] [118] and UK (papers cited in [107]). The first major review of lead poisoning in waterfowl appeared in 1959 [12], another in 1986 [118] and a third in 1995 that also included the effects of shot on non-waterfowl species [122]. The most recent review was published in the proceedings of a conference organised by The Peregrine Fund in the USA in 2008 [7] [51] [62] [75] [84] [95] [99] [120] [136] [142] [147]. Spent pellets are ingested by animals either deliberately (mistaken for food or grit) or accidentally (e.g. embedded pellets in the tissues of prey). Ironically, the species that appear to have been most affected, in terms of the number of individuals poisoned, are those that are the usual quarry of hunters such as wildfowl (e.g. ducks, geese) and game-birds (e.g. pheasant, partridge, grouse). However,

raptors, such as eagles, buzzards, falcons, red kites and vultures, are also at risk when they prey on or scavenge animals that have been shot with shotgun pellets or fragmenting bullets or that have high levels of biologically-incorporated lead in their tissues. This secondary poisoning risk also extends to humans, such as First Nation Cree in Canada, who live in communities that depend on a regular supply of game animals for subsistence.

The accumulating evidence on the effects of spent lead projectiles on the environment, wildlife and the potential consequences for humans has convinced many stakeholders that action should be taken to ban the use of lead in ammunition. Bans on its use over wetlands and for wildfowling have been in force in several countries within Europe and North America for many years, but few Governments, so far, have opted to phase it out in all types of ammunition. This may be due partly to concerns about the performance, safety, availability and cost of non-lead projectiles, but also about the extent to which lead poisoning actually depresses populations. While the effect of lead toxicosis is real, its impact relative to other causes of mortality is small and populations can recover through compensatory breeding [143]. The number of birds shot through hunting far exceeds the number poisoned, yet the populations seem to replace the previous year's losses. The purpose of this review is to summarise the evidence on wildlife contamination through use of lead in ammunition and the potential consequences of such contamination for both human and wildlife health. This will support the development of relevant policy in this area and identify any key knowledge gaps. Most of the information has been derived from peer-reviewed articles in scientific journals, but also from published reports from government agencies, such as the US Fish & Wildlife Service and the Canadian Wildlife Service and conference proceedings.

1. Determining the extent of contamination

The effects of lead ammunition on the environment and wildlife have been associated with three main shooting activities: wildfowling (shooting over wetlands), game-shooting/pest control (on agricultural land, moorlands, forests) and target shooting (clay pigeons, trap and skeet, rifle and pistol shooting at static targets) (Table 1). *Prima facie*, spent ammunition is the most likely source of any lead found in animals that live in and around any shooting site, but as lead is ubiquitous other sources cannot be ruled out, including mine wastes, natural weathering of rocks and atmospheric lead from car exhausts (less likely since the ban on lead in petrol).

1.1 Lead in ammunition

Shotguns fire ammunition that in 12-bore calibre contain 100-600 spherical pellets in each cartridge, depending on shot size: the nominal diameter (and weight) of a No. 9 lead pellet is approximately 2 mm (0.05 g), No. 6 2.8 mm (0.13 g) and No. 4 3.3 mm (0.2 g). After exiting the barrel, the pellet mass spreads out so that, even if the target is hit, many shot are likely to miss and fall to earth. Rifle and pistol cartridges contain a single projectile, which, depending on its design, may fragment or just deform on impact with the target. In cartridges designed for hunting game, such as deer, bullet fragments may be contained within the body of the animal, but sometimes

all or part of the bullet may pass through and exit the body. Hunting with a rifle is often an individual pursuit and given the nature of the ammunition, dispersion of lead into the environment is likely to be, overall, localised and at a relatively low density. In comparison, shotguns disperse the ammunition by design, so that contamination of the environment is largely unavoidable and where hunting is carried out as a group activity (e.g. organised pheasant shoots), such contamination can be potentially widespread. In contrast, intensive target shooting with rifles or shotguns on specified ranges can result in high concentrations of lead locally in the soils of the bullet catchers (stop butts) or in the shot-fall zones.

Table 1. Shooting activities and the contamination that arises from the use of lead ammunition and the species that are at risk of poisoning as described by selected references. Although comparison of pellet densities is difficult, the examples shown broadly relate the type of shooting to the degree of contamination. Lead in crops is either soluble lead absorbed through the roots or lead dust adsorbed onto leaves. No data have been found assessing the risk to mammalian predators and scavengers.

Activity	Contamination source	Species at risk
Wildfowling Ducks, geese	Spent pellets 300,000/ha [92] >2,000,000/ha [110] Embedded shot [3]	Primary poisoning Ducks, geese, swans [91] [140] Secondary poisoning Mammalian predators and scavengers? Raptors [156] Humans [67]
Game shooting Pheasants, partridges, grouse, deer, wild boar	Spent pellets - 107,639/ha [77] 560,000/ha [60] Embedded shot & bullet fragments [36]	Primary poisoning Pheasants [19], partridges [18] [107], grouse [146] Secondary poisoning Mammalian predators and scavengers? Raptors [97] [159] Humans [124]
Clay pigeon/target shooting	Spent pellets – 3.7 x 10 ⁹ /ha [133] Bullets [35] Crops [89] [112]	Primary poisoning Small mammals [132] Passerines [155] Secondary poisoning Shrews [80]
Pest control Rabbits, rodents	Embedded shot/bullet fragments [72]	Secondary poisoning Raptors [72]

Lead shot contains 95% lead and up to 1.5% antimony as a hardening agent and less than 1% arsenic as an aid to forming spherical shot [56]. These amounts may vary according to manufacturer, but there is no evidence that, at those levels, antimony and

arsenic cause problems in birds [96]. Other elements found in shot include tin, selenium, manganese, cadmium, chromium, copper and nickel, the last two helping the shot to resist deformation during firing. Bullets consist of a metallic core of lead hardened with 2-4% antimony encased wholly or partly by a cupro-nickel steel jacket [154]. On striking the target, the core and jacket of such bullets often separate, with the lead core breaking up into large and small particles [95]. Hunting bullets are intentionally designed to expand or break up on impact with the fragments creating multiple wound channels that incapacitate the animal rapidly and result in a quick kill.

1.2 Measuring the amount of spent ammunition in soils

The degree to which shotgun pellets contaminate the environment is determined by counting the number of spent pellets present in shot-fall zones. The usual technique is to take core samples of soils or sediments at regular intervals along transects across the affected area. Shot are extracted by passing the soil/sediment samples through one or more sieves and can be identified visually as being lead, or by scratching the surface with a scalpel to reveal a shiny grey/silver surface [98]. Lead shot can be separated from steel shot using a magnet or by the resistance of the pellet to compression by pliers [139]. Results are often presented as a density of pellets per unit area. A comparison of pellet densities, as reported in the literature, to determine relative levels of contamination between sites can be difficult due to different core depths, distribution of shot vertically in the core, timing (beginning or end of hunting season) and the size of sieve used [86]. The efficiency of the pellet recovery method also needs to be known. This can be assessed by seeding core samples with pellets before they are sieved. In cores from mudflats, saltmarshes, flood meadows, flight ponds and lake bottoms, an overall recovery rate of 94.4% was recorded, allowing data to be corrected [92]. Pellet densities will also vary according to shot-fall patterns, which in turn will depend on the location and number of shooting positions. For example, where there was a single shooting position, a shot fall zone with a radius of 250 m from that point was assumed [92]. In a different study, samples were taken in the area of greatest shot deposition, which was determined to be approximately 80-140 m from the shooting point [114].

1.2.1 Wetlands – before the lead shot ban

Although pellet density is dependent primarily on the shooting intensity over a particular wetland, it is also dependent on the rate of settlement of pellets into the sediment. Pellets may be buried quickly if there is a high siltation rate. Core samples were taken from 22 sites at 9 wetland habitats across Britain during 1980-81 [92]. No pellets were found at 3 sites, but elsewhere densities ranged from 20,400/ha to 300,000/ha in the top 15 cm of sediment. Densities did not differ significantly in relation to open and closed hunting seasons. Over 60% of pellets were recovered in the top 5 cm of sediment and pellet settlement experiments revealed that where clay soils were present, the majority of pellets remained in the top 2 cm after 9 months. A very high density was recorded in 1986 on a tidal meadow that was inside the shot-fall zone of a trap and skeet range in New Jersey, USA [114]. Core samples were taken from the area of greatest shot fall (90-150 yds (82.3-137.2 m) from the shooting point) and pellet density was calculated to be 217 million/ha in the top 7.5 cm of

sediment. In California in 1986-7, sediment samples were taken from a wetland site before and after the hunting season [110]. In October 1986, 272 lead pellets were recovered from 200 sediment cores giving an estimated density of 2,142,200 pellets/ha and in May 1987 the density had increased slightly to 2,299,700/ha; 3 of the 309 pellets recovered were steel. At a hunting reserve near Darwin, Australia, 129 core samples were taken from swamp substrates in a study carried out between 1988 and 1990 [158]. The cores were taken in groups of three at 30-m intervals using a steel pipe with an internal diameter of 8 cm and to a depth of 40 cm. Lead shot was found in 17/115 cores examined with a mean number of shot/core of 0.17 ± 0.42 (SD). This gave an average density of 33 pellets/sq m or 330,000/ha and the distribution of pellets appeared to follow a random scatter. The sediment samples were taken before the hunting season and at least 8 months after the previous season closed. From information on shooting intensity, the estimated annual shot input was close to 17 million pellets.

1.2.2 Game-shooting

Although the main focus in the literature (particularly before lead shot bans became widespread) has been on the risk to waterfowl, the risks to other game-birds (often referred to in North American literature as ‘upland game’), such as pheasants, partridges, grouse, mourning doves, bobwhite quail etc. has received considerable attention. Shooting of these birds takes place on farmland, moorland and forests with less intense hunting pressure (shooters per unit area) than on wetlands, but potentially more widespread contamination – up to 96,000 pellets/ha [39]; 107,639/ha was calculated for the top 1 cm of a field used for dove hunting after the hunting season ended – a 300% increase over the pre-hunting period [77]. However, in the USA, upland game shooting is often carried out over selected agricultural fields managed to provide food for species such as doves. With intense hunting pressure this can result in high deposition rates of lead pellets – in one study, hunters fired, on average, 8.6 shots for each bird killed [54]. This was investigated in two fields, one of 8.9 ha and another of 5.1 ha [126]. In the larger field, no spent pellets were found in the top 1 cm of soil before and after 4 days of hunting, even though 60 hunters had deposited nearly 65,000 pellets/ha onto the ground. In the other field, after hunters had deposited pellets at the rate of over 1 million/ha during 4 days, only 6,342 pellets/ha were found during soil sampling. As this was considerably lower than reported in other studies, the authors suggested that the soil sampling protocols may not always provide accurate estimates of spent-shot availability. Minor differences in the depth of a soil sample and the physical characteristics of the soil (e.g. texture) and moisture content may affect the weight and volume of soil samples and consequently the estimates of available shot. Moreover, estimates become biased when vegetation is removed prior to sampling or when plots with standing vegetation are sampled, because pellets may be embedded in plant stalks.

1.2.3 Shooting ranges

In contrast to waterfowl and game shooting, target shooting is often carried out all year round. A characteristic of clay pigeon, trap and skeet ranges is the intensity of use and the discharge of up to 36 g of lead pellets per cartridge over a relatively small

(40-100 ha), well-defined area [32]. This can result in pellet densities of up to 3.7 billion/ha in the top 7.5 cm of soil/sediment (\equiv c. 400 t lead) [133]. On rifle and pistol ranges, lead tends to accumulate in a very restricted area (immediately behind a target) of a stop butt. A maximum total lead concentration of 68 g per kg in the top 5 cm of soil 9 m behind the target was recorded against a natural background level of 10-30 mg/kg [154] and >300 g lead/kg dry soil was measured to a depth of 0.6 m below the sloping surface of the stop butt at a small-bore shooting range in Denmark [6]. The size and shape of bullets, in contrast to spherical shotgun pellets, mean that there is a smaller relative surface area exposed to environmental action and whole bullets are less likely to be ingested by birds and mammals. However, shot strikes the ground with relatively low velocity, while bullets fired on rifle and pistol ranges strike the stop butt at much higher, sometimes supersonic, speeds. This can result in projectile fragmentation and the creation of a lead dust with an aerial fallout zone [13]. These dust particles are potentially soluble and mobile. If not dissolved within the soil, they may fall onto the surface of vegetation and be accidentally eaten by herbivores.

1.3 Fate of lead in soils

An investigation of the decomposition of lead pellets at Danish shooting ranges predicted that half of the lead in a pellet will be transformed into lead compounds, mainly cerussite, hydrocerussite ($\text{Pb}_3(\text{CO}_3)_2(\text{OH})_2$) and anglesite, within 40-70 years and that pellets will be completely transformed within 100-300 years [69]. Under certain circumstances, such as intensive mechanical soil treatment, these periods may be considerably shorter, i.e. 15-20 and 30-90 years, respectively. Lead is much more soluble under acidic (pH <6.0) conditions than at neutral or alkaline (high pH) conditions; very little lead remains in solution at pH >8.0 [122]. The lead compounds form as surface coatings on the pellets or in the surrounding soil. This transformation is accelerated when the pellets are mixed with the upper soil layers by ploughing and other agricultural treatments, which dislodges the crust material exposing fresh metallic lead. Similarly, on rifle ranges, crust material may also be removed from decomposing bullets by the impact of other bullets fired into the stop butt. Moreover, in bullets with a lead core covered by a copper jacket, the jacket may not always separate from the core on impact with the stop butt. It has been found that where lead and copper coexist in large fragments, the solubility of lead is increased due to the galvanic corrosion reaction [35].

In an acid sandy soil, the transformation products are soluble and mobile, but where the pH is high and/or there is a high content of organic matter, the products are only sparingly soluble [20] [69] [122]. With such high shot-deposition rates on shooting ranges, weathering can result in a significant elevation of lead levels in soil, water, and vegetation, but high CaCO_3 , Fe, Al, and P contents may counteract this [20]; with adequate amounts of phosphorus, insoluble lead phosphate (pyromorphite) can be formed. Lead particles or lead compounds can spread through the environment as airborne dust, or be suspended in storm or river run-off or as dissolved lead in surface or ground water [122]. The mobility of lead in these forms will be reduced by vegetation that slows water run-off and by a high organic content in clay soils to which lead will adsorb.

At a clay pigeon range in the north of England, the impact of lead-pellet deposition on soils and crops was assessed [89]. The range was on farmland and pellets fell onto oil-seed rape plants. Soil samples were collected across the shot-fall zone and the greatest concentration of lead (5,000-10,620 mg/kg) was found between 80 and 140 m from the firing point; pellet density was greatest at a distance of 110 m and zero at 300 m. 'Plant-available' lead concentrations ranged from 1,000-4,102 mg/kg at 90-140 m from the shooting point, substantially higher than 20 mg/kg that is found in uncontaminated soil. Soil pH ranged from 4.9-6.6. Samples of oil-seed rape plants were also analysed at 100 m (contaminated) and 300 m (uncontaminated) from the firing point: lead concentration in the roots was 470 mg/kg (dw) and 10 mg/kg respectively; corresponding levels in the stems were 62 mg/kg and 4 mg/kg and in seeds 148 mg/kg and not detected. The dissolved lead that was available to plants appeared to be responsible for decreased crop yields.

A similar study was carried out at a clay pigeon range on agricultural land in New Zealand [112]. Soil samples were taken across the shot-fall zone and sieved and the 'plant-available' lead in the fraction <2 mm was determined by extraction with EDTA (ethylenediamine tetraacetic acid). Sieving removed about 85% of the total lead deposited on the range (i.e. particles >2 mm). Lead concentrations were measured in 5 plant species grown in soil from the range: barley (*Hordeum vulgare*), lettuce (*Lactuca sativa*), perennial ryegrass (*Lolium perenne*), radish (*Raphanus sativus*) and white clover (*Trifolium repens*). Concentrations of EDTA-extractable lead ranged from 4,000-8,300 mg/kg at 100-160 m from the shooting positions and were comparable to those found in areas contaminated with mining wastes. Background levels of <25 mg/kg were found beyond 220 m from these positions. Lead concentrations in the leaves and roots of all 5 plant species increased significantly with an increase in soil lead concentration. Generally, higher concentrations were found in the roots compared with those in the leaves. There was also a reduction in plant yield with increasing levels of lead in the soil. The study showed that the oxidation of lead pellets over many years had produced lead concentrations in soils and plants that were far in excess of guideline levels for environmental and human health. It was also apparent that there was a substantial volume of lead which had not yet weathered and without remediation the risk of contamination would remain for many years to come.

The fate of shotgun pellets on ground used for pheasant shooting was assessed at a site in Cheshire, UK [129]. The shooting ground consisted of a mixed deciduous woodland and rough pasture. Lead levels in the soils, soil pore water, plants, earthworms (*Lumbricus terrestris*) and in the hair of small mammals (*Apodemus sylvaticus*, *Clethrionomys glareolus*) were determined and compared with those in control areas. The soil pH on both shooting and non-shooting areas ranged from 4.3-6.09 and was therefore acidic. Lead concentration was significantly higher in rye grass from the shooting field, in moss in the shooting woodland and in worm gut contents from the shooting wood. Soluble lead and/or lead particles adhering to the hair of the small mammals were thought to be more important than lead absorbed into the hair for the higher levels found in the shooting woodland. Overall, it was found that the bioavailability of lead in soils from both shot and non-shot sites was very low although higher in the shooting areas. Lead movement from soil water to higher trophic levels was minor and lead concentrations in biota were below statutory levels in all locations.

