Poisoning of birds and other wildlife from ammunition-derived lead in the UK

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ABSTRACT

Lead is toxic to animals and thousands of tonnes of lead ammunition, primarily gunshot, are deposited and accumulate in the UK environment annually. Lead derived from ammunition now appears to be the most significant geographically widespread and common source of unregulated environmental lead contamination in the UK to which wildlife is exposed. The effects of lead from ammunition have primarily been studied in birds, with the two main exposure pathways being direct ingestion of spent gunshot (*e.g.* by wildfowl and terrestrial gamebirds, that mistake it for grit or food), and ingestion by predators and scavengers of lead gunshot, bullets, or fragments from these, in the flesh of their prey. Numerous studies conducted in the UK and overseas over the last 65 years have shown that lead poisoning from ammunition sources is geographically widespread and causes substantial suffering and mortality in many avian taxa. While relatively few studies have focussed on non-avian taxa in the UK, this does not imply that risks do not exist.

Broad estimates indicate that in the UK in the order of 50,000-100,000 wildfowl (c. 1.5-3.0% of the wintering population) are likely to die each winter (*i.e.* during the shooting season) as a direct result of lead poisoning. For migratory swans, this represents a quarter of all recorded deaths. Wildfowl that die outside of the shooting season will be additional, as will those that die of causes exacerbated by lead poisoning. Several hundred thousand wildfowl a year may suffer welfare effects. Estimates of mortality for terrestrial gamebirds in the UK are less accurate and precise, but indicate that in the order of hundreds of thousands of birds may die from lead poisoning annually. Studies in North America show that lead poisoning kills a substantial proportion of certain species of predatory and scavenging birds, but equivalent studies have not yet been conducted in the UK. A few studies from the UK have reported lead poisoning in certain raptor species, and the source and pathways exist for a wider range of species to be affected.

Key words: *Lead, ammunition, wildlife, birds, poisoning, mortality, UK, welfare*

INTRODUCTION

Lead is a naturally occurring toxic metal that has been used by humans for centuries, and is consequently widely distributed in the environment. Increasing knowledge of the negative effects of even low levels of exposure to lead on human health has resulted in society taking many actions to reduce emissions of, and exposure to, lead such as its removal from petrol and paint. For example, atmospheric lead emissions were estimated to have declined by 98% between 1990 and 2011 in England largely due to the phasing out of the use of lead additives in petrol (Thistlethwaite *et al.* 2013).

Current legislative controls and monitoring of industrial, municipal and agricultural lead emissions in the UK are such that cases of clinical lead poisoning from these sources in wildlife are likely to be rare. High concentrations of lead derived from sources other than ammunition and fishing weights exist in some soils in urban areas and near centres of current and historical industrial activity, especially mining and smelting.

Some lead derived from anglers' lead weights used before restrictions on their sale and use were introduced in 1987 is present in some wetlands and rivers, but additional lead from this source has probably been added at a low rate since then through a small amount of permitted use, and any illegal use that may have occurred. The mute swan *Cygnus olor* is the species reported to have been significantly affected by the ingestion of anglers' lead weights in the UK (Birkhead 1982, Birkhead and Perrins 1986) probably because of their habit of frequenting urban rivers and lakes where fishing activity is high. The recorded decrease in the incidence of lead poisoning in mute swans (Sears and Hunt 1990) and corresponding increase in their populations following the 1987 restrictions (Kirby *et al.* 1994) suggests that restrictions were largely successful. Newth *et al.* (2012) similarly found that the proportion of deaths attributable to lead poisoning in a sample of mute swans decreased significantly over time after restrictions, *i.e.* from 25% between 1971 and 1987 (pre-restrictions) to 4.6% between 1988 and 1999 and 2% between 2000 and 2010.

Beyond these sources, lead derived from ammunition now appears to be the only significant, geographically widespread and common source of unregulated environmental lead contamination to which wildlife is exposed.

This paper aims to bring together a broad range of evidence to illustrate the pathways by which wildlife is exposed to ammunition-derived lead and review the extent and impact of the problem in the UK.