Soil samples were collected from 14 sites on a private island in southern Ontario, where there was intensive hunting of indigenous game and imported farm-reared game-birds and also clay pigeon shooting [60]. On the clay pigeon ranges, pellet density varied from 759/0.25 m² to 2051/0.25 m² (c. 30-82 million/ha). On the hunting fields, pellet density varied from 0-14/0.25 m² in relation to frequency of tillage. Soil lead concentrations were 3-199 µg/g and on only 4/14 sites the concentration exceeded 150 µg/g, which was the upper limit for unpolluted soil in rural Ontario. The pH for the 14 soil samples ranged from 6.8-7.5, neutral to slightly alkaline and the organic content ranged from 9-31%; 9 sites were strongly to extremely calcareous, while the others were non- or weakly calcareous. The study showed that despite a high deposition of lead pellets, the pellets remained relatively inert.

1.4 Fate of lead in animals

The principal measure of lead ammunition exposure in waterfowl and game-birds is the presence of lead pellets in the gizzard. Birds have a two part stomach, a glandular proventriculus and a muscular gizzard. Hydrochloric acid, mucus and a digestive enzyme, pepsin, are secreted in the proventriculus and start breaking down the food material, which then passes to the gizzard. The gizzard acts like mammalian teeth, grinding up food and making it easier for the digestive enzymes to break it down. In most birds the gizzard contains grit to aid the grinding process and spent shot may be taken in by birds seeking grit, or may be mistaken for seed and swallowed. The pH in the gizzard is low: in a bald eagle 1.3, snowy owl 2.5, duck 2.1 [37]. In this acidic environment, the lead in shot or bullet fragments is dissolved and then absorbed from the gut into the blood stream. Within 8 hours of dosing mallards with lead shot, blood lead concentrations exceeded background levels and within 2 days had reached a maximum and then declined to normal by day 36 for 90% of the birds [113]. Once in the blood, lead is deposited in other organs and the levels in the liver, kidney or bone are commonly reported in relation to exposure rates. Levels in the liver may stay elevated for several months following exposure, but lead in bone is relatively immobile and may accumulate over a bird's lifetime [96].

1.4.1 Detecting ingested pellets

Ingested pellets can be detected by fluoroscopy (X-ray) of an intact gizzard or the gizzard contents, or by visual examination of the contents [98]. However, use of X-rays is often impractical, especially in field-based studies. A more practicable method is to wash the contents into a dish and allow plant material and other food items to float off leaving grit and shot in the bottom [98]. The heavier particles can then be examined by the unaided eye or under a binocular microscope, the latter proving to be more reliable and similar in efficacy to X-ray. It is also necessary to distinguish embedded pellets (often referred to as 'shot-in' i.e. fired from a gun) from ingested pellets, the former leaving entry holes in the gizzard lining. Even if a hole is not found, an embedded lead pellet can be identified by its dark grey colour, normal size and shape or angular with a flattened, uneroded surface; ingested shot has an eroded and pitted surface and is sometimes undersize [3] [12]. Pellets with sharp edges have

been classified as shot-in [31]. Pieces of feathers around the pellet or in the gizzard cavity also indicate embedded shot [77]. However, shot-in pellets can be mistaken for ingested pellets if they have entered the gizzard lumen some time before the birds are actually killed [79]. Steel pellets can be detected with a magnet [3], but the pellets do not deform on hitting a bird making it harder to distinguish ingested from embedded pellets [148]. Also, fluoroscopy cannot distinguish lead from steel pellets [57].

The effectiveness of the different methods for detecting ingested pellets has been evaluated. Visual examination missed 24% of ingested pellets because small, eroded pellets could not be distinguished from seeds and grit; X-rays of intact gizzards missed 28% of ingested pellets as they failed to detect small pellets obscured by grit, food and the muscular wall of the gizzard (study cited by [118]). Visual examination of grit in 3,389 mallard gizzards found ingested pellets in 6.3%, with the rate rising to 7.9% when grit samples were X-rayed and then to 8.2% when food samples that had been separated from the grit were also X-rayed [3]. The incidence of shot-in pellets was considered high enough (5.8% in 13,246 mallards) to influence interpretation of data if such pellets were not properly identified.

In most studies, the gizzard samples are obtained from hunter-killed birds. In a compilation of studies carried out from 1973-84 in the USA, 8.9% of 171,697 duck gizzards had ingested pellets [118]. A potential bias can be introduced as lead-poisoned birds are more likely to be taken by hunters ($\times 1.65$ – [12]). Accounting for this, the revised rate reduces to 5.4%, but a much larger percentage of wildfowl consume pellets, because if a duck doesn't die after eating a pellet, the pellet will disappear in c. 20 days (either eroded or voided). Thus, when a mallard is found with an ingested pellet, it means the pellet was eaten within the preceding 20 days. In North America, ducks spend 150 days in migration and on the wintering grounds, giving 7.5 intervals of 20 days during this period. Multiplying 5.4 by 7.4 gives c. 40% of ducks ingesting lead in a single season. The correction does not take account of ducks that die of lead poisoning before they are shot, or ducks that ingest pellets, survive and ingest again, or ducks that pick up pellets in northern breeding grounds. Most often, birds ingest a single pellet at a time and from a compilation of reports 63% of gizzards with pellets contained only one pellet and 13.6% contained 2 pellets, 0.6% 10 pellets [118].

Ingestion rates measure the exposure of birds to pellets and do not measure lead absorption, as pellets are not always retained and lead is not always absorbed. The amount of ingested lead absorbed and the rate of absorption from the intestine into the blood stream are related to the number of shot ingested and their retention times, the physical and chemical composition and the volume of food ingested, the quantity and characteristics of grit ingested, and the physiological condition of the bird [96].

1.4.2 Measuring lead absorption

As lead is ubiquitous, a diagnosis of abnormally high levels of lead in blood, liver or other organs depends on establishing a threshold below which lead concentrations are deemed to represent normal background exposure. For blood, a background level was established about 30 years ago as <0.2 ppm (20 $\mu\text{g}/\text{dl}$) and 0.5 ppm was considered toxic but sublethal [118]. In a recent (2006) study on condors in California, a

background level of 3.75 $\mu\text{g}/\text{dl}$ blood lead was used, substantially lower than the previous criterion [23]. The higher level was established when environmental lead exposures were greater than they are now and before there was widespread recognition of the precautions necessary to avoid sample lead contamination during collection, processing, and analysis.

Once in the bloodstream, lead may be deposited in other organs within 24 h of exposure and the liver, in particular, may show elevated lead levels for several months if a large amount of lead has been absorbed. Thus, liver lead concentrations are used commonly to measure exposure and to separate background from abnormal levels. The livers of 301 waterfowl from 16 different species were analysed to obtain background levels of metals, including lead, from a relatively unpolluted area of the Ouse Washes, England [104]. The birds were collected from 1971-3 and had all been shot by wildfowling. In 86% of the samples, the lead concentration was $<0.6 \mu\text{g}/\text{g}$ dry weight (dw) and in 5.2% it was $>6 \mu\text{g}/\text{g}$ dw. Later, other investigators chose either $\leq 10 \mu\text{g}/\text{g}$ dw [91] or $<6 \mu\text{g}/\text{g}$ dw [96] to indicate background exposure. Background liver lead levels were established for 28 bird species collected in Maryland, USA [8]. The birds had no shot in their gizzards and none showed signs of lead poisoning: average concentrations ranged from 0.5-3.3 $\mu\text{g}/\text{g}$ wet weight (ww). A concentration of 38 $\mu\text{g}/\text{g}$ dw (10 $\mu\text{g}/\text{g}$ ww) has been suggested as a defensible criterion for identifying lead-poisoned waterfowl in the absence of pathological observations [14]. It should be noted that authors sometimes quote wet weights or dry weights. Use of wet weights can produce sizeable errors as the degree of drying can be difficult to quantify and it has been recommended that drying to constant weight is necessary to produce reliable data [1]. An approximate conversion factor is 1 $\mu\text{g}/\text{g}$ wet weight = 3-4 $\mu\text{g}/\text{g}$ dry weight [96].

Bone lead reflects accumulation over a lifetime from all sources and a threshold value may be seen as entirely arbitrary. Average background lead levels in the bones of 13 bird species ranged from 2.0-13 $\mu\text{g}/\text{g}$ ww [8]. Some investigators have used $>20 \mu\text{g}/\text{g}$ dw to indicate elevated exposure [157], while others have used a lower limit of $>10 \mu\text{g}/\text{g}$ dw [96].

Data on lead residues are often highly skewed – many authors calculate medians instead of means for measures of central tendency. Statistical analyses are often carried out on log-transformed data. Very high individual values are thought to be indicative of contamination of the tissue with embedded shot fragments, some of which may be hard to detect. It has been suggested that birds shot with lead pellets should not be used for lead determinations unless X-rayed before chemical analysis and then measurements should be made on at least two different samples of the tissue examined [44].

An alternative approach to measuring lead levels in various tissues is to measure the effects on enzyme systems. As lead interferes with the production of haemoglobin, one of its precursors, protoporphyrin (PP), accumulates in the erythrocytes where it forms a complex with zinc. The amount of zinc protoporphyrin present can be measured either directly by a haematofluorimeter or, after extraction from the blood, by spectrophotometry. PP was measured in mallards after the birds had been intubated with a single dose of 8, 2 or 1 No. 4 lead pellets, each weighing 190-210 mg [113]. Blood lead concentrations were also determined. In control birds, free of lead shot, PP

concentrations did not exceed 40 µg/dl during the 5-week trial and blood lead levels did not exceed 0.40 ppm. In the treated birds, PP concentrations peaked 8 days post-treatment, 6 days later than peak lead levels in the blood. The mean PP concentration on day 8 for birds given 8 pellets was 1070 µg/dl and for birds given a single pellet 129 µg/dl. PP levels were also measured in 1,137 mallards, 264 canvasbacks (*Aythya valisineria*) and 864 Canada geese live-trapped in up to 4 different areas and related to blood lead levels in order to compare exposure rates by the different methods [3]. Threshold levels were 0.5 ppm for lead in blood and 40 µg/dl (0.4 ppm) for PP. In mallards, an average of 8.1% of samples equalled or exceeded the threshold for blood lead and an average of 3.9% equalled or exceeded the threshold for PP. Corresponding figures for Canada geese were 6.5% and 3.1% and for canvasbacks 5.7% and 3.8% respectively. The differences between blood lead and PP were significant ($p < 0.05$) for mallards and Canada geese and probably relate to blood lead levels peaking sooner and remaining elevated longer than PP [113]. As factors other than lead can result in the accumulation of erythrocyte protoporphyrin, enhanced PP is not by itself proof of elevated blood lead concentration.

Lead interferes with the synthesis of haem, a precursor of haemoglobin, probably by blocking particular chemical sites on the enzymes. One particular enzyme (ALAD – δ -aminolevulinic acid dehydratase) appears to be especially sensitive and its activity in blood falls when the blood lead concentration rises to around 20-40 µg/dl [131]. ALAD activity was depressed by nearly 80% in bald eagles which had been force-fed lead shot [59]. This occurred within 24 h of ingestion and paralleled a rise in blood lead from < 0.1 ppm to a mean concentration of 0.8 ppm. In mallards force-fed lead shot, ALAD activity had decreased by 53% one month after dosing and was 30% below control levels after 3 months [41]; at 4 months post-treatment activity had returned to normal in some birds. In a modification of the technique, ALAD activity was expressed as the ratio of activated:non-activated [119]. This simplified comparison of values between widely different species, because it did not depend on an absolute measure of enzyme activity, which might vary significantly among species or even among individuals of the same species. In birds exposed to normal background lead levels, activity ratios should be close to unity. In 19 mallards with blood lead levels ≤ 15 µg/dl (background exposure), the activity ratio was 1.19 ± 0.15 . In 21 birds with a mean blood lead of 251 µg/dl and considered to have a highly elevated exposure, the ratio was 17.7 ± 9.1 .

2. Species at risk

2.1 Waterfowl

The waterfowl species that is commonly reported as being poisoned with lead shot is the mallard (*Anas platyrhynchos*), perhaps because it is one of the commonest and most widespread of ducks that inhabits wetlands. Game-reared mallards have often been used as experimental animals to test their susceptibility to the constituents of different types of shotgun pellet (e.g. [64] [70] [117]). However, any species of waterfowl that visits a wetland where hunting occurs is at some degree of risk. Examination of gizzards from 11 species shot by wildfowling on the Ouse Washes in Cambridgeshire and Norfolk 1968-1973 found ingested pellets in 6 species [140]:

mallard, pintail (*Anas acuta*), shoveler (*A. clypeaia*), teal (*A. crecca*), moorhen (*Gallinula chloropus*) and snipe (*Gallinago gallinago*). A more extensive survey of British wildfowl was carried out during the 1979/80 and 1980/81 shooting seasons [91]. Ingested pellets were found in the gizzards of 13/24 species shot by hunters, or found dead, including the mute swan (*Cygnus olor*), pink-footed goose (*Anser brachyrhynchus*), pochard (*Aythya ferina*), tufted duck (*A. fuligula*) and goldeneye (*Bucephala clangula*).

Incidences of ingested pellets do not measure how much lead is absorbed from the gut and how many birds are likely to die. Experiments with captive birds have been carried out in an attempt to provide data on this relationship, although it has been argued that such studies produce only tenuous results, as conditions in the wild can never be truly replicated in captivity [118]. Also, it is difficult for ethical and logistical reasons to establish full dose-response relationships, particularly for species that are already endangered. However, the few studies that have been carried out may be useful in determining which species are likely to be most at risk and those which are at a lower risk.

2.1.1 Susceptibility to poisoning

Several studies have been conducted to determine the quantities of lead which produce toxic effects and the speed with which symptoms appear. Wild-caught adult mallard were intubated (a flexible tube inserted through the mouth down into the gizzard) with lead in a soluble form, lead nitrate, at dosage levels of 3, 6, 8 and 12 mg of lead/kg body weight daily until death [24]. The birds were maintained on a diet of cracked corn (maize) and wheat. A daily ingestion of 6 mg/kg lead for 137 days apparently had no effect, but 8 mg/kg/day killed 6/6 birds in an average of 29 days and 12 mg/kg/day killed 6/6 birds in 25 days. Poisoned birds displayed a marked lethargy after 3-4 days on the 8-12 mg/kg dose followed by 'wing-drop' on day 6. Paralysis of both legs and wings was observed in several birds 1-2 days before death. Green bile staining of the feathers in the ventral region was also seen just before death. At post mortem birds were anaemic and emaciated with an absence of fatty tissue – at death the birds had lost, on average, 42% of their initial body weight. There was a reduction in the size of the liver and atrophy due to necrosis. There was excess bile in the gall bladder and duodenum and some of it had regurgitated into the gizzard and proventriculus. Gizzard muscles were atrophied and other muscles, including those of the heart, were flabby. The red cell (erythrocyte) count was low and cells were malformed and small.

Other studies have used a different, and perhaps more realistic, protocol to determine lead shot toxicity. This involves dosing separate groups of birds with different numbers of lead shot. The shot size chosen is regarded as one commonly used against the species under study. A control group is normally included. Ten captive-reared, adult black ducks (*Anas rubripes*) were dosed with a single No. 4 lead pellet [100]. Six birds died within 6 days of dosing, all showing signs of acute lead poisoning. The level of mortality was unexpectedly high, especially as the birds were fed a balanced diet high in calcium, but as the control birds lost weight as well as the dosed birds, it was suggested that some unknown stress factor had rendered the treated ducks more susceptible to lead poisoning.

In contrast, groups of wild black ducks and mallards were intubated given with a single dose of either 2 or 5 No 6 pellets and another group with 1 pellet every 3-4 days for 2 weeks (5 pellets in total) [22]. Mortality for black ducks receiving a single dose of 2 or 5 pellets was 1/4 and 2/4 respectively and for mallards 0/6 and 2/6 respectively. For both species the mortality for the chronically dosed groups was 40%. These rates were regarded as low and attributed, in part, to the diet of animal foods and aquatic vegetation, which contained high levels of calcium; ducks that feed on cereal grains appear to be more susceptible to lead poisoning. Mortality was also directly related to the length of pellet retention, which varied considerably among individual birds: retention was shortest for asymptomatic birds, intermediate for symptomatic birds and longest for dead birds. The effect of diet confounds the interpretation of toxicity studies, in which a mallard may succumb after ingesting a single pellet, while another appears outwardly normal after ingesting 32 pellets [38].

Canada geese (*Branta canadensis*) were given a single dose of 2, 5, 10, 25, 50 or 100 No 4 lead pellets [26]. A goose on the lowest dose showed no overt symptoms, but other birds on higher doses did so in 5-7 days. Symptoms were similar to those given above for mallards: weakness, lethargy, anorexia, green diarrhoea, loss of weight, but also swollen heads. Post-mortem findings were also similar: impaction of proventriculus, green-stained gizzard lining, distended gall bladder, discoloured liver and flaccid heart. Days to death was inversely related to dose: 6 days for geese given 50 or 100 shot up to 72 days for a goose given 5 shot. It was concluded that 4-5 No 4 lead pellets were a lethal dose to Canada geese. The erosion rate of pellets was monitored by X-ray in one bird which retained all 5 shot in its gizzard: 65% of pellet volume was eroded in the first 3 days, 99% was gone by day 37 and pellet material was undetectable by day 48. Other birds passed pellets into the intestine but did so mostly within the first 3 days and rarely thereafter. The authors suggested that in 'acute' lead poisoning a bird may appear to be in good condition but will likely have pellets in its gizzard. If no pellets are found, the level of lead in the blood should be abnormally high. The presence of pellets in the gizzard is not, by itself, conclusive of lead poisoning.

In contrast to ingested lead pellets, embedded pellets produce no long-term ill-effects beyond those associated with the initial injury when the pellets are fired in. Five No. 4 lead pellets were implanted 1.3 cm deep into the breast muscles of 10 game-farm mallards [117]. The birds were monitored for one year and the pellets recovered at the end of the trial. Each pellet was encapsulated by connective tissue and appeared unchanged from the date it was embedded.

2.1.2 Ingestion rates – before a lead shot ban

Birds ingest spent pellets either by mistaking it for food or as grit. Grit facilitates the mechanical grinding and pulverisation of food in the gizzard and may also provide additional minerals, especially calcium [49]. After examining 90 species of North American (non-waterfowl) birds, a log-linear relationship was found between mean grit size and bird body mass (log₁₀), but a similar relationship was not found in 9 species found on Mediterranean wetlands [40]. Gizzards of granivorous birds contained more grit particles than those of insectivores, omnivores and frugivores.

For waterfowl, factors influencing the ingestion of pellets include feeding habits, pellet settlement rates, depth of water, size of pellets and season. Magpie geese (*Anseranas semipalmata*) are particularly susceptible to ingesting pellets as the birds feed on tubers of sedges which are of similar size to lead shot [158]. In a sample of shot birds collected in 1988-9, 21.4% (n=103) had ingested pellets in the gizzard. These birds sometimes dig down 20 cm or more into sediments to find food – in this study in Australia, clay soils prevented pellets sinking beyond reach into the sediment. Mallards are prone to ingest pellets as they feed extensively on cereal grains and weed seeds [118]. In wetlands that are hunted intensively, diving ducks may be more exposed to pellet ingestion than dabblers (intermediate) or grazers because of the high pellet density in the sediments [40]. However, the risk to individual species may depend on local factors. In the Ebro Delta, Spain during 1992-3, 213 hunter-killed waterfowl were examined [86]. The species with the highest percentage of birds with pellets in the gizzard was a dabbling duck, the northern pintail (*Anas acuta*) 70.8% (n=24), while in a diving duck, the red-crested pochard (*Netta rufina*), prevalence was 20% (n=20). It was suggested that in the shallow waters of the Ebro Delta, the feeding methods played a less important role in pellet ingestion.

In the USA, high water in spring may decrease the availability of lead and if pellets then sink into soft sediments it may become unavailable to waterfowl in the following season [118]. The prevalence of ingested shot increases during the hunting season (October-January) in the USA [148] [157], presumably linked to the large amount number of fresh pellets deposited in the sediments as the season progresses. However, this is not always the case and at some sites prevalence rates have been higher before the season opens than when it ends [110] [162]. This trend has also been observed in the UK (inland duck shooting season September-January) [91] [140] and may be due to changes in feeding/gritting behaviour as a result of disturbance at the start of the shooting season or that autumnal rains raise water levels putting spent pellets out of reach of some birds. While it is unknown whether or not lead-shot ingestion affects waterfowl adversely at the population level, its impact on a particular species is largely determined by factors operating at a local scale [40].

Ingested pellets were found in 9% of 380 mallards shot on the Ouse Washes between 1968 and 1973 [140]. In 10 other species examined, the highest incidences were 10.1% in 89 pintail, 6.3% of 32 moorhens and 3.2% of 278 teal; no ingested pellets were found in 220 wigeon (*Anas penelope*). Of the duck gizzards with ingested pellets, 63% contained one pellet and 15% had two: mallard and pintail had the highest average pellet counts, 2.3 and 3.1 respectively. It was estimated that 400-700 waterfowl (3-5%) had died of lead poisoning annually out of an average total population of about 14,500. In the 1979/80 and 1980/81 shooting seasons, 4.2% of 820 mallards shot in different parts of the UK contained ingested pellets [91]. In other wildfowl, the highest incidences were 11.8% in 17 gadwalls (*Anas strepera*), 11.7% in 77 tufted ducks, 10.9% in 64 pochards and 7.1% in 42 greylag geese (*Anser anser*). In wildfowl found dead, ingested pellets were found in 10% of 20 Bewick's swans (*Cygnus columbianus*) and in 2/5 Whooper swans (*C. cygnus*). Of the 86 birds from all species that had ingested spent pellets, 53% contained only one pellet. It was estimated that 2.3% (about 8000 birds) of the British wild mallard population would die each winter following lead shot ingestion.

2.1.3 Ingestion rates – after a lead shot ban

Legislation came into force in 1999 in England prohibiting the use of lead shot for shooting all quarry species of wildfowl, including mallard, anywhere in the country, and for any shotgun shooting on specified inland and coastal wetland sites. In the US, non-toxic (i.e. lead-free) shot zones were established in portions of the Mississippi Flyway beginning in 1977, and in 1991 mandatory use of non-toxic shot for waterfowl hunting went into effect throughout the United States [116]. Non-toxic shot zones first appeared in Canada in 1990 and 1991. A national regulation in 1999 prohibited the use of lead shot for the purpose of hunting all migratory game birds anywhere in Canada (except for upland species - American woodcock (*Scolopax minor*), mourning doves (*Zenaida macroura*), and rock doves (*Columbia livia*) [120]. In addition to these countries, by 2007 prohibitions on the use of lead shot over some or all wetlands had been imposed in Belgium, Cyprus, Denmark, Germany, Hungary, Latvia, Netherlands, Norway, Spain, Sweden and Switzerland [84]. Similar bans have also been imposed in Australia and New Zealand.

In the USA, the effect of local and national lead shot bans on waterfowl has been determined in several studies. A local ban on lead shot was imposed at a pond in Colorado where in 1973-4 925 Canada geese had died of lead poisoning [138]. The geese had apparently obtained the spent pellets from agricultural fields surrounding the pond. In the following two hunting seasons, poisoning was substantially reduced even though some lead shot was still used, but it was suggested that changes in land surface conditions (and therefore pellet availability) might have had a greater impact than the regulation. In another study carried out in 1982-85 in Kansas, pellet ingestion rates in mallards were compared in relation to wetlands where, since 1980, a lead shot ban was in force and those where lead could still be used [48]. In 754 gizzards from birds killed by hunters on wetlands with the ban, 4% contained steel pellets and 3.2% had lead pellets. At two sites with no ban, the respective incidences were 1.4% and 5.6% (n=72), and 0.9% and 10.2% (n=108). It was concluded that the ban on lead shot had reduced the mortality rate from lead poisoning from 1.97% to 1.03%.

In a compilation of data obtained from states along the Mississippi Flyway during the hunting seasons of 1977-79 just after a non-toxic shot regulation had been imposed, it was concluded that the severity of pellet ingestion and the potential for lead poisoning had not changed appreciably since a previous analysis of data collected between 1938 and 1954 [4]. It was thought that the full effects of the ban would not become evident until spent lead pellets were no longer available to waterfowl in all states in the flyway. Where bans were imposed locally, it was not possible to determine the site of lead pellet ingestion conclusively [110]. Lead exposure found at one location might have been caused by pellet ingestion at one or more other locations. To overcome this, captive-reared, wing-clipped mallards were used as sentinel animals to establish site-specific risks of lead poisoning [110]. The probabilities of birds dying of lead poisoning were compared among 3 wetlands over a 4-year period 1986-89. In one site (P8), open for hunting, a lead-shot ban had been imposed in 1986; in two others (T19, TF) hunting was allowed on adjacent land, although it had ceased in 1973 around one wetland (T19). Pellet densities were estimated at >2,000,000/ha in P8, >173,000/ha in T19 and <16,000/ha in TF. Only lead pellets were recovered from sediment samples in T19 and TF; in P8 3/309 pellets

were steel. The likelihood of birds dying differed between P8 and TF in all years and differed between P8 and T19 in 1986 and 1989, but not in 1987-8. It was thought that the clay soils prevented pellets settling below the level of availability for mallards and demonstrated that lead poisoning can occur long after hunters switch to non-toxic shot.

A further survey was carried out in the Mississippi Flyway in 1996-7, 5-6 years after the nationwide ban (1991) on lead shot. In 15,147 gizzards from mallards shot by hunters, 8.9% contained ingested pellets [5]. Of these (1,345 gizzards), 68.2% contained only non-toxic pellets and it was suggested that while the shot ingestion rate had not changed significantly, ingestion of toxic pellets had been reduced by about two-thirds. Even though it was found that hunter compliance with the non-toxic shot regulations was almost 99%, it was likely that ducks were still ingesting pellets that had been deposited before the ban. Nevertheless, the mortality rate from lead poisoning in the flyway had declined from 4% in 1938-54 to $\leq 1.44\%$ in 1996-7. Diving ducks, which feed deeper into sediments, would have been more likely to pick up old pellets than dabbling ducks, but, overall, it was estimated that 1.4 million of 90 million ducks in 1997 were spared from fatal lead poisoning.

No references to similar surveys on UK wetlands were found.

2.2 Game-birds

In the UK, all hunted species are potentially at risk of ingesting spent pellets, such as the grey partridge (*Perdix perdix* – [107]), red-legged partridge (*Alectoris rufa* – [18], red grouse (*Lagopus lagopus scoticus* – [146] and ring-necked pheasant (*Phasianus colchicus* – [19]).

2.2.1 Susceptibility to poisoning

Groups of willow ptarmigan (*Lagopus lagopus*) were dosed with 1, 3 or 6 No 6 pellets; each pellet weighed approximately 100 mg [50]. Two out of 9 birds in the 3-pellet group and 2/9 in the 6-pellet group died in 8-15 days. Symptoms and post-mortem findings were similar to those seen in mallards [24]. About 50% of the pellets were excreted in the faeces in 6 days and 93% in 12 days. After expelling pellets, the body weights of surviving birds increased. The authors suggested that as ptarmigan appear to have a strong ability to void lead pellets through their digestive system, provided pellet deposition rates are low in their habitat, free-living birds are unlikely to suffer lead poisoning.

Although not game-birds, passerines living close to a trap and skeet range in Maryland, USA were poisoned by lead, but the lead source (pellets, soil, or diet) was not determined [155]. To demonstrate that spent pellets were a potential source, captive cowbirds (*Molothrus ater*) were dosed with one No. 7.5 lead pellet. One group of 10 birds was dosed with a new pellet and another group of 10 birds was divided in two with one half dosed with a new pellet and the remainder dosed with a weathered pellet retrieved from the shot-fall zone. The first group was fed a commercial diet and the second group was given the same diet but blended with a commercial wild-bird

seed mix, cracked corn and grit. In the first group, no birds died or showed overt signs of poisoning. In the second group, 1/5 birds dosed with new pellets and 2/5 dosed with weathered pellets died one day post-treatment and the surviving birds all showed symptoms of poisoning. In the first group, 9/10 pellets were excreted within 24 h of dosing; in the second group, all the survivors all excreted pellets within 1-2 days. The erosion of the weathered pellets was significantly greater than the erosion of the new pellets and it was suggested that as the lead salts in the pellet crust were more soluble than metallic lead, they would be more easily removed in the gizzard. However, the rapid voiding of pellets by song-birds was likely to offer some protection from poisoning, although the high erosion rates might offset the benefit.

2.2.2 Ingestion rates

Shot ingestion rates have been recorded for game-birds in the UK. In one study, post-mortems on 1,318 grey partridges found dead between 1947 and 1992 were carried out to determine the cause of death [107]. Lead poisoning, presumably following pellet ingestion was diagnosed from signs of extreme emaciation, green staining of the gizzard and gut contents, necrosis of the gizzard lining and anaemia. The mortality rate attributable to lead increased from 0.3% in the period 1947-58 to 3.4% in 1963-1992. In the latter period, 4.5% of 446 gizzards of adult birds contained lead pellets, which was 52% of the ingestion rate of 8.6% recorded for waterfowl 1979-81 [91]. In another review of historical data, only one of 637 (0.16%) red-legged partridges autopsied between 1933 and 1992 was found with at least one ingested pellet in its gizzard [18]. The birds originated from shooting estates across Great Britain and over such a long time period different investigators were involved and some ingested pellets might have been missed, particularly if lead poisoning was not suspected. A sample of 144 hunter-killed partridges was collected during the 2001-2 season and ingested pellets were found in 2 gizzards (1.4%). Ingestion rates for ring-necked pheasants (*Phasianus colchicus*) collected between 1996 and 2002 from 32 shooting estates in 11 English counties were determined [19]. In 1996 and 1997 birds were shot with rifles in April of each year, but in 1999-2000 and 2001-2 random samples were taken from hunter-killed birds. In total, 437 pheasants were examined and 3% of gizzards contained shot: there were no differences among years, seasons or sexes.

In North America, ingestion rates for some game-bird species have been recorded. Between 1996 and 1998 in Quebec, Canada, the gizzards of hunter-killed rock and willow ptarmigans (*Lagopus mutus*, *L. lagopus*), ruffed grouse (*Bonasa umbellus*) and spruce grouse (*Dendragapus canadensis*) were examined, but ingested pellets were only found in 1.2% of 155 ruffed grouse [111] – sample sizes for the other three species ranged from 22-46. Gizzards of mourning doves (*Zenaidura macroura*) were collected from birds shot on public hunting areas in Tennessee, USA [77]. Twenty-three of 1,949 gizzards (1.1%) contained ingested pellets: 14 contained 1 pellet, but one contained 24 pellets. A similarly low ingestion rate of 0.3% (2/574) was recorded for mourning doves shot by hunters during 1998-99 at a managed shooting field in Missouri, USA [126]. However, at another site where only non-toxic shot could be used, 15/310 (4.8%) had ingested pellets with 6 birds ingesting ≥ 7 pellets. This difference in prevalence between the two sites indicated that doves feeding in fields where lead shot was used might have succumbed to acute lead poisoning and thus

were unavailable to hunters, resulting in an underestimate of ingestion rates. Much higher rates of ingestion were found in common pheasant (*Phasianus colchicus*) (16/47- 34%) and chukar (*Alectoris chukar*) 6/76 – 8%) that were harvested on a private island in southern Ontario [74]. The number of pellets/fragments found in pheasant gizzards ranged from 1-66 and 5% of the gizzards contained >10 pellets. The high ingestion rates were most likely due to the high pellet density (560,000/ha on hunting fields - >82,000,000/ha on a clay pigeon range in the top 10 cm of soil [60]), and to the birds being farm-reared rather than (presumably) wild.

2.3 Raptors

Around 1980, it emerged that raptors might be at risk of poisoning from the lead in spent ammunition. In the UK, falconers' birds and casualties from wild bird hospitals were examined between 1979-1982 [81]. Lead pellets were found in the stomach of a buzzard (*Buteo buteo*) and a captive North American peregrine (*Falco peregrinus anatum*). Two captive snowy owls (*Nyctea scandiaca*) died of lead poisoning and the likely source of the lead was bullet fragments in trimmings from the carcasses of deer that had been fed to the birds. From 1981-1992, 424 bodies from 16 species of British birds of prey found dead from accidents, starvation or other causes were examined [101]. For some species, the sample size was too small to draw conclusions, but using arbitrary criteria to distinguish background lead levels from acute exposure, 1/26 peregrines (*Falco columbarius*) and 1/56 buzzards were likely to have died of lead poisoning following shot ingestion. Some sparrowhawks (*Accipiter nisus*) had liver lead levels that were suggestive of exposure to lead shot. Red kites (*Milvus milvus*) reintroduced into England were exposed to lead from shot in the diet, but it was considered unlikely that poisoning from lead at the levels recorded threatened the conservation programme [97]. Raptors including those considered to be endangered such as the Spanish Imperial eagle (*Aquila adalberti*), white-tailed eagle (*Haliaeetus albicilla*) and California condor (*Gymnogyps californianus*) are particularly at risk to any additional mortality as these species are generally long-lived and slow-breeding and are vulnerable to increases in adult mortality, especially if the populations are already small [42].

2.3.1 Susceptibility to poisoning

In contrast to waterfowl and game-birds, predatory and scavenging birds can expel shot with the indigestible parts of their prey in the form of regurgitated pellets. American kestrels (*Falco sparverius*) were fed 1 No. 9 pellet each day for 60 days [134]. Each lead pellet was placed inside a dead mouse. The birds regurgitated the pellet the day after dosing by which time 2% of the pellet (≈ 1 mg lead) had been eroded by the birds' digestive system. No birds showed any overt signs of poisoning and none died. However, it was not always certain that each bird had ingested the pellet it was offered and perhaps only $\frac{1}{3}$ to $\frac{1}{2}$ of the pellets offered were eaten. In another test by the same investigator, biologically-incorporated lead (mallards that had died of lead poisoning) was fed to kestrels each day for 60 days. The dose was estimated to be 29.3 ppm/day. Similarly, no birds died or showed overt symptoms of poisoning. However, in both tests the treated birds showed elevated levels of lead in the liver and bones compared with controls.

To ensure a more uniform exposure, kestrels were fed diets containing 10 ppm or 50 ppm powdered lead for at least 5 months and no significant gross lesions were found at autopsy; kidney and liver weights did not differ from those of controls [45]. In another test with biologically-incorporated lead, kestrels were fed baits containing 0.5, 120, 212 or 448 ppm (dry weight) lead 6 times a week over 60 days [30]. Birds tolerated the equivalent of 28 mg/kg/day of lead with no weight loss, anaemia or other clinical signs. It was suggested that biologically-incorporated lead, in this case cockerels fed a diet containing 2000 ppm lead (as lead acetate) for 4 weeks, might have been less available as most of the lead was probably in bone tissue rather than in the liver and kidney of the chicken.

Each of 5 bald eagles (*Haliaeetus leucocephalus*) was intubated or force-fed 10 No 4 pellets [105]. Additional doses of 10 pellets were given if all pellets from the first and subsequent doses were regurgitated. In total, the 5 birds were given, respectively, 10, 20, 30, 80 and 156 pellets: there was considerable variation in the regurgitation rate of pellets from 12 hours to 48 days. Four of the 5 birds died in 10-125 days and the fifth became blind and was euthanised on day 133. By the time the birds died they had lost 16-23% of their pre-treatment body weight and on post-mortem they were emaciated with cardiovascular and renal lesions. The authors suggested that lead levels in the liver >10 ppm and kidney >5 ppm (ww) of bald eagles indicate acute exposure. They also suggested that the fate of an individual eagle ingesting shot was difficult to predict as it was dependent on retention time, number of shot retained, the amount of lead eroded and individual susceptibility.