PHYSIOLOGICAL EFFECTS OF LEAD

Lead is a non-essential metal that has no biological benefit to living organisms and is toxic to all vertebrates. Lead is also toxic to invertebrates but sensitivities appear to vary considerably (Eisler 1988). It is an accumulative metabolic poison that is non-specific, affecting a wide range of physiological and biochemical systems. These include the haematopoietic, vascular, nervous, renal and reproductive systems (Eisler 1988, USATSDR 2007, EFSA 2010, Franson and Pain 2011). Lead occurs primarily in inorganic form in the environment and lead in ammunition is in its elemental metallic form. In this paper, the term "lead" refers to inorganic lead. Following absorption, the effects of lead upon an animal's body systems are independent of source.

The toxic effects of lead are broadly similar in all vertebrates. In wild animals these effects are well known from numerous experimental and field studies. These have been reviewed many times (*e.g.* Eisler 1988, Pattee and Pain 2003, Franson and Pain 2011, Ma 2011). Although the present paper deals with wildlife in general, we have focussed upon birds because they are by far the most significantly studied taxon and are significantly affected. Clinical signs of poisoning are often associated with chronic exposure to lead in birds. Chronic exposure is extended exposure at a level that is not necessarily likely to cause immediate failure of biological functioning or death, although death may eventually result. Signs include anaemia, lethargy, muscle wasting and loss of fat reserves, green diarrhoea staining the vent, wing droop, lack of balance and coordination and other neurological signs such as leg paralysis or convulsions (*e.g.* Locke and Thomas 1996, Wobeser 1997, Friend and Franson 1999, Eisler 2000, Pattee and Pain 2003). In cases where birds die rapidly following acute exposure to high levels of lead, many of these signs may be absent.

Numerous experiments have been conducted where captive birds from many taxa, including wildfowl and raptors, have been dosed with lead gunshot and blood lead concentrations and physiological responses reported relative to controls (*e.g.* Pattee *et al.* 2006, Hoffman *et al.* 1981, 1985, reviews in Eisler 1988, see also Pattee and Pain 2003, and Franson and Pain 2011). In some instances, lead ammunition or ammunition fragments are eliminated rapidly from a bird's alimentary canal with little lead absorption, but they are also often retained until completely eroded, with the lead becoming soluble salts and much of it being absorbed by the bird. The acidic conditions in birds' stomachs and the strong mechanically grinding action in the gizzards of certain bird species facilitate erosion and solubilisation of lead ammunition, and blood lead concentrations can rapidly become elevated after ingestion of gunshot (*e.g.* see Hoffman *et al.* 1981, 1985, Pain and Rattner 1988, Pattee *et al.* 2006). Absorbed lead is transported in the bloodstream and deposited rapidly into soft tissues, primarily the liver and kidney, into bone, and the growing feathers of birds. Lead in bone is retained for long periods and bone lead concentrations increase over an animal's lifetime, whereas lead in soft tissues has a much shorter half-life (often

weeks to months). Consequently, highest lead concentrations are generally found in bone, followed by kidney and liver, with intermediate concentrations in brain and blood, and lowest concentrations in muscle (Longcore *et al.* 1974a, Johnson *et al.* 1982, Custer *et al.* 1984, Garcia Fernandez *et al.* 1995). However, in cases of acute lead poisoning, concentrations in soft tissues may be very elevated relative to those in bone. Blood lead is a good indicator of recent exposure and usually remains elevated for weeks or months after exposure. The degree and duration of elevation of blood lead depends largely upon the amount absorbed and the duration of exposure. While lead in bone is less mobile than in other tissues, it can be mobilised under certain conditions. For example, lead may be mobilised from medullary bone together with calcium, when calcium is required for eggshell formation (Finley and Dieter 1978).

The first measurable biochemical effect of lead, occurring at very low blood lead levels, is inhibition of the activity of the blood enzyme delta-aminolevulinic acid dehydratase (δ-ALAD), necessary for haem synthesis in erythrocytes (Hernberg *et al.* 1970, Tola *et al.* 1973, Pain 1987, 1989, Martinez-Lopez *et al.* 2004). While some reduction in ALAD activity appears to be tolerated in birds, protracted inhibition in ALAD activity can be associated with haemolytic anaemia (Pain and Rattner 1988, Mateo *et al.* 2003). As in other animals, lead can affect a wide range of body systems influencing reproduction, productivity, behaviour and the immune system (for a selection of specific studies on a range of bird species see Longcore *et al.* 1974a, 1974b, Clemens *et al.* 1975, Finley *et al.* 1976, Finley and Dieter 1978, Dieter and Finley 1978, 1979, Kendall *et al.* 1981, Veit *et al.* 1983, Kendall and Scanlon 1982, 1984, Chasko *et al.* 1984, Fimreite 1984, Buerger *et al.* 1986, Pain and Rattner 1988, Trust *et al.* 1990, Redig *et al.* 1991, Franson and Smith 1999, Fair and Myers 2002, and for reviews see Scheuhammer 1987, Eisler 1988, Burger and Gochfeld 2000, Franson and Pain 2011).