2.3.2 Exposure to poisoning

The risk to scavengers is increased if carcasses of shot animals are not retrieved. This is particularly likely if the bodies have no economic value such as pest species. Richardson ground squirrels (*Spermophilus richardsonii*) are controlled by poison baits and shooting and the bodies of shot squirrels are not usually recovered leaving them available to scavenging birds of prey [72]. The distribution and amount of lead in squirrels shot with .22in hollow-point bullets was assessed. Radiographs of 15 carcasses showed that 14 had visible bullet fragments that were mostly present as minute particles. Excising the tissue along the bullet tract and analysing for lead content gave a median value of 3.23 mg per carcass (range 0.01-17.21 mg). It appeared that a proportion of the bullet had exited the body in each case. The study considered the risk to two scavenging hawks (*Buteo swainsonii*, *B. regalis*) and the authors speculated that a hawk could receive a lethal dose if it ate 6.5 carcasses over 23 days. The dust-sized lead particles found in the muscle of the shot squirrels had a large surface area, which would facilitate dissolution and absorption making them less likely to be regurgitated. It was suggested that any risk to these and other scavengers could easily be reduced by retrieving all shot animals, or using lead-free bullets if available. Another option that has been suggested for shooting burrowing squirrels still retains lead but uses a non-expanding full-metal-jacket bullet. Only 7% of 29 prairie dogs (*Cynomys ludovicianus*) shot with such a bullet contained fragments compared with 87% of 30 prairie dogs that contained fragments after being shot with an expanding bullet (lead core exposed at bullet tip) [106]. The mean weight of fragments in carcasses shot with the non-expanding .223in bullet (original weight

3.554 g) was 43 mg, of which 19.8 mg consisted of core material (98% lead), compared with 317.8 mg of fragments from the expanding .223in bullet (original weight 3.558 g), of which 235.7 mg consisted of core material; on average, 73.1% of fragments from the expanding bullet weighed <25 mg each.

It is common practice for hunters to remove the internal organs (gralloching) from deer that have just been shot. The purpose is to reduce contamination of the meat by the gut contents, especially if the gastrointestinal tract has been damaged by the bullet. Also, draining the blood by cutting the main blood vessels will reduce tainting of the meat. The excised organs are often left behind when deer carcasses are taken away to be butchered. These offal piles, which may contain bullet fragments, are then likely to be scavenged. In addition, whole carcasses from deer that have been wounded, but not recovered, may eaten by scavengers – around 10% of shot deer were not recovered in one study [94]. Whole or partial remains of 38 deer (*Odocoileus virginianus*, *O. hemionus*) shot with rifle bullets were radiographed [61]. The bullets ranged in weight from 100 to 180 grains (6.5-11.7 g) and 34 were copper-jacketed, lead-core projectiles and 4 were monolithic copper expanding ‘X-bullets’. Shot distances varied from 37 to >200 m. In 18/20 offal piles, where a leaded bullet had been used, 160 fragments (range 2-521), on average, were identified. There were 416-783 fragments in 5 whole deer carcasses, 25-472 in 10 carcasses containing thoracic organs but no abdominal viscera and 38-544 in 9 eviscerated deer. The fragments were mostly small (<2 mm), some estimated to be about 0.5 mm. The copper ‘X-bullets’ did not fragment to the same extent – a total of 6 fragments were found in 4 whole deer and in only one offal pile. Given the small size and irregular shape of the bullet particles and the acidic conditions in raptors’ stomachs, it was suggested that offal piles and unretrieved wounded deer should be considered as potentially poisonous to avian scavengers. Food-related mortality was thought to be responsible for the deaths of 3/14 wild California condors probably from carcasses of mule deer (*Odocoileus hemionus*), coyotes (*Canis latrans*) and California ground squirrels (*Spermophilus beecceyi*) [159]. Of the 14 condors, 5 (36%) had elevated lead levels (>0.7 ppm) in their blood and a copper-coated lead bullet fragment weighing 0.176 g was found in the gizzard of one bird. Using isotope ratios, the lead in the blood of 18 wild condors closely matched the lead in ammunition [23].

In bald eagles (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*) the main source of lead is believed to be lead ammunition ingested by or embedded in the tissues of waterfowl, other game-birds, small and large mammals. Eagles of these two species found dead in the Canadian prairie provinces were sent to wildlife agencies and examined for lead exposure [156]. Of 127 birds autopsied from 1990-96, 17% had elevated lead concentrations based on kidney levels above 8 ppm dw or liver levels above 6 ppm dw; 12/127 (12%) had kidney concentrations above 20 ppm dw or above 30 ppm in the liver and were classified as lead poisoned. Although the prevalences of lead poisoning in the two species were similar, poisoned bald eagles were mostly found in areas of high waterfowl hunting intensity while poisoned golden eagles were found in areas of low waterfowl hunting activity. This suggested that bald eagles probably acquired the lead from waterfowl and the golden eagles from non-waterfowl game-birds and mammals. The effect of lead poisoning on the populations of these birds was unknown and the bodies examined were not a random sample and the prevalence figures were probably biased as they depended on people finding dead birds, perhaps by chance, and then sending them for examination.

Similarly, injured bald and golden eagles admitted to The Raptor Center in Minnesota, USA provided information on the incidence of lead poisoning before (1980-90) and after (1991-95) the lead-shot ban for waterfowl hunting [73]. Eagles were sent in from 11 states and lead levels in blood were classified on the basis of <1 ppm indicating chronic exposure and >1 ppm acute exposure. The prevalence of lead poisoning before the ban was 17.5% (n=193) and after the ban 26.8% (n=134), but this difference fell short of statistical significance (p=0.08). However, fewer birds were found with acute poisoning after the ban than before (35% before, 24% after), but those with chronic poisoning increased (before 50%, after 64%) – these differences were significant (p=0.007). That the prevalence of poisoning had not decreased after the ban was attributed to the birds finding other food sources (mammals, game-birds other than waterfowl) that were still able to be legally shot with lead ammunition and birds migrating from other countries (in this case Canada) where, at the time of the investigation, a lead-shot ban was not in force.

2.4 Animals on shooting ranges

The potential risk to human health for people using indoor and outdoor shooting ranges has been recognised for some time, but over the last 10 years there has been an increase in studies exploring the ecological implications of lead contamination at small-arms ranges. Such ranges are often intensively used by military and law enforcement personnel and for competitive target shooting resulting in high concentrations of lead and other metals in relatively small areas. No information was found on the number of shooting ranges in the UK, but in 1991 it was estimated that 220 million clay pigeons were released with at least one shot being fired at each [89]. In the USA, there are over 12,000 ranges [65] and over 200 were identified in the state of Ontario, Canada [32]. In addition to raptors and game-birds, species at most risk are those that have home ranges overlapping the shot-fall zones. Shrews (*Sorex araneus*), wood mice (*Apodemus sylvaticus*) and bank voles (*Clethrionomys glareolus*) collected from an abandoned range were found to have lead levels much higher than those from uncontaminated areas [80]. Other studies have found that green frogs (*Rana clamitans*) and passerines are also at risk [132] [155] and that some plants will absorb lead into their roots [83] [89]. Shrews probably accumulate lead through eating earthworms and it was shown experimentally that one of the end products of lead oxidation in soil, cerussite ($\text{Pb}(\text{CO}_3)_2$), can bioaccumulate in the earthworm *Lumbricus terrestris* [33]. Because metallic lead degrades so slowly, the burden on the ecosystem in and around a range is likely to exist for many years without remediation.

2.5 Other species

Unexpected or unusual cases of poisoning caused by lead ammunition have been reported. Rare woodpeckers (*Picus canus*, *Dendrocopus leucotos*) in Sweden were thought to have picked out pellets shot into trees while they were searching for food [90]. Only 3 birds were found with elevated levels of lead in the liver and testing of another 40 dead woodpeckers of other species found no further cases. In Israel, two captive bottlenose dolphins (*Tursiops truncatus*) were found to have ingested airgun

pellets [127]. The pellets consisted of 40% lead, but also contained copper and zinc. One animal, a male, died and on post-mortem the liver, brain and other organs were abnormal. Liver lead concentrations were elevated and it was concluded that chronic lead toxicosis was the cause of death. The other dolphin, a female, appeared normal, but was treated for lead poisoning and the pellets were removed. The source of the pellets was not known, but could have been children, as spectators in the dolphinarium, throwing pellets to attract the animals' attention.

Contaminated silage has also been responsible for poisoning cattle [46] [108]. In these cases, the fields where the grass was growing had been used for clay pigeon shooting and it was estimated that in one field >3 t of lead had been discharged [46]: 14 g of lead pellets were removed from the rumen of one cow at slaughter. Although early studies had indicated that metallic lead was relatively inert in the ruminant digestive tract, it was suggested that concentrates and silage fed to high yielding dairy cows would lower the rumen pH values leading to the gradual dissolution of pellets. However, samples of the contaminated silage contained soluble lead, which might also have contributed to the lead that was absorbed.

Elevated blood levels (≥ 10 $\mu\text{g}/\text{dl}$) were found in common ravens (*Corvus corax*) living in National Parks in Wyoming, USA [28]. The birds were scavenging hunter-killed large ungulates and their offal piles which were probably contaminated with rifle-bullet fragments. During the hunting season 47% (n=136) of the birds exhibited high blood lead concentrations compared with 2% (n=147) during the non-hunting season. Repeat blood samples taken 2-3 days apart on 7 ravens with high blood levels revealed that when the birds were maintained on uncontaminated food their blood lead returned to baseline in ≤ 2 weeks. This suggested a relatively short half-life *in vivo*, but at the time of the study there were no quantitative data to separate background from abnormally high levels for blood lead in ravens.

An outbreak of lead poisoning in greater flamingos (*Phoenicopterus ruber roseus*) occurred in Spain during November 1992 to March 1993 and November 1993 to February 1994 [85]. Over 100 flamingos were found dead or moribund and 53/57 gizzards examined contained between 1 and 277 pellets and 55/57 livers contained lead concentrations ranging from <2.5-992.2 $\mu\text{g}/\text{g}$ dw (median 192.3 $\mu\text{g}/\text{g}$) – an elevated lead level was taken to be >5 $\mu\text{g}/\text{g}$ dw. Other instances of lead poisoning in flamingos have been reported, but in this case it was thought that adverse weather conditions reduced water levels in specific wetland locations allowing the birds to feed in an area that was normally too deep for them.

3. Risk to humans from spent ammunition

Changes within the last three decades have decreased lead exposure in humans living in Europe, North America and elsewhere through the elimination of leaded petrol, lead in solder and in paint. A report in 1983 concluded that most adults in the UK obtained their lead from food, but it was unclear to what extent the lead was biologically incorporated (e.g. crop plants taking up lead through their roots and transferring it to the edible parts) or the result of deposition (e.g. lead dust adhering to leaf surfaces) [131]. A number of studies looking at the risks to humans from lead-contaminated food have concentrated particularly on communities that depend heavily

on hunting for subsistence. There are also reports on case studies that show that lead in ammunition can be absorbed into the blood stream from the human gastrointestinal tract and can cause clinical symptoms of poisoning.

It has been suggested that birds in the early stages of lead poisoning are more likely to be taken by hunters [12]. If so, such birds might be considered fit for human consumption, while those in the later stages of poisoning would probably be discarded, especially if they appeared emaciated. Thus, some birds with tissue-bound lead could be eaten by humans. Equally, birds not poisoned, but with embedded pellets in the muscles, might also be eaten. Cases of poisoning in humans following lead pellet ingestion have been reported. In one case, a single pellet, lodged in the colon, was found by X-ray [53]; it was 6 mm in diameter, weighed 1.7 g and had probably been embedded in meat from hunter-killed wild boar or deer. The blood lead level was 550 $\mu\text{g/l}$ (normal $<40 \mu\text{g/l}$) and the patient suffered from malaise, fatigue and diffuse gastrointestinal symptoms. After the pellet was voided, the blood lead level returned to near normal within 9 months. Lead pellets can also be retained in the appendix for several years [82]. In 7 patients, each with 1-2 ingested pellets, the blood lead levels were almost twice as high as controls, but did not approach toxic levels. Up to 9 pellets had previously been detected in two of these patients, with some pellets being present for at least 6 years. It appears that the isotonic environment in the appendix and the neutral to alkaline pH result in very slow dissolution and low absorption of lead.

The risk to humans from eating meat with biologically-incorporated lead or embedded pellets or pellet fragments has been considered in several studies. One study collected nearly 4000 hunter-killed birds from 44 species, mostly waterfowl but also game-birds such as ptarmigan and grouse [124]. The birds were collected between 1988 and 1995 in Canada before the nationwide ban on the use of lead shot for taking migratory game-birds. The pectoral muscles were analysed as a representative edible portion of game animals and lead levels $>2 \mu\text{g/g}$ dry weight (dw) were considered to be 'elevated' and $>5 \mu\text{g/g}$ dry weight was regarded as 'high'. An initial analysis was made of tissues pooled by species and location and from those sample pools with high lead concentrations, tissues from 190 individual birds were selected together with tissues from 19 birds from pools with low concentrations. In 40/190 individuals (21%) the lead concentration was high ranging from 5.5 to 3,910 $\mu\text{g/g}$. An important finding was the difference in concentration between the left and right pectoral muscles of an individual bird and even between sub-samples of the same muscle. This strongly suggested that the lead was not biologically incorporated which would give an even distribution throughout the tissue. Although visible lead fragments or pellets had been removed from the samples, radiography detected residual particles of 1 mm or less. It is possible that such small fragments would be easily swallowed by humans and retained within the gastrointestinal tract, whereas larger foreign bodies would be rapidly expelled. An inverse relationship was found between particle size and lead absorption when particles ranging in size between 0 and 250 μm diameter were incorporated into diets and fed to laboratory rats [10]. Blood lead concentrations for rats given particles $<38 \mu\text{m}$ were 2.3 times greater than those of rats fed particles of 150-250 μm . One interpretation of the results was that the relative proportion of lead oxides to metallic lead was higher in the smaller particles and that the oxides were more readily absorbed.

Human populations most at risk of lead poisoning are those that by tradition consume relatively large quantities of game taken by shooting. Game eaten by First Nation Cree in northern Ontario was analysed and included ducks, geese, grouse and shorebirds all taken with lead shot and moose (*Alces alces*) and caribou (*Rangifer tarandus*) harvested with rifles [149]. Other game such as beaver (*Castor canadensis*), hare (*Lepus americanus*) and walleye (*Stizostedion vitreum*) were taken with non-shooting techniques. Lead concentrations in all fish (n=30) and mammal (n=45) samples were below the detection limit of <0.3 µg/g wet weight (ww), but 9% of 371 (edible) muscle samples from birds contained >0.5 µg/g ww with 19,900 µg/g recorded in one Canada goose (*Branta canadensis*). Of 26 muscle samples that were radiographed, 2 contained embedded lead. The lead concentrations probably resulted from contamination of the tissues with shot fragments, but the absence of detectable lead in moose and caribou was questioned on the grounds that the heterogeneous scattering of a fragmenting bullet could easily result in a tissue sample that, by chance, contained no lead.

Similar results were obtained from analyses of tissues from seabirds in Greenland, particularly the thick-billed murre (*Uria lomvia*) and common eider (*Somateria mollissima*) [66] [67]. Lead shot is still used and hunting is mainly for subsistence and seabirds are an important component of the diet. In a sample of 50 murre, the average number of pellets found in each bird was 3.7 (range 0-12) [66]. After all visible pellets were removed, a soup was made from each of 30 skinned, eviscerated carcasses and analysed for lead content along with the boiled pectoral muscles. Lead concentration in the soup was considered low (mean 6.3 µg/l) but the breast meat had a mean of 0.22 µg/g ww, more than 10 times higher than in birds not killed with lead shot. Once again, the source of the lead was probably small fragments ranging from particles 1-2 mm long to very fine dust, which was created during collisions of the pellets with bone. Lead levels in the breast muscles of eiders were 44 times higher than in eiders that had not been shot (6.1 µg/g ww (n=25) v. 0.14 µg/g ww (n=24)) and 8 times higher than in 32 shot murre (0.73 µg/g ww [67]). These levels were compared with a residue guideline for bird meat of 0.3 µg/g ww that existed in Denmark in 2002. As other food items found in Greenland were low in lead, a meal of 200 g of eider meat, which could contain 1220 µg lead, would be close to the FAO/WHO guideline (1993) of 25 µg/kg body weight per week for both adults and children. However, the CONTAM panel of the European Food Safety Authority (EFSA) has concluded that there is no evidence of a threshold for a number of critical effects including developmental neurotoxicity and nephrotoxicity in adults, and that this guideline is no longer appropriate (EFSA Journal 2010; 8(4):1570 - www.efsa.europa.eu).

In a follow-up study, 50 adult men from Greenland gave blood samples to test for lead concentrations [68]. There was a clear relationship between the number of bird meals (61% murre, 29% eider) and blood lead with a mean concentration of 15 µg/l for men that ate no birds up to a mean of 128 µg/l for those eating birds regularly. These results were further supported in a cross-sectional population survey in which participants who ate seabirds less than once a week had blood lead concentrations of 75 µg/l, while those eating seabirds several times a week had concentrations of 170 µg/l [15].

In 2002, it was strongly suggested that 30,000 Spanish waterfowl hunters and their families were at risk of secondary lead poisoning from eating poisoned birds [52]. Since then Spain has extended its ban on the use of lead shot on wetlands [84]. The risk to hunters was assessed from lead levels in the livers of waterfowl shot during the hunting season. Liver is considered a delicacy among many Spanish and other European hunters. Using data collected between 1990 and 1994 on 11 species of Anatidae (e.g. pintail, shoveler, teal, mallard, greylag goose) plus coot (*Fulica atra*) and common snipe, liver lead concentrations were classified in relation to the EU limit of 0.5 mg/kg ww for poultry offal and 0.1 mg/kg ww for poultry muscle and an arbitrary criterion of 5 mg/kg ww. Of 411 birds, 6.1% had concentrations 0.1-0.5 mg/kg, 27.3% 0.5-5 mg/kg and 13.1% >5 mg/kg. The highest concentration was 114.6 mg/kg in the liver of a shoveler duck and half of the 24 pintail livers exceeded 5 mg/kg. Overall, 40.4% of the livers exceeded the EU limit for poultry offal. It was argued that as hunters and their families and friends were likely to eat 'dozens of birds' during the several weeks of the hunting season, there was a significant health risk posed by the increased ingestion of lead. It is interesting to note that the setting of a threshold concentration of lead in the body below which there are no harmful effects was considered meaningless because of the variation in response between individuals [131]: in men, blood lead concentrations >150 µg/dl sometimes produce no obvious symptoms, while symptoms may occur at concentrations <60 µg/dl. Nevertheless, given a general lead burden in the population, it was thought prudent to reduce further exposure to anthropogenic lead in the environment.

While it is possible that the various lead-shot bans in relation to hunting waterfowl have reduced the secondary poisoning risk to humans (or at least not increased it), lead shot is still used to take other game-birds. In Canada, tissues from hunter-killed ruffed grouse (*Bonasa umbellus*), spruce grouse (*Dendragapus canadensis*), rock ptarmigan (*Lagopus mutus*) and willow ptarmigan (*L. lagopus*) were examined for lead contamination [111]. In 256 gizzards from all species, ingested pellets were found only in 2/155 ruffed grouse – a single pellet was found in each case. Mean bone lead concentrations did not exceed 2.13 µg/g for ruffed grouse, willow or rock ptarmigans. Only 1% of 100 ruffed grouse bone samples exceeded 5 µg/g. However, lead concentrations in muscle samples from the ptarmigans were below the detection limit in over 70% of the samples, but in 7 birds the concentration was >5 µg/g. Overall, it was concluded that the lead levels in these birds were not high enough to constitute a risk to human health unless very large numbers were eaten in a short time (≥20 bird meals/month) based on a TDI (tolerable daily intake) for lead of 3.57 µg/kg body weight (Health Canada, 1999, equivalent to the FAO/WHO limit of 25 µg/kg body weight/week).

In contrast, traditional recipes used to prepare meat contaminated with lead have been shown to increase concentrations to a point where the FAO/WHO limit could be exceeded [87]. Lead pellets were manually inserted into the breasts of farm-reared quail (*Coturnix coturnix japonica*). The pellets had been fired from a gun to create the surface distortions that occur during discharge from a firearm and impact on a target. Each breast was embedded with 0, 1, 2, or 4 No. 6 pellets and the experiment assessed the effect of vinegar (6% acetic acid) on the rate of lead dissolution. The presence of vinegar during cooking increased significantly the transfer of lead to the meat and the concentration of lead was positively related to the number of shot. The liquid in which the breasts were cooked had a mean pH of 4.6 ± SD 0.1. After cooking, the lead

concentration did not increase if the meat was stored for up to 28 days. A separate experiment in which single pellets were immersed in either water or water with 3% acetic acid (pH 2.5) and heated to either 25 or 90°C confirmed that acidic conditions and high temperature will dissolve lead. One No. 6 pellet (0.11 g) could release a quantity of lead sufficient to exceed the EU limit of 0.1 µg/g ww for meat if the meat was cooked in vinegar; with 4 pellets, the level could be up to 300 times the limit. It was calculated that one meal of 200 g contaminated meat could exceed the 'lowest observed adverse effect level' (LOAEL) for different age groups (60 µg/day for children ≤6 years old, 750 µg/day for adults). These levels are estimates of the amount of lead that needs to be ingested in order to raise blood lead levels up to 10 µg/dl in children and 30 µg/dl in adults [21]. They reflect that children absorb proportionally more lead from the diet than do adults: respectively, 0.16 and 0.04 µg lead/dl blood per µg lead/day ingested [151]. The experiments with traditional recipes attempted to assess the risk of lead contamination by minimising confounding variables wherever possible. One such variable is the fragmentation of lead pellets as they pass through the body resulting in a heterogeneous distribution of lead in a tissue and different values for lead concentration depending on what part of a sample or sub-sample is analysed. Because of this, some authors claim that the risk to human health is difficult to assess with any degree of confidence [74].

The EU limit for lead in meat (excluding offal) applies to bovine animals, sheep, pigs and poultry, but the regulations do not apply to game meat. To determine the risk to consumers of game meat in Switzerland, blood samples from hunters and family members of hunters were analysed for lead concentration and the results compared with lead levels in samples from blood donors [55]. There was no statistically significant difference in blood lead levels between the two groups, each divided by sex as an additional factor, with the median values less than 6 µg/dl in all groups. The hunters consumed 2.2 wild game meals per week on average, which approximated to a daily intake of 50 g compared with a population average intake of about 1 g of game meat. However, some individuals in the control group had blood lead levels that exceeded 10 µg/dl, which can signify the onset of lead poisoning; the highest reported among the hunters was 17.1 µg/dl. Other than age and sex, there was no background information on the individuals in the control group and the blood samples from the hunters were taken outside the hunting season and at a different time of year to blood from the controls. There is therefore some doubt about the authors' conclusion that game meat did not present consumers with a risk of lead exposure. Similarly, the risk to human health from consuming game-birds that might be contaminated with lead shot was assessed as 'low' and 'non-negligible' from consuming wild ducks [25]. The report did not give the details of how these assessments were made, referring instead to an unpublished account. There was no assessment of the risk to health from consuming wild deer taken with lead bullets.

The fragmentation of bullets on penetrating an animal's body occurs as much by design as by the incidental striking of hard tissue such as bone. Consequently, lead particles can be dispersed far from the bullet's pathway necessitating removal of a large amount of tissue in order to prevent human ingestion of lead in the processed game meat. In Poland, muscle tissue samples were collected from 10 wild boar (*Sus scrofa*) and 10 red deer (*Cervus elaphus*) immediately after they had been shot [36]. The samples were taken from around the entry and exit wounds and along the bullet track and at a radius from it of 5-30 cm. Muscle tissue from parts of the body far from

the wound tract were taken to serve as controls. In one wild boar, >1000 mg/kg ww of lead was found near the entry wound, with >700 mg/kg being recorded near the exit wound and 3.3 mg/kg was found 30 cm from the bullet track. The 'control' tissue in this animal contained 0.3 mg/kg, which exceeded the EU limit of 0.1 mg/kg ww (Commission Regulation (EC) No 1881/2006) for lead in pig meat. Overall, the dispersion of lead through the tissues was highly variable and depended as much on bullet factors (type, expansion index, terminal velocity) as on animal factors (age, body weight, bone hardness, skin thickness).

In a more controlled study, 30 white-tailed deer (*Odocoileus virginianus*) were each shot with the same type of bullet, which weighed 9.72 g and consisted of a lead core (68% by weight) and a copper jacket (32%); lead was exposed only at the tip of the bullet [62]. The eviscerated carcasses were radiographed and between 15 and 409 metal fragments were found widely dispersed in each one. Each carcass was then sent to a commercial meat processing plant and processed into boneless steaks and ground meat packages. Radiography of the 234 meat packages from the 30 deer found at least one fragment in 74 (32%) of which 12 contained 3-8 fragments each. In total, of the 5,074 metal particles found in the carcasses, 155 (3.1%) were found in the meat packages. The average weight of lead from all fragments in each package was 17.2 mg (range 0.2-168 mg) and its likely origin as bullet lead was confirmed by analysis of lead isotope ratios. To test the bioavailability of the lead fragments in meat, contaminated processed venison was fed to pigs as a model for the absorption of lead from the human gastrointestinal tract. Pigs were fed 1.26-1.54 kg of meat containing fluoroscopically visible metal fragments in two feedings 24 hours apart, but the dose of lead received by each pig was unknown; a control group was fed meat with no fluoroscopically visible fragments. Blood samples were taken from all pigs before the trial and on days 1, 2, 3, 4, 7 and 9 post-treatment. The mean blood lead concentrations of treated pigs were significantly higher than those of controls on days 1, 2 and 3, with a maximum observed value of 3.8 µg/dl, but did not differ on days 4, 7 and 9. The reduction in blood lead concentrations in the treated animals was interpreted as sequestration to soft tissues and ultimately to bone. The maximum concentration found in this trial approached a level that could cause adverse effects in humans and even with meticulous care during processing some lead fragments will remain. In the USA and Canada, hunters donate wild game meat to various humanitarian organisations and during the 2007/8 hunting season over 1 million kg was donated [7]. The risk of lead exposure to consumers of this meat depends on a number of variables such as hunting method, ammunition preferences and butchering procedures, all of which may vary regionally and are not yet clearly understood.

4. Identifying the origin of lead in animals.

If birds have lead in their blood or organs and at the same time lead pellets are found in their gizzards, it's a reasonable assumption that the lead source is ammunition. However, if no pellets are found the source of the exposure is less certain and waterfowl have been poisoned by lead in sediments contaminated with mining wastes [16] [128]. Within the last 10 years or so, researchers have begun measuring stable lead isotope ratios to identify the source of lead in exposed animals or at least to eliminate certain potential sources. Lead has 4 stable isotopes, 3 of which ^{206}Pb , ^{207}Pb and ^{208}Pb are produced from the decay of ^{238}U , ^{235}U and ^{232}Th

respectively [34]; the fourth isotope, ^{204}Pb , is not radiogenic. Currently mined ores contain different amounts of the radiogenic isotopes depending on the geological age of the rock and the extent of uranium and thorium deposition. The isotopic compositions of the lead ores are retained in the products that are eventually made from them. The ratio of $^{206}\text{Pb}:^{207}\text{Pb}$ is most commonly used in analytical work and sources differing in ratio by $<0.05\%$ can be distinguished [34].

One of the first studies to use this technique to distinguish sources of lead exposure in wildlife analysed archived tissues from herring gulls (*Larus argentatus*), various species of dabbling ducks, common loons (*Gavia immer*), bald eagles and golden eagles (*Aquila chrysaetos*) and also samples of lead shot ammunition obtained from hunters or retailers [125]. Additional samples included shot pellets recovered from the digestive systems of lead-poisoned trumpeter swans (*Cygnus buccinator*) and a bald eagle and breast muscle samples from game-birds which were thought to contain embedded shot fragments. The herring gulls were chosen as a species that seldom ingests lead pellets or fishing weights and the mean $^{206}\text{Pb}:^{207}\text{Pb}$ ratio in the leg bones was 1.137 ± 0.007 and within the range for lead in petrol in Canada (1.13-1.16). The ratios for waterfowl bones, eagle and loon kidneys and the breast muscles were more variable, but did not overlap the ratios for lead found in Precambrian mining and smelting wastes (0.93-1.07). However, they did match the range of ratios found for different types of lead shot (mean 1.18 ± 0.05 , range 1.07-1.27).

Isotope ratios $^{206}\text{Pb}:^{207}\text{Pb}$ and $^{208}\text{Pb}:^{207}\text{Pb}$ were used to determine the source of lead in dead or moribund marbled teal (*Marmaronetta angustirostris*) and white-headed ducks (*Oxyura leucocephala*), both globally threatened species [137]. The $^{206}\text{Pb}:^{207}\text{Pb}$ ratio found in the livers and bones of adult white-headed ducks did not differ from the ratio for lead pellets found in gizzards of birds collected in an earlier study in the same area in Spain. However, the $^{208}\text{Pb}:^{207}\text{Pb}$ ratios did differ between liver and lead pellets, but not between bone and lead pellets. For marbled teal, the $^{206}\text{Pb}:^{207}\text{Pb}$ and $^{208}\text{Pb}:^{207}\text{Pb}$ ratios in the liver of adult birds and lead shot were similar, but the $^{208}\text{Pb}:^{207}\text{Pb}$ ratio differed between bones and pellets ($^{206}\text{Pb}:^{207}\text{Pb}$ ratios did not differ). In chicks, $^{206}\text{Pb}:^{207}\text{Pb}$ ratios in individuals with low lead concentrations did not match those of shot but as the lead concentration in tissues increased they became similar. This study showed the value of using more than one isotope ratio and as there was some overlap in $^{206}\text{Pb}:^{207}\text{Pb}$ ratios for both lead pellets and soils from the area, the use of isotope ratios alone cannot identify the source of lead without other evidence.

In another study in Canada, wing bones from American woodcock (*Scolopax minor*) were analysed [121]. Ingested pellets are apparently rarely found in woodcock, perhaps because they are quickly voided. The birds eat primarily earthworms and in doing so ingest relatively large amounts of soil. The authors hypothesised that worms and soil could have been contaminated with lead deposited over the years from the combustion of leaded petrol. Hunter-killed woodcock were collected from sites across eastern Canada and the wingbones of 424 young of the year were analysed. Lead concentrations ranged from 1.4 to 280 $\mu\text{g/g dw}$ and in 31% of individuals the concentration exceeded 20 $\mu\text{g/g}$, which is considered to be highly elevated. The isotope ratios of bones with low concentrations were similar to ratios for soils and earthworms sampled from the same area, but lead levels in the soils were mostly low and regarded as uncontaminated (i.e. $<45 \mu\text{g/g}$) with ratios typical of natural geologic

lead. Only a few soil samples were contaminated above background levels and the ratios were consistent with petrol lead. In the bones with $>20 \mu\text{g/g}$, the range of $^{206}\text{Pb}:^{207}\text{Pb}$ ratios was 1.02 to 1.22 with an average of 1.18 ± 0.03 similar to the range found in a previous study [125]. The authors pointed out that this similarity did not prove that lead-pellet ingestion was responsible for the high lead concentrations in woodcock, but they could not think of another likely source.

In the UK, lead isotopic ratios ($^{206}\text{Pb}:^{207}\text{Pb}$ and $^{208}\text{Pb}:^{206}\text{Pb}$) in liver samples of red kites (*Milvus milvus*) were consistent with those in lead shot extracted from regurgitated pellets [97]. Differences in isotopic signatures were detected in the bones of red grouse (*Lagopus lagopus scoticus*) collected from shooting estates in Scotland and Yorkshire [146]. Lead levels in the bones were measured and classified as background exposure ($<10 \mu\text{g/g dw}$), higher than background ($10\text{-}20 \mu\text{g/g}$) and highly elevated ($>20 \mu\text{g/g}$). In birds from the Scottish estates, 24.5% ($n=196$) had lead levels above background compared with 84.2% ($n=38$) from the Yorkshire estate. For birds with low lead levels in both areas, the $^{206}\text{Pb}:^{207}\text{Pb}$ ratios were consistent with those from uncontaminated soil (~ 1.15). For the grouse with high levels, leaded petrol as the source was ruled out as its ratio (~ 1.08) was lower than that found in all the bones. Together with a second isotope ratio, $^{208}\text{Pb}:^{207}\text{Pb}$, the source of lead in Yorkshire birds with highly elevated levels was likely to have been a combination of galena mining and lead pellets – the isotope ratios for many birds were midway between those characteristic for galena lead and those for lead pellets. Historically, lead-ore mining and smelting had been practiced near the Yorkshire estate, but had never occurred on the Scottish estates and thus the signatures for Scottish grouse with highly elevated levels were entirely consistent with those for lead pellets. Speculatively, the deposition of lead pellets over many decades combined with the acidic nature of moorland soils could have resulted in mobilisation and incorporation of lead into the soils, with a consequent shift in the isotopic signatures away from the natural background source towards those for gunshot.

In a conservation project to protect the California condor (*Gymnogyps californianus*), a breeding programme has been set up and descendants of captive – bred birds are being released to supplement the wild population. The success of the project is apparently under threat from lead fragments left in unretrieved carcasses or gut piles that the birds feed on. To obtain direct evidence of this, lead concentrations and isotope ratios ($^{207}\text{Pb}:^{206}\text{Pb}$, $^{208}\text{Pb}:^{206}\text{Pb}$) were measured in 18 condors living in the wild and in 8 pre-release birds and in diet and ammunition samples [23]. Using a blood lead level of 37.5 ng/ml ($3.75 \mu\text{g/dl}$) to distinguish background from elevated levels, the $^{207}\text{Pb}:^{206}\text{Pb}$ ratio for lead in the blood differed between the pre-release birds and wild birds with a lead concentration $>37.5 \text{ ng/ml}$. Thus, after release the condors were exposed to a lead source that was different to the (background) source during captivity. The mean $^{207}\text{Pb}:^{206}\text{Pb}$ ratio for lead in still-born dairy calves fed to pre-release birds was $0.8346 \pm 0.0046 \text{ SD}$ ($n=7$), and similar to the value for background environmental lead in California, but was higher than the mean ratio for ammunition samples ($0.8136 \pm 0.0035 \text{ SD}$, $n=18$). Condors with high blood lead levels had ratios that approached those of the ammunition. To test whether feathers could reveal a history of lead exposure, a retriex feather was serially sampled along its 24 cm length – condor feathers grow $\sim 5 \text{ mm/day}$. The isotope signatures differed between the older (distal) parts of the rachis and vane and the proximal parts, the latter having a ratio that was similar to that for ammunition.

Use of isotope ratios has also linked human exposure to lead to an ammunition source [150]. Ratios for lead in the blood of people living among subsistence hunting communities were compared with those in lichens (potentially contaminated with atmospheric lead) and samples of ammunition used to hunt wild game. Ratios for ^{206}Pb : ^{204}Pb and ^{206}Pb : ^{207}Pb revealed that lichens and lead ammunition were distinct groupings, but there was some overlap between humans and ammunition. The study also attempted to distinguish the lead in game killed with shot (suggested from previous studies to be main source) from game killed with bullets, but the ratios from the two types of ammunition were indistinguishable.

5. Potential mitigation measures

5.1 Range management

Where lead shot can still be used legally, the contamination can be reduced by various methods of range management. These fall into two broad categories: i) leaving the lead where it is, but making it unreachable or immobilised and ii) removing lead pellets through reclamation procedures.