Many factors may affect an individual bird's susceptibility to lead poisoning including its sex and breeding condition, the physical and chemical constituents of its diet and environmental factors such as temperature and food stress. For example, in some experimental studies, ingestion of just one lead gunshot has been sufficient to cause ill health or death in birds (*e.g.* Holladay *et al.* 2012, Pain and Rattner 1988), while in others, birds have survived higher doses. It is therefore difficult to generalise about the magnitude of impact on an individual bird of ingesting a set amount of lead from ammunition (unless this is large). However, it is currently considered that there are no identified "no observed adverse effect levels" (NOAEL) or "predicted no effect concentrations" (PNEC) for lead in humans (EFSA 2010) and thus likely for other vertebrates.

While the dose-response relationship can vary among individuals and species, the health impacts of exposure to lead show great consistency across experimental studies. When the large numbers of studies conducted are considered together, particularly those studies that have examined large numbers of birds over time, generalisations can be made. The diagnosis of large scale and geographically extensive wildfowl mortality from lead poisoning following gunshot ingestion was first reported in the USA in the 1950s (*e.g.* Bellrose 1959), supported by extensive *post mortem* data. These findings were subsequently further supported by numerous experimental studies where captive wildfowl were fed lead gunshot (see above). Studies of survival of birds in relation to exposure to lead gunshot have also been conducted. Tavecchia *et al.* (2001) analysed recoveries between 1960 and 1971 of adult mallard *Anas platyrhynchos* ringed in the Camargue, France, for which the amount and type of lead exposure (ingested or embedded gunshot) had been determined by X-radiography. Ingested gunshot was present in the gizzard of 11% of birds and embedded gunshot was present in 23% of birds. Annual survival of mallards containing more than one gunshot in the gizzard was 19% lower than in unaffected birds. Survival was also lower by 19% for birds with any embedded gunshot and the effects of gizzard and embedded gunshot together were additive. Based upon the proportion of birds with gunshot in the gizzard and the estimated effect of gunshot on survival, these authors estimated that 1.5% of wintering mallards may die from lead poisoning due to ingested gunshot every year in the Camargue. Mortality from embedded gunshot and wounding would be additional to this. Guillemain *et al.* (2007), analysed recovery data from 40,000 teal *Anas crecca* that had been trapped and X-rayed in the Camargue, France (1957–1978), and also found reduced survival from one or more ingested pellets.

In addition to the direct impacts of lead on welfare and survival, indirect effects are likely to occur. These may include: increased susceptibility to infectious disease *via* lead's immunosuppressive effects (Grasman and Scanlon 1995, Trust *et al.* 1990); and increased susceptibility to death from a range of other causes, such as collision with power lines (Kelly and Kelly 2005 – *via* its effects on muscular strength and coordination) and being shot (*e.g.* shown by Bellrose 1959, Heitmeyer *et al.* 1993, Demendi and Petrie 2006 and others).

EXPOSURE ROUTES

There are four main routes by which birds and other wild animals (vertebrates and invertebrates) can be exposed to ammunition-derived lead:

1. Direct ingestion of spent lead gunshot deposited in the environment. This affects mainly wildfowl, other waterbirds and terrestrial gamebirds.
2. Ingestion of lead gunshot or bullets, or fragments from these, in the flesh of either dead or living animals that have been shot but remain unretrieved. This affects mainly predatory or scavenging birds, primarily raptors, and potentially some carnivorous mammals.
3. Ingestion of soil, water, or lower organisms contaminated with lead that has degraded from lead ammunition and entered the environment.
4. Absorption of lead mobilised from pellets shot into the tissues of animals that have been wounded but survived.