After 10 years of hunting at a wetland in Missouri, USA lead shot taken 5.1 cm deep in front of two shooting positions was more than 4 times as numerous in uncultivated as in cultivated soils [47]. It appeared that more pellets moved deeply into the soil than returned to the surface as a result of cultivation. Both disking and ploughing moved lead pellets deeper than 10 cm into the soil and out of reach of dabbling ducks, but ploughing was more effective than disking to redistribute pellets deeper than 20 cm to make them unavailable to swans [139]. In this case, one disadvantage of cultivation was that an important food source for some waterbirds, swamp timothy (*Crypsis schoenoides*), was reduced and took 2 years to recover. To minimise the risk of grouse on moorlands ingesting lead pellets, it was suggested that the heather in the shot-fall zones should be allowed to grow tall and dense [146]. This would reduce its nutritional attractiveness and discourage the birds from entering it for cover. It was also suggested that piles of crushed grit, high in calcium e.g. crushed oystershells) should be provided, as the absorption of dietary lead is reduced when dietary calcium intake is high.

High CaCO_3 , Fe, Al and P contents were favourable for immobilising lead in shooting ranges [20] and various forms of phosphorus have been added to soils in an attempt to achieve this. Phosphorus can convert lead to insoluble, and biologically unavailable, forms of pyromorphite. Tests found that the addition of 1% phosphorus to contaminated sediments reduced the bioavailability of lead to mallards, as measured by a reduced accumulation of the metal in the blood, liver and kidney [58]. Adding lime to the soil can change the pH to a range within which lead cannot be dissolved (6.5-8.5). Encouraging vegetative ground cover and applying mulches and compost can provide natural complexing agents to bind lead and decrease its mobility. For example, pine trees (*Pinus sylvestris*) were able to bind 30% of soil lead with their roots [130]. This was considered to be a relatively cheap, but time-consuming, option, especially where the annual growth period is short. Other phytostabilisation

techniques have included testing the ability of fungi to accumulate lead and other metals.

In Finland, the most common remediation method has been soil excavation and disposal often complemented by isolation measures [130]. The estimated cost in 2006 to decontaminate 50-60 shooting ranges that were located up to 100 m from residential areas and thus presented potentially a 'significant' risk to human health was €250 million, compared with an annual expenditure to decontaminate 400 ranges of €50-70 million. Shooting ranges are issued with 10-year permits during which time environmental risks are determined and risk management actions have to be implemented. The authorities have insisted on bullet collection and recycling systems and this has encouraged the use of rubber granule traps and other shock absorbing material. Lead may be physically separated from soil by hydrodynamic, density or gravity separation. Shot can also be removed by vacuuming, but this is not feasible in wooded marsh or densely vegetated areas and hand-raking and sifting is considered too time-consuming. Removal of lead pellets from soils through reclamation procedures, such as gravity separation, was considered impractical for large areas [110]. Importantly, the effectiveness of these methods to reduce the contamination from lead ammunition has not been thoroughly tested. In the USA, a manual for the construction and environmental management of small arms ranges has been published [65]. This includes recommendations to control the dispersion of lead shot by, for example, realigning the shooting stands and the angles and trajectories of clay pigeons to concentrate spent pellets into a smaller area and the use of shot curtains to help recover and recycle lead.

5.2 Alternatives to lead in ammunition

5.2.1 Steel

As an alternative to lead shot, steel shot has been available for many years, but the ballistic properties of lead and steel differ, with steel pellets approximately 30% lighter than lead pellets of the same diameter and significantly harder [123]. As the terminal energy in a steel pellet is less than that of a lead pellet, it was argued that the crippling rate (birds wounded rather than killed) would increase. This was avoided by hunters using steel shot two sizes larger than the lead loads they were used to. Other changes in the specifications of steel shot cartridges to avoid increased chamber pressures and excessive barrel wear have largely overcome objections to the use of steel shot. However, steel pellets can generate aesthetic problems due to corrosion leading to excessive rust formation and there may also be a potential risk of excessive chromium load on the environment from steel pellets – apparently the chromium content can be 27% [130]. Concerns have also been expressed about the increased risk of ricochet off hard surfaces and that the timber value of trees with embedded pellets is reduced. It is argued that hunters need to adjust their hunting practice and technique and recognise that steel shot will never perform in exactly the same way as lead [11].

5.2.2 Tungsten

Tungsten has been used as a lead-replacement constituent in ammunition. At 19.3 g/cm³, it is denser than lead (11.36 g/cm³) [95]. Some of the environmental effects of tungsten and other tungsten alloying components present in munitions on soil biota and plants have been assessed [135]. Dissolution of tungsten powder significantly acidified soils and when the powder was mixed with soils at rates higher than 1% by weight, the bacterial component was reduced, the fungal biomass increased and worms (*Eisemia foetida*) and ryegrass died. Tungsten in spoils at an abandoned mine in Cumbria bioaccumulated in the common heather (*Calluna vulgaris*) [160]. However, a recent review concluded that, even with heavy pellet densities (>69,000/ha) and with each pellet containing up to 96% tungsten, the soil concentration would fall far below 1% and therefore, in practice, there would be little environmental risk [145]. The United States Fish & Wildlife Service has recently approved another tungsten alloy shot for duck shooting [153].

Groups of mallards were dosed with 8 No 4 pellets consisting of steel, lead or tungsten/nickel/iron [17]. Although the tungsten composite and steel pellets were eroded in the birds' gizzards, no mortality was observed during 30 days post dosing and there were no clinical signs of toxicity from exposure to the different metals and no lesions in the kidney or liver; in contrast 9/10 birds dosed with lead pellets died, the first death occurring 6 days post dosing. In another study, groups of mallards were dosed with 8 No 4 steel or lead pellets, or 8 BB-size tungsten-iron or tungsten-polymer pellets [70]. After 30 days all pellets were eroded with the tungsten-polymer pellets losing 80% of their weight. Although tungsten was detected in the femur, liver and kidney of ducks, no birds dosed with tungsten-iron, tungsten-polymer or steel died or showed clinical signs of poisoning.

5.2.3 Bismuth

Bismuth (Bi) is another alternative to lead but requires the addition of a small amount of tin to reduce its brittleness [123]. Its performance is comparable to that of lead in shotgun ammunition. The toxicity of 5 bismuth pellets embedded in the peritoneal cavity of laboratory mice was investigated in order to replicate the effects on wild animals wounded by hunting and surviving for long periods [103]. Each pellet (shot size 12) contained 91% bismuth and 9% tin. Traces of bismuth were found in the renal tubules and in the nervous system after 4 and 9 weeks, but no ill effects were observed. Five No 4 pellets of steel or bismuth/tin were embedded into the breast muscles of game-farm mallards their condition was monitored for 1 year after dosing [117]. There was a minimal inflammatory response in the muscles of the Bi-dosed ducks, but slightly more fibrosis and inflammation around the steel pellets because of 'rust' formation on each pellet and tissue reaction to the oxidised metal. There was a slight increase in the concentration of Bi in the kidneys and liver. Game-farm mallards were each orally dosed with 12-17 pellets (equivalent in mass to 5 No 4 lead pellets) composed of 39% tungsten, 44.5% bismuth and 16.5% tin (by weight) and monitored their condition for 32 days [109]. Despite undergoing extensive erosion inside the gizzard, the pellets caused no mortality, physiological abnormalities, changes in food consumption or loss of body weight.

5.2.4 Copper

Copper (Cu) is sometimes present in shotgun pellets as a trace element [56] and is commonly used to coat lead bullets to reduce barrel fouling. The fragmentation of rifle bullets on striking hunted animals and the recent evidence on the consequent risk of lead poisoning in hunters or scavengers has initiated a search for non-fragmenting materials. An all-copper bullet has been developed, which still expands in the animal to give an efficient kill, but appears not to break up on impact [95]. This reduces the potential pollution risk from spent ammunition. Copper is a relatively abundant element, moderately soluble and in contrast to lead is an essential micronutrient, but at elevated levels it can be toxic [43]. In mammals it is generally non-toxic, because there is an efficient homeostatic mechanism. This was demonstrated in one study in which large-scale increases in copper ingestion were observed in field voles (*Micotus agrestis*) and wood mice (*Apodemus sylvaticus*) living close to a copper refinery, but the total body concentrations remained constant [63].

In a test to determine the acute toxicity of various substitute materials for lead shot, 24 game-farm mallards were intubated with 8 No. 6-size copper pellets and were observed for 60 days post dosing [64]. One duck died 41 days after dosing and had lost 27% of its initial body weight; the surviving ducks had lost, on average, 12% of their initial body weight. However, the death was not attributed to copper shot as the kidney tubules did not show haemoglobin casts that indicate copper poisoning in animals. The majority of the ducks had retained most or all of the shot by the end of the trial.

In contrast to its effect on mammals, copper can be exceedingly toxic to aquatic biota including fish [43]. The mobility of metals between trophic levels may depend on whether, for different animal groups, the metals are essential or non-essential. Thus, copper is regulated and lead is accumulated, but in animals with haemocyanin in their oxygen-carrying system (e.g. isopods and molluscs) copper may accumulate (and be toxic) while lead does not [76].

5.3 Extending the lead shot ban to include all lead ammunition.

When lead poisoning incidents occur, it is reasonable to consider, initially, that the problem is relatively minor and localised and that the least restrictive sort of regulation will manage it [123]. This approach led to a ban on the use of lead shot for waterfowl hunting firstly in the USA and later in many other countries including the UK. As research continued, additional sites of concern (e.g. shooting ranges) were found and other problems, not originally foreseen, were uncovered, such as the secondary poisoning of raptors and the transboundary export/import of lead poisoning through embedded or ingested shot [123].

Where waterfowl migrate across country boundaries, any reduction in lead shot use should involve all nations along the flyways. The Maastricht Treaty of 1992 provided the policy and legislative framework for the EU to act, but the issue of subsidiarity meant that individual countries would have to implement their own regulations [144]. By 2007, the use of lead shot for hunting waterfowl had been regulated in 14 countries in Europe [84], all except one being in the EU. Far fewer countries have regulated the use of lead shot in other forms of recreational shooting.

In 1997, it was argued that the ban should be widened on the basis that the rate of shot deposition (in the USA) from upland hunting greatly exceeded that from waterfowl hunting and that the risk of multispecies poisoning had been demonstrated [141]. The lack of action by governments in North America was attributed to differences in the responsibilities between federal and state agencies, an overt emphasis by government agencies on the burden of scientific proof for every situation and opposition from hunters and international shooting organisations. It was suggested that lack of scientific certainty should give way to The Precautionary Principle (Rio Declaration on Environment and Development, 1992) to prevent the serious or irreversible environmental damage, predicted by many, from occurring [141]. This Principle appears to have been adopted by the Hokkaido Government when, in 2000, it banned the use of lead bullets to hunt deer as Steller's sea eagles and white-tailed eagles were thought to be at risk of extinction [88].

The enforcement of local shot bans is difficult if lead shot is still legally available for use in other types of shooting. Under the shooting conditions that exist in North America, compliance with a ban appears to be high as conservation officers can easily check what ammunition a shooter is using, but where hunting occurs on privately owned lands, as in England, compliance may be difficult to enforce [143]. Attempting to reduce pollution without having replacement products available will be met with major non-compliance, but ammunition manufacturers have now produced a range of so-called 'non-toxic' substitutes for use in shotguns; non-lead bullets are available for some calibres, but not yet for all rifles and pistols used in the various shooting disciplines.. Increased uptake of alternatives by shooters for whatever reason is likely to lower the cost of alternative products by increasing the economy of scale [143].

6. Conclusions

The problem of poisoning from lead ammunition has existed for over 100 years and has been reported for about the same length of time, although more frequently in the last 40 years. Most research has been conducted in North America where game-shooting and wildfowling are often carried out on public grounds. There has been relatively little research in the UK where shooting is carried out on private land and compliance with the lead shot ban for wildfowling has apparently been poor (in 2002, 40/58 (69%) mallards had shot-in lead pellets [29]). It is not known for certain if all waterfowl and game-birds are similarly susceptible to lead poisoning from ingested pellets, as dose-response relationships have, understandably, been determined for only a few species (e.g. mallards, black ducks, Canada geese and ptarmigan) and then only partially. Thus, the distinction between background lead levels and abnormal lead levels is not well-defined, particularly as early analytical methods were thought to be flawed [23]. Nevertheless, it seems reasonable to assume that most birds are susceptible, although any similarities in response may be overridden by local factors (diet, retention time, type of grit) in determining how many birds will show symptoms of poisoning.

Even where local factors present a high risk of poisoning, for many hunters, the perception is that, as very few dead birds are found, the problem must be negligible. For investigators, the lack of bodies makes it difficult to estimate the scale of the

problem. Large numbers of birds that die in a short period of time have been reported [12], but this rarely happens and if the population numbers appear unchanged the following year then the impact would seem to have been minimal. Indeed, the effect on populations of quarry species is likely to be confounded by captive-reared birds that are released to supplement wild populations. In the USA, before the lead-shot bans, it was estimated that for all waterfowl in North America the annual loss due to lead poisoning was 2-3% [12]. This compares with a natural mortality rate of 22.2% in a population of around 90 million [118]. If the natural loss of up to 20 million waterfowl goes largely unnoticed then the loss of up to 4 million birds from lead poisoning will be easily overlooked. Instead of large-scale sudden die-offs, deaths occur as day-to-day losses and predators and scavengers are able to remove the bodies of sick and dead birds rapidly [102] [161].

Reports from the USA suggest that the nationwide lead-shot ban had, after 5 years and a high rate of compliance, reduced the prevalence of lead poisoning in waterfowl. If, after an additional 13 years, up to 4 million waterfowl have been spared by the switch to non-toxic shot, then there is a case for extending the ban to include terrestrial game-birds, especially on animal welfare grounds. The effects of poisoning cause distress and severe pain that can last for days or months and, in Europe, affects an estimated 10,000-100,000, mostly birds, annually [115]. However, wounding by a bullet or shot also causes distress and severe pain lasting from minutes to days, but affects 200,000-2,000,000 birds and mammals annually (perhaps around 2% of all hunted animals). Arguably, improving the training and competence of shooters to reduce crippling rates might improve animal welfare more than using non-toxic shot.

It is also doubtful that reducing lead poisoning will necessarily decrease mortality rates in game species, particularly if poisoning is a form of compensatory mortality rather than additive mortality. Under the additive hypothesis, hunting and natural mortalities operate to increase total mortality, but under the compensatory hypothesis, reduced rates of natural mortality can compensate for hunting losses at low to intermediate levels of hunting [2]; total mortality only increases when hunting losses pass a 'compensation point'. Compensatory mortality may result in higher rates of natural mortality if hunting is reduced so that total mortality remains unchanged. In one study, the investigator considered if a loss of 2.3% due to lead poisoning in a mallard population would act in a totally additive fashion while losses of 15% due to hunting would be compensatory [148]. If compensatory, then actual losses due to lead poisoning would be substantially less. Thus the case for reducing lead on conservation grounds is unproven.

The case for protecting raptors from lead poisoning for conservation reasons is more compelling. From the limited data available, the evidence does not seem to suggest that raptors are more susceptible to lead poisoning *per se* than other groups of birds, but their populations are naturally relatively small and more sensitive to increases in adult mortality from any cause. Their prey may include, as well as waterfowl, animals that can still be shot legally with lead ammunition. The risk to scavengers could be reduced by changing hunters' practices i.e. retrieving all game and non-game (e.g. pests) animals that have been killed with lead ammunition and not leaving behind unwanted body parts (e.g. gut piles). Whether that can be achieved by voluntary codes of practice or by legislation is questionable – in the UK, the latter raises issues of compliance and enforcement across large areas of private land.

However, animals that are wounded and die later or recover may carry embedded bullet/pellet fragments, so the risk is not entirely eliminated. Although tests on American kestrels suggested that they might be less likely to be poisoned by lead shot, it is not known, for example, if kestrels in the UK would respond in a similar fashion. As bald eagles seemed to be more prone to lead poisoning, it is possible that birds not too distantly related (e.g. golden eagles) in the UK might be equally susceptible.

The effect of spent lead ammunition on birds and mammals that are not quarry species is largely unknown. The few studies that have been carried out suggest that passerines also ingest spent shot, but small mammals are unlikely to do so. For herbivores and seed-eaters, any uptake of lead is likely to be from contaminated food plants (absorbed or adsorbed lead) or, for shrews, from earthworms. Population losses are likely to be localised to those individuals whose home ranges overlap shot-fall zones and will be difficult to detect if poisoning is chronic in nature rather than acute.

Human health risk assessment of contaminated meat is complex since there are no identified thresholds for certain effects of lead on health, such as chronic kidney disease in adults and intelligence quotient (IQ) deficits in children [93] [152]. This probably means that it would be very difficult to determine, by experiment, what a safe level of lead might be, but an alternative approach to assessing the risk is to derive a margin of exposure (MOE), which relates a dose that causes a low but measurable response to the estimated human intake of the substance [9]. The risk management approach to lead is to reduce exposure to the lowest level possible. An added complication is that different studies that have examined lead contamination in game do not consistently show the same level of exposure. This appears to be caused, in part, by the nature of the contamination: an uneven scattering of lead fragments in a carcass can give samples that show, by chance, a very low or very high risk. It is likely that exposure to lead will tend to be lower from biologically-incorporated lead, because if lead levels are very high, as in a bird with chronic lead poisoning, this will render game unattractive to eat. (In retrospect, past studies of lead poisoning in waterfowl and game-birds invariably measured lead levels in hunter-killed birds and what was assumed to be biologically-incorporated lead (lead dissolved in the gizzard and absorbed into the blood stream) might have been, in some instances, embedded lead. The procedures used by most investigators specifically exclude tissue samples with obvious gunshot damage, but results from more recent studies have suggested that some embedded lead particles might have been so small and presumably caused so little tissue damage that they could have been easily missed.)

Some mitigation measures, other than banning lead ammunition, have been suggested for certain situations, such as lead recovery procedures or immobilising the lead using various techniques. There is little or no information concerning the effectiveness of these methods in reducing the incidence of lead poisoning in wildlife in either the short or long term. If they were shown to be cost-effective and there was a desire to reduce the lead deposited in these areas, then continuing to use lead ammunition on target ranges whilst reducing or removing any risks of lead contamination/poisoning, might be possible.

7. References

1. Adrian, W. J. & Stevens, M. L. (1979). Wet versus dry weights for heavy metal toxicity determinations in duck liver. *Journal of Wildlife Diseases* **15**: 125-126.
2. Allen, M. S., Miranda, L. E., & Brock, R. E. (1998). Implications of compensatory and additive mortality to the management of selected sportfish populations. *Lakes & Reservoirs: Research and Management* **3**: 67-79.
3. Anderson, W. L. & Havera, S. P. (1985). Blood lead, protoporphyrin, and ingested shot for detecting lead poisoning in waterfowl. *Wildlife Society Bulletin* **13**: 26-31.
4. Anderson, W. L., Havera, S. P., & Montgomery, R. A. (1987). Incidence of ingested shot in waterfowl in the Mississippi Flyway, 1977-1979. *Wildlife Society Bulletin* **15**: 181-188.
5. Anderson, W. L., Havera, S. P., & Zercher, B. W. (2000). Ingestion of lead and nontoxic shotgun pellets by ducks in the Mississippi Flyway. *Journal of Wildlife Management* **64**: 848-857.
6. Astrup, T., Boddum, J. K., & Christensen, T. H. (1999). Lead distribution and mobility in a soil embankment used as a bullet stop at a shooting range. *Journal of Soil Contamination* **8**: 653-665.
7. Avery, D. & Watson, R. T. (2009) Distribution of venison to humanitarian organizations in the USA and Canada. In RT Watson, M Fuller, M Pokras & WG Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080\ilsa.2009.0114.
8. Bagley, G. E. & Locke, L. N. (1967). The occurrence of lead in tissues of wild birds. *Bulletin of Environmental Contamination and Toxicology* **2**: 297-305.
9. Barlow, S., Renwick, A. G., Kleiner, J., Bridges, J. W., Busk, L., Dybing, E., Edler, L., Eisenbrand, G., Fink-Gremmels, J., Knaap, A., Kroes, R., Liem, D., Müller, D. J. G., Page, S., Rolland, V., Scletter, J., Tritscher, A., Tueting, W., & Würtzen, G. (2006). Risk assessment of substances that are both genotoxic and carcinogenic: report of an international conference organized by EFSA and WHO with support from ILSI Europe. *Food and Chemical Toxicology* **44**: 1636-1650.
10. Barltrop, D. & Meek, F. (1979). Effect of particle size on lead absorption from the gut. *Archives of Environmental Health* **34**: 280-285.
11. Beintema, N. (2002) Steel shot - some technical and safety aspects. <http://www.unep-aewa.org>.
12. Bellrose, F. C. (1959). Lead poisoning as a mortality factor in waterfowl populations. *Illinois Natural History Survey Bulletin* **27**: 235-288.

13. Bennett, J. R., Kaufman, C. A., Koch, I., Sova, J., & Reimer, K. J. (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**: 91-101.
14. Beyer, W. N., Franson, J. C., Locke, L. N., Stroud, R. K., & Sileo, L. (1998). Retrospective study of the diagnostic criteria in a lead-poisoning survey of waterfowl. *Archives of Environmental Contamination and Toxicology* **35**: 506-512.
15. Bjerregaard, P., Johansen, P., Mulvad, G., Pedersen, H. S., & Hansen, J. C. (2004). Lead sources in human diet in Greenland. *Environmental Health Perspectives* **112**: 1496-1498.
16. Blus, L. J., Henny, C. J., Hoffman, D. J., & Grove, R. A. (1995). Accumulation in and effects of lead and cadmium on waterfowl and passerines in northern Idaho. *Environmental Pollution* **89**: 311-318.
17. Brewer, L., Fairbrother, A., Clark, J., & Amick, D. (2003). Acute toxicity of lead, steel, and an iron-tungsten-nickel shot to mallard ducks (*Anas platyrhynchos*). *Journal of Wildlife Diseases* **39**: 638-648.
18. Butler, D. A. (2005). Incidence of lead shot ingestion in red-legged partridges (*Alectoris rufa*) in Great Britain. *Veterinary Record* **157**: 661-662.
19. Butler, D. A., Sage, R. B., Draycott, R. A. H., Carroll, J. P., & Potts, D. (2005). Lead exposure in ring-necked pheasants on shooting estates in Great Britain. *Wildlife Society Bulletin* **33**: 583-589.
20. Cao, X., Ma, L. Q., Chen, M., Hardison Jr, D. W., & Harris, W. G. (2003). Weathering of lead bullets and their environmental effects at outdoor shooting ranges. *Journal of Environmental Quality* **32**: 526-534.
21. Carrington, C. D., Sheehan, D. M., & Bolger, P. M. (1993). Hazard assessment of lead. *Food Additives and Contaminants* **10**: 325-335.
22. Chasko, G. G., Hoehn, T. R., & Howell-Heller, P. (1984). Toxicity of lead shot to wild black ducks and mallards fed natural foods. *Bulletin of Environmental Contamination and Toxicology* **32**: 417-428.
23. Church, M. E., Gwiazda, R., Risebrough, R. W., Sorenson, K., Chamberlain, C. P., Farry, S., Heinrich, W., Rideout, B. A., & Smith, D. R. (2006). Ammunition is the principal source of lead accumulated by California condors re-introduced to the wild. *Environmental Science & Technology* **40**: 6143-6150.
24. Coburn, D. R., Metzler, D. W., & Treichler, R. (1951). A study of absorption and retention of lead in wild waterfowl in relation to clinical evidence of lead poisoning. *Journal of Wildlife Management* **15**: 186-192.
25. Coburn, H. L., Snary, E. L., Kelly, L. A., & Wooldridge, M. (2005). Qualitative risk assessment of the hazards and risks from wild game. *Veterinary Record* **157**: 321-322.

26. Cook, R. S. & Trainer, D. O. (1966). Experimental poisoning of Canada geese. *Journal of Wildlife Management* **30**: 1-8.
27. Craig, J. R., Rimstidt, J. D., Bonnaffon, C. A., Collins, T. K., & Scanlon, P. F. (1999). Surface water transport of lead at a shooting range. *Bulletin of Environmental Contamination and Toxicology* **63**: 312-319.
28. Craighead, D. & Bedrosian, B. (2008). Blood levels of Common ravens with access to big-game offal. *Journal of Wildlife Management* **72**: 240-245.
29. Cromie, R. L., Brown, M. J., Hughes, B., Hoccum, D. G., & Williams, G. (2002). Prevalence of shot-in pellets in mallard purchased from game dealers in England in winter 2001/2002. In: *RSPB. 2002. Compliance with the Lead Shot Regulations (England) during winter 2001/02*. RSPB, Sandy, UK.
30. Custer, T. W., Franson, J. C., & Pattee, O. H. (1984). Tissue lead distribution and hematologic effects in American kestrels (*Falco sparverius* L.) fed biologically incorporated lead. *Journal of Wildlife Diseases* **20**: 39-43.
31. Danell, K., Andersson, Å., & Marström, V. (1977). Lead shot pellets dispersed by hunters: ingested by ducks. *Ambio* **6**: 235-237.
32. Darling, C. T. R. & Thomas, V. G. (2003). The distribution of outdoor shooting ranges in Ontario and the potential for lead pollution of soil and water. *Science of the Total Environment* **313**: 235-243.
33. Darling, C. T. R. & Thomas, V. G. (2005). Lead bioaccumulation in earthworms, *Lumbricus terrestris*, from exposure to lead compounds of differing solubility. *Science of the Total Environment* **346**: 70-80.
34. Delves, H. T. & Campbell, M. J. (1993). Identification and apportionment of sources of lead in human tissue. *Environmental Geochemistry and Health* **15**: 75-84.
35. Dermatas, D., Menouno, N., Dutko, P., Dadachov, M., Arienti, P., & Tsaneva, V. (2004). Lead and copper contamination in small arms firing ranges. *Global Nest: the International Journal* **6**: 141-148.
36. Dobrowolska, A. & Melosik, M. (2008). Bullet-derived lead in tissues of the wild boar (*Sus scrofa*) and red deer (*Cervus elaphus*). *European Journal of Wildlife Research* **54**: 231-235.
37. Duke, G. E., Jegers, A. A., Loff, G., & Evanson, O. A. (1975). Gastric digestion in some raptors. *Comparative Biochemistry & Physiology* **50A**: 649-656.
38. Eisler, R. (1988). Lead hazards to fish, wildlife, and invertebrates: a synoptic review. *US Fish & Wildlife Service Biological Reports* 85(1.14): 1-134.
39. Ferrandis, P., Mateo, R., López-Serrano, F. R., Martinez-Haro, M., & Martínez-Duro, E. (2008). Lead-shot exposure in Red-legged partridge (*Alectoris rufa*) on a driven shooting estate. *Environmental Science & Technology* **42**: 6271-6277.

40. Figuerola, J., Mateo, R., Green, A. J., Mondain-Monval, J. Y., Lefranc, H., & Mentaberre, G. (2005). Grit selection in waterfowl and how it determines exposure to ingested lead shot in Mediterranean wetlands. *Environmental Conservation* **32**: 226-234.
41. Finley, M. T. & Dieter, M. P. (1978). Erythrocyte δ -aminolevulinic acid dehydratase activity in mallard ducks: duration of inhibition after lead shot dosage. *Journal of Wildlife Management* **42**: 621-625.
42. Fisher, I. J., Pain, D. J., & Thomas, V. G. (2006). A review of lead poisoning from ammunition sources in terrestrial birds. *Biological Conservation* **131**: 421-432.
43. Flemming, C. A. & Trevors, J. T. (1989). Copper toxicity and chemistry in the environment: a review. *Water, Air, and Soil Pollution* **44**: 143-158.
44. Frank, A. (1986). Lead fragments in tissues from wild birds: a cause of misleading analytical results. *Science of the Total Environment* **54**: 275-281.
45. Franson, J. C., Sileo, L., Pattee, O. H., & Moore, J. F. (1983). Effects of chronic dietary lead in American kestrels (*Falco sparverius*). *Journal of Wildlife Diseases* **19**: 110-113.
46. Frape, D. L. & Pringle, J. D. (1984). Toxic manifestations in a dairy herd consuming haylage contaminated by lead. *Veterinary Record* **114**: 615-616.
47. Fredrickson, L. H., Baskett, T. S., Brakhage, G. K., & Cravens, V. C. (1977). Evaluating cultivation near duck blinds to reduce lead poisoning hazard. *Journal of Wildlife Management* **41**: 624-631.
48. Furness, J. C. & Robel, R. J. (1987). Estimated effectiveness of a waterfowl nontoxic shot zone. *Transactions of the Kansas Academy of Science* **90**: 143-146.
49. Giofriddo, J. P. & Best, L. B. (1996). Grit use patterns in North American birds: the influence of diet, body size, and gender. *Wilson Bulletin* **108**: 685-696.
50. Gjerstad, K. O. & Hanssen, I. (1984). Experimental lead poisoning in Willow ptarmigan. *Journal of Wildlife Management* **48**: 1018-1022.
51. Green, R. E., Hunt, W. G., Parish, C. N., & Newton, I. (2009). Effectiveness of action to reduce exposure of free-ranging California condors in Arizona and Utah to lead from spent ammunition. In RT Watson, M Fuller, M Pokras & WG Hunt (Eds.). *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080\ilsa.2009.0218.
52. Guitart, R., Serratos, J., & Thomas, V. G. (2002). Lead-poisoned wildfowl in Spain: a significant threat for human consumers. *International Journal of Environmental Health Research* **12**: 301-309.

53. Gustavsson, P. & Gerhardsson, L. (2005). Intoxication from an accidentally ingested lead shot retained in the gastrointestinal tract. *Environmental Health Perspectives* **113**: 491-493.
54. Haas, G. H. (1977). Unretrieved shooting loss of Mourning doves in north-central South Carolina. *Wildlife Society Bulletin* **5**: 123-125.
55. Haldimann, M., Baumgartner, A., & Zimmerli, B. (2002). Intake of lead from game meat - a risk to consumers' health. *European Food Research & Technology* **215**: 375-379.
56. Hall, S. L. & Fisher Jr, F. M. (1985). Heavy metal concentrations of duck tissues in relation to ingestion of spent shot. *Bulletin of Environmental Contamination and Toxicology* **35**: 163-172.
57. Havera, S. P., Whitton, R. M., & Shealy, R. T. (1992). Blood lead and ingested and embedded shot in diving ducks during spring. *Journal of Wildlife Management* **56**: 539-545.
58. Heinz, G. H., Hoffman, D. J., & Audet, D. J. (2004). Phosphorus amendment reduces bioavailability of lead to mallards ingesting contaminated sediments. *Archives of Environmental Contamination and Toxicology* **46**: 534-541.
59. Hoffman, D. J., Pattee, O. H., Wiemeyer, S. N., & Mulhern, B. (1981). Effects of lead shot ingestion on δ -aminolevulinic acid dehydratase activity, hemoglobin concentration, and serum chemistry in Bald eagles. *Journal of Wildlife Diseases* **17**: 423-431.
60. Holdner, J., Wainman, B., Jayasinghe, R., van Spronsen, E., Karagatzides, J. D., Nieboer, E., & Tsuji, L. J. S. (2004). Soil and plant lead of upland habitat used extensively for recreational shooting and game bird hunting in southern Ontario, Canada. *Bulletin of Environmental Contamination and Toxicology* **73**: 568-574.
61. Hunt, W. G., Burnham, W., Parish, C. N., Burnham, K. K., Mutch, B., & Oaks, J. L. (2006). Bullet fragments in deer remains: implications for lead exposure in avian scavengers. *Wildlife Society Bulletin* **34**: 167-170.
62. Hunt, W. G., Watson, R. T., Oaks, J. L., Parish, C. N., Burnham, K. K., Tucker, R. L., Belthoff, J. R., & Hart, G. (2009). Lead bullet fragments in venison from rifle-killed deer: potential for human dietary exposure. In RT Watson, M Fuller, M Pokras & WG Hunt (Eds.). *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080\ilsa.2009.0112.
63. Hunter, B. A., Johnson, M. S., & Thompson, D. J. (1987). Ecotoxicology of copper and cadmium in a contaminated grassland ecosystem. III. Small mammals. *Journal of Applied Ecology* **24**: 601-614.
64. Irby, H. D., Locke, L. N., & Bagley, G. E. (1967). Relative toxicity of lead and selected substitute shot types to game farm mallards. *Journal of Wildlife Management* **31**: 253-257.

65. ITRC (Interstate Technology & Regulatory Council). (2005). *Environmental Management at Operating Outdoor Small Arms Firing Ranges. SMART-2*. Washington, D.C.: Interstate Technology & Regulatory Council, Small Arms Firing Range Team. Available on the Internet at <http://www.itrcweb.org>.
66. Johansen, P., Asmund, G., & Rigét, F. (2001). Lead contamination of seabirds harvested with lead shot - implications to human diet in Greenland. *Environmental Pollution* **112**: 501-504.
67. Johansen, P., Asmund, G., & Rigét, F. (2004). High human exposure to lead through consumption of birds hunted with lead shot. *Environmental Pollution* **127**: 125-129.
68. Johansen, P., Pedersen, H. S., Asmund, G., & Rigét, F. (2006). Lead shot from hunting as a source of lead in human blood. *Environmental Pollution* **142**: 93-97.
69. Jørgensen, S. S. & Willems, M. (1987). The fate of lead in soils: the transformation of lead pellets in shooting-range soils. *Ambio* **16**: 11-15.
70. Kelly, M. E., Fitzgerald, S. D., Aulerich, R. J., Balander, R. J., Powell, D. C., Stickle, R. L., Stevens, W., Cray, C., Tempelman, R. J., & Bursian, S. J. (1998). Acute effects of lead, steel, tungsten-iron and tungsten-polymer shot administered to game-farm mallards. *Journal of Wildlife Diseases* **34**: 673-687.
71. Kendall, R. J., Lacher Jr, T. E., Bunck, C., Daniel, B., Driver, C., Grue, C. E., Leighton, F., Stansley, W., Watanabe, P. G., & Whitworth, M. (1996). An ecological risk assessment of lead shot exposure in non-waterfowl avian species: upland game birds and raptors. *Environmental Toxicology and Chemistry* **15**: 4-20.
72. Knopper, L. D., Mineau, P., Scheuhammer, A. M., Bond, D. E., & McKinnon, D. T. (2006). Carcasses of shot Richardson's ground squirrels may pose lead hazards to scavenging hawks. *Journal of Wildlife Management* **70**: 295-299.
73. Kramer, J. L. & Redig, P. T. (1997). Sixteen years of lead poisoning in eagles, 1980-95: an epizootiologic view. *Journal of Raptor Research* **31**: 327-332.
74. Kreager, N., Wainman, B. C., Jayasinghe, R. K., & Tsuji, L. J. S. (2008). Lead pellet ingestion and liver-lead concentrations in upland game birds from southern Ontario, Canada. *Archives of Environmental Contamination and Toxicology* **54**: 331-336.
75. Krone, O., Kenntner, N., Trinogga, A., Nadiarzadeh, M., Scholz, F., Sulawa, J., Totscher, K., Schuck-Wersig, P., & Zieschank, R. (2009). Lead poisoning in White-tailed sea eagles: causes and approaches to solutions in Germany. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0207.

76. Laskowski, R. & Hopkin, S. P. (1987). Accumulation of Zn, Cu, Pb and Cd in the garden snail (*Helix aspersa*): implications for predators. *Environmental Pollution* **91**: 289-297.
77. Lewis, J. C. & Legler, E. (1968). Lead shot ingestion by Mourning doves and incidence in soil. *Journal of Wildlife Management* **32**: 476-482.
78. Lin, Z., Comet, B., Qvarfort, U., & Herbert, R. (1995). The chemical and mineralogical behaviour of Pb in shooting range soils from central Sweden. *Environmental Pollution* **89**: 303-309.
79. Lumeij, J. T. & Scholten, H. (1989). A comparison of two methods to establish the prevalence of lead shot ingestion in mallards (*Anas platyrhynchos*) from the Netherlands. *Journal of Wildlife Diseases* **25**: 297-299.
80. Ma, W. (1989). Effect of soil pollution with metallic lead pellets on lead bioaccumulation and organ/body weight alterations in small mammals. *Archives of Environmental Contamination and Toxicology* **18**: 617-622.
81. MacDonald, J. W., Randall, C. J., Ross, H. M., Moon, G. M., & Ruthven, A. D. (1983). Lead poisoning in captive birds of prey. *Veterinary Record* **113**: 65-66.
82. Madsen, H. H. T., Skjødt, T., Jørgensen, P. J., & Grandjean, P. (1988). Blood lead levels in patients with lead shot retained in the appendix. *Acta Radiologica* **29**: 745-746.
83. Manninen, S. & Tanskanen, N. (1993). Transfer of lead from shotgun pellets to humus and three plant species in a Finnish shooting range. *Archives of Environmental Contamination and Toxicology* **24**: 410-414.
84. Mateo, R. (2009). Lead poisoning in wild birds in Europe and the regulations adopted by different countries. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0107.
85. Mateo, R., Dolz, J. C., Serrano, J. M. A., Belliure, J., & Guitart, R. (1997). An epizootic of lead poisoning in Greater flamingos (*Phoenicopterus ruber roseus*) in Spain. *Journal of Wildlife Diseases* **33**: 131-134.
86. Mateo, R., Martinez-Vilalta, A., & Guitart, R. (1997). Lead shot pellets in the Ebro delta, Spain: densities in sediments and prevalence of exposure in waterfowl. *Environmental Pollution* **96**: 335-341.
87. Mateo, R., Rodríguez-de la Cruz, M., Videl, D., Reglero, M., & Camarero, P. (2006). Transfer of lead from shot pellets to game meat during cooking. *Science of the Total Environment* **372**: 480-485.
88. Matsuda, H. (2003). Challenges posed by the precautionary principle and accountability in ecological risk assessment. *Environmetrics* **14**: 245-254.

89. Mellor, A. & McCartney, C. (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**: 124-129.
90. Mörner, T. & Petersson, L. (1999). Lead poisoning in woodpeckers in Sweden. *Journal of Wildlife Diseases* **35**: 763-765.
91. Mudge, G. P. (1983). The incidence and significance of ingested lead pellet poisoning in British wildfowl. *Biological Conservation* **27**: 333-372.
92. Mudge, G. P. (1984). Densities and settlement rates of spent shotgun pellets in British wetland soils. *Environmental Pollution (Series B)* **8**: 299-318.
93. Needleman, H. L. & Bellinger, D. (1991). The health effects of low level exposure to lead. *Annual Review of Public Health* **12**: 111-140.
94. Nixon, C. M., Hansen, L. P., Brewer, P. A., Chelsvig, J. E., Esker, T. L., Etter, D., Sullivan, J. B., Koerkenmeier, R. G., & Mankin, P. C. (2001). Survival of white-tailed deer in intensively farmed areas of Illinois. *Canadian Journal of Zoology* **79**: 581-588.
95. Oltrogge, V. (2009). Success in developing lead-free, expanding-nose centerfire bullets. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0305.
96. Pain, D. J., Amiard-Triquet, C., & Sylvestre, C. (1992). Tissue lead concentrations and shot ingestion in nine species of waterbirds from the Camargue (France). *Ecotoxicology and Environmental Safety* **24**: 217-233.
97. Pain, D. J., Carter, I., Sainsbury, A. W., Shore, R. F., Eden, P., Taggart, M. A., Konstantinos, S., Walker, L. A., Meharg, A. A., & Raab, A. (2007). Lead contamination and associated disease in captive and reintroduced red kites *Milvus milvus* in England. *Science of the Total Environment* **376**: 116-127.
98. Pain, D. J. & Eon, L. (1993). Methods of investigating the presence of ingested lead shot in waterfowl gizzards: an improved technique. *Wildfowl* **44**: 184-187.
99. Pain, D. J., Fisher, I. J., & Thomas, V. G. (2009). A global update of lead poisoning in terrestrial birds from ammunition sources. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0108.
100. Pain, D. J. & Rattner, B. A. (1988). Mortality and hematology associated with the ingestion of one Number Four lead shot in Black ducks, *Anas rubripes*. *Bulletin of Environmental Contamination and Toxicology* **40**: 159-164.
101. Pain, D. J., Sears, J., & Newton, I. (1995). Lead concentrations in birds of prey in Britain. *Environmental Pollution* **87**: 173-180.

102. Pain, D. J. (1991). Why are lead-poisoned waterfowl rarely seen?: the disappearance of waterfowl carcasses in the Camargue, France. *Wildfowl* **42**: 118-122.
103. Pamphlett, R., Danscher, G., Rungby, J., & Stoltenberg, M. (2000). Tissue uptake of bismuth from shotgun pellets. *Environmental Research* **82**: 258-262.
104. Parslow, J. F. L., Thomas, G. J., & Williams, T. D. (1982). Heavy metals in the livers of waterfowl from the Ouse Washes, England. *Environmental Pollution (Series A)* **29**: 317-327.
105. Pattee, O. H., Wiemeyer, S. N., Mulhern, B. M., Sileo, L., & Carpenter, J. W. (1981). Experimental lead-shot poisoning in Bald eagles. *Journal of Wildlife Management* **45**: 806-810.
106. Pauli, J. N. & Buskirk, S. W. (2007). Recreational shooting of prairie dogs: a portal for lead entering wildlife food chains. *Journal of Wildlife Management* **71**: 103-108.
107. Potts, G. R. (2005). Incidence of ingested lead gunshot in wild grey partridges (*Perdix perdix*) from the UK. *European Journal of Wildlife Research* **51**: 31-34.
108. Rice, D. A., McLoughlin, M. F., Blanchflower, W. J., & Thompson, T. R. (1987). Chronic lead poisoning in steers eating silage contaminated with lead shot - diagnostic criteria. *Bulletin of Environmental Contamination and Toxicology* **39**: 622-629.
109. Ringelman, J. K., Miller, M. W., & Andelt, W. F. (1993). Effects of ingested tungsten-bismuth-tin shot on captive mallards. *Journal of Wildlife Management* **57**: 725-732.
110. Rocke, T. E., Brand, C. J., & Mensik, J. G. (1997). Site-specific lead exposure from lead pellet ingestion in sentinel mallards. *Journal of Wildlife Management* **61**: 228-234.
111. Rodrigue, J., McNicoll, R., Leclair, D., & Duchesne, J. F. (2005). Lead concentrations in ruffed grouse, rock ptarmigan, and willow ptarmigan in Quebec. *Archives of Environmental Contamination and Toxicology* **49**: 97-104.
112. Rooney, C. P., McLaren, R. G., & Cresswell, R. J. (1999). Distribution and phytoavailability of lead in a soil contaminated with lead shot. *Water, Air, and Soil Pollution* **116**: 535-548.
113. Roscoe, D. E., Nielsen, S. W., Lamola, A., & Zuckerman, D. (1979). Simple quantitative test for erythrocytic protoporphyrin in lead-poisoned ducks. *Journal of Wildlife Diseases* **15**: 127-136.
114. Roscoe, D. E., Widjeskog, L., & Stansley, W. (1989). Lead poisoning of northern pintail ducks feeding in a tidal meadow contaminated with shot from a trap and skeet range. *Bulletin of Environmental Contamination and Toxicology* **42**: 226-233.

115. Sainsbury, A. W., Bennett, P. M., & Kirkwood, J. K. (1995). The welfare of free-living wild animals in Europe: harm caused by human activities. *Animal Welfare* **4**: 183-206.
116. Samuel, M. D. & Bowers, E. F. (2000). Lead exposure in American black ducks after implementation of non-toxic shot. *Journal of Wildlife Management* **64**: 947-953.
117. Sanderson, G. C., Anderson, W. L., Foley, G. L., Havera, S. P., Skowron, L. M., Brawn, J. W., Taylor, G. D., & Seets, J. W. (1998). Effects of lead, iron, and bismuth shot embedded in the breast muscles of game-farm mallards. *Journal of Wildlife Diseases* **34**: 688-697.
118. Sanderson, G. C. & Bellrose, F. C. (1986). A review of the problem of lead poisoning in waterfowl Jamestown ND: Northern Prairie Wildlife Research Centre Online 4: 34pp.
119. Scheuhammer, A. M. (1989). Monitoring wild bird populations for lead exposure. *Journal of Wildlife Management* **53**: 759-765.
120. Scheuhammer, A. M. (2009). Historical perspective on the hazards of environmental lead from ammunition and fishing weights in Canada Ingestion of Lead from Spent Ammunition. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0105.
121. Scheuhammer, A. M., Bond, D. E., Burgess, N. M., & Rodrigue, J. (2003). Lead and stable lead isotope ratios in soil, earthworms, and bones of American woodcock (*Scolopax minor*) from Eastern Canada. *Environmental Toxicology and Chemistry* **22**: 2585-2591.
122. Scheuhammer, A. M. & Norris, S. L. (1995). A review of the environmental impacts of lead shotshell ammunition and lead fishing weights in Canada. Occasional paper (Canadian Wildlife Service) no. 88.
123. Scheuhammer, A. M. & Norris, S. L. (1996). The ecotoxicology of lead shot and lead fishing weights. *Ecotoxicology* **5**: 279-295.
124. Scheuhammer, A. M., Perrault, J. A., Routhier, E., Braune, B. M., & Campbell, G. D. (1998). Elevated lead concentrations in edible portions of game birds harvested with lead shot. *Environmental Pollution* **102**: 251-257.
125. Scheuhammer, A. M. & Templeton, D. M. (1998). Use of stable isotope ratios to distinguish sources of lead exposure in wild birds. *Ecotoxicology* **7**: 37-42.
126. Schultz, J. H., Millsbaugh, J. J., Washburn, B. E., Wester, G. R., Lanigan III, J. T., & Franson, J. C. (2002). Spent-shot availability and ingestion on areas managed for Mourning doves. *Wildlife Society Bulletin* **30**: 112-120.
127. Shlosberg, A., Bellaiche, M., Regev, S., Gal, R., Brizzi, M., Hanji, V., Zaldel, L., & Nyska, A. (1997). Lead toxicosis in a captive bottlenose dolphin (*Tursiops*

- truncatus*) consequent to ingestion of air gun pellets. *Journal of Wildlife Diseases* **33**: 135-139.
128. Sileo, L., Creekmore, L. H., Audet, D. J., Snyder, M. R., Meteyer, C. U., Franson, J. C., Locke, L. N., Smith, M. R., & Finley, D. L. (2001). Lead poisoning of waterfowl by contaminated sediment in the Coeur d'Alene River. *Archives of Environmental Contamination and Toxicology* **41**: 364-368.
 129. Sneddon, J., Clemente, R., Riby, P., & Lepp, N. W. (2009). Source-pathway-receptor investigation of the fate of trace elements derived from shotgun pellets discharged in terrestrial ecosystems managed for game shooting. *Environmental Pollution* **157**: 2663-2669.
 130. Sorvari, J., Antikainen, R., & Pyy, O. (2006). Environmental contamination at Finnish shooting ranges - the scope of the problem and management options. *Science of the Total Environment* **366**: 21-31.
 131. Southwood, T. R. E. (1983). Royal Commission on Environmental Pollution: 9th Report – Lead in the environment. HMSO, London.
 132. Stansley, W. & Roscoe, D. E. (1996). The uptake and effects of lead in small mammals and frogs at a trap and skeet range. *Archives of Environmental Contamination and Toxicology* **30**: 220-226.
 133. Stansley, W., Widjeskog, L., & Roscoe, D. E. (1992). Lead contamination and mobility in surface water at trap and skeet ranges. *Bulletin of Environmental Contamination and Toxicology* **49**: 640-647.
 134. Stendell, R. C. (1980). Dietary exposure of kestrels to lead. *Journal of Wildlife Management* **44**: 527-530.
 135. Strigul, N., Koutsospyros, A., Arienti, P., Christodoulatos, C., Dermatas, D., & Braida, W. (2005). Effects of tungsten on environmental systems. *Chemosphere* **61**: 248-258.
 136. Stroud, R. K. & Hunt, W. G. (2009). Gunshot wounds: a source of lead in the environment. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0109.
 137. Svanberg, F., Mateo, R., Hillstrom, L., Green, A. J., Taggart, M. A., Raab, A., & Meharg, A. A. (2006). Lead isotopes and lead shot ingestion in the globally threatened marbled teal (*Marmaronetta angustirostris*) and white-headed duck (*Oxyura leucocephala*). *Science of the Total Environment* **370**: 416-424.
 138. Szymezak, M. R. (1978). Steel shot use on a goose hunting area in Colorado. *Wildlife Society Bulletin* **6**: 217-225.
 139. Thomas, C. M., Mensik, J. G., & Feldheim, C. L. (2001). Effects of tillage on lead shot distribution in wetland sediments. *Journal of Wildlife Management* **65**: 40-46.

140. Thomas, G. J. (1975). Ingested lead pellets in waterfowl at the Ouse Washes, England, 1968-73. *Wildfowl* **26**: 43-48.
141. Thomas, V. G. (1997). The environmental and ethical implications of lead shot contamination of rural lands in North America. *Journal of Agricultural & Environmental Ethics* **10**: 41-54.
142. Thomas, V. G. (2009). The policy and legislative dimensions of nontoxic shot and bullet use in North America. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0311.
143. Thomas, V. G. & Guitart, R. (2003). Lead pollution from shooting and angling, and a common regulative approach. *Environmental Policy and Law* **33**: 143-149.
144. Thomas, V. G. & Owen, M. (1996). Preventing lead toxicosis of European waterfowl by regulatory and nonregulatory means. *Environmental Conservation* **23**: 358-364.
145. Thomas, V. G., Roberts, M. J., & Harrison, P. T. C. (2009). Assessment of the environmental toxicity and carcinogenicity of tungsten-based shot. *Ecotoxicology and Environmental Safety* **72**: 1031-1037.
146. Thomas, V. G., Scheuhammer, A. M., & Bond, D. E. (2009). Bone lead levels and lead isotope ratios in red grouse from Scottish and Yorkshire moors. *Science of the Total Environment* **407**: 3494-3502.
147. Tranel, M. A. & Kimmel, R. O. (2009). Impacts of lead ammunition on wildlife, the environment, and human health - a literature review and implications for Minnesota. In RT Watson, M Fuller, M Pokras, & WG Hunt (Eds.). *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans*. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0307.
148. Trost, R. E. (1980). Ingested shot in waterfowl harvested on the Upper Mississippi national wildlife refuge. *Wildlife Society Bulletin* **8**: 71-74.
149. Tsuji, L. J. S., Nieboer, E., Karagatzides, J. D., Hanning, R. M., & Katapatuk, B. (1999). Lead shot contamination in edible portions of game birds and its dietary implications. *Ecosystem Health* **5**: 183-192.
150. Tsuji, L. J. S., Wainman, B. C., Martin, I. D., Sutherland, C., Weber, J.-P., Dumas, P., & Nieboer, E. (2008). The identification of lead ammunition as a source of lead exposure in First Nations: the use of lead isotope ratios. *Science of the Total Environment* **393**: 291-298.
151. US Environmental Protection Agency. (1986). Air quality criteria for lead. EPA-600/8-83/028aF. Research Triangle Park, NC, EPA.

152. US Environmental Protection Agency. (2006). Air quality criteria for lead. EPA/600/R-5/144aF. Research Triangle Park, NC, EPA.
153. US Fish & Wildlife Service. (2009). Migratory bird hunting; approval of tungsten-iron-fluoropolymer shot alloys as non-toxic for hunting waterfowl and coots. *Federal Register* 74(201), 53665-53671.
154. Vantelon, D., Lanzirrotti, A., Scheinost, A. C., & Kretzschmar, R. (2005). Spatial distribution and speciation of lead around corroding bullets in a shooting range soil studied by micro-x-ray fluorescence and absorption spectroscopy. *Environmental Science & Technology* **39**: 4808-4815.
155. Vyas, N. B., Spann, J. W., Heinz, G. H., Beyer, W. N., Jaquette, J. A., & Mengelkoch, J. M. (2000). Lead poisoning of passerines at a trap and skeet range. *Environmental Pollution* **107**: 159-166.
156. Wayland, M. & Bollinger, T. (1999). Lead exposure and poisoning in bald eagles and golden eagles in the Canadian prairie provinces. *Environmental Pollution* **104**: 341-350.
157. White, D. H. & Stendell, R. C. (1977). Waterfowl exposure to lead and steel shot on selected hunting areas. *Journal of Wildlife Management* **41**: 469-475.
158. Whitehead, P. J. & Tschirner, K. (1991). Lead shot ingestion and lead-poisoning of magpie geese *Anseranas semipalmata* foraging in a northern Australian hunting reserve. *Biological Conservation* **58**: 99-118.
159. Wiemeyer, S. N., Scott, J. M., Anderson, M. P., Bloom, P. H., & Stafford, C. J. (1988). Environmental contaminants in California condors. *Journal of Wildlife Management* **52**: 238-247.
160. Wilson, B. & Pyatt, F. B. (2006). Bio-availability of tungsten in the vicinity of an abandoned mine in the English Lake District and some potential health implications. *Science of the Total Environment* **370**: 401-408.
161. Wobeser, G. & Wobeser, A. G. (1992). Carcass disappearance and estimation of mortality in a simulated die-off of small birds. *Journal of Wildlife Diseases* **28**: 548-554.
162. Zwank, P. J., Wright, V. L., Shealy, P. M., & Newsom, J. D. (1985). Lead toxicosis in waterfowl on two major wintering areas in Louisiana. *Wildlife Society Bulletin* **13**: 17-26.