The first two of these appear to be the most significant exposure routes. We do not deal with the last exposure route in this paper because, while there is strong evidence that embedding of lead ammunition occurs (*e.g.* see Table 1), there is uncertainty about whether this causes increases in tissue lead levels. While there is evidence that ducks with embedded lead gunshot survive less well (Tavecchia *et al.* 2001), this might be due to wounding, irrespective of gunshot type, rather than the toxic effects of absorption of lead from embedded gunshot.

The exposure routes plus the outcomes are illustrated and summarised in Figure 1.

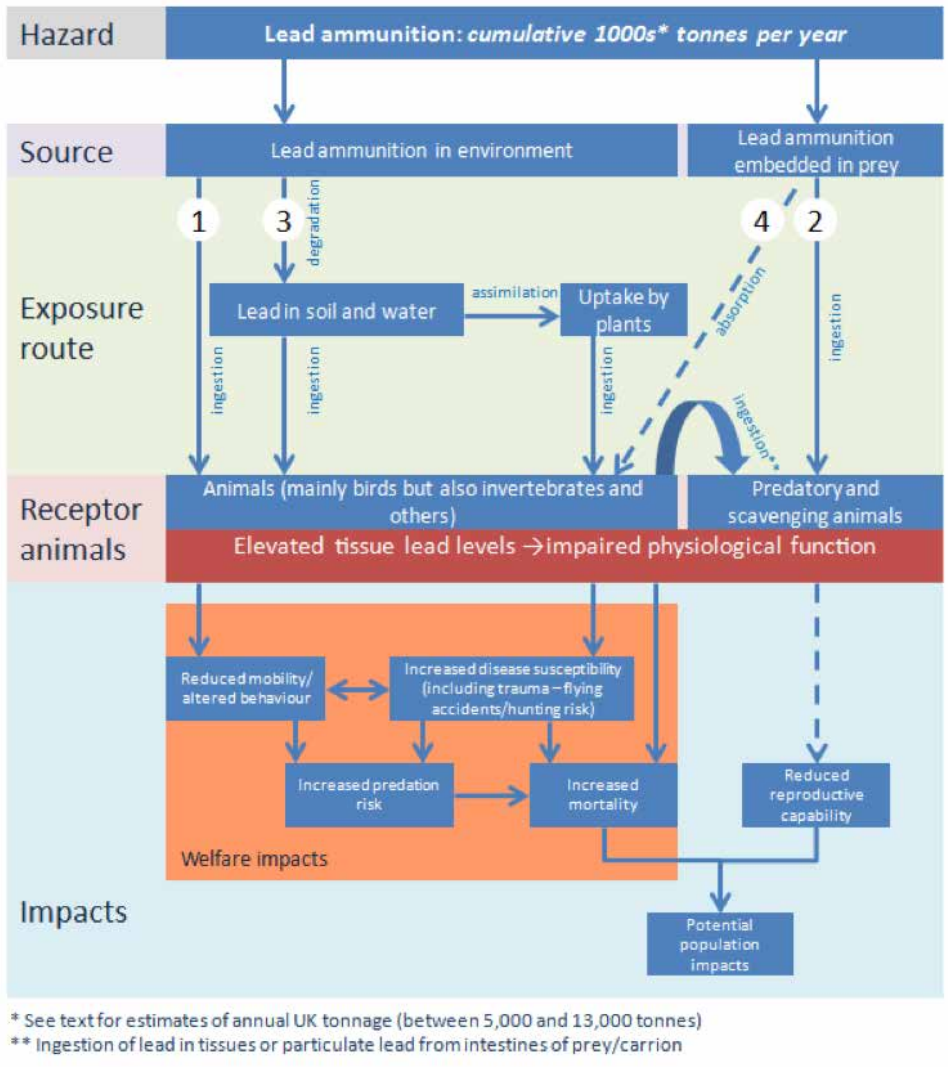

Figure 1: **Schematic illustrating and summarising the 4 exposure routes (see text) and range of impacts on wildlife of poisoning from lead ammunition sources.**

Amounts of lead from ammunition in the environment and its availability to wildlife

AMMUNITION DEPOSITED INTO THE ENVIRONMENT (*i.e.* OF RELEVANCE TO EXPOSURE ROUTES 1 AND 3)

The sport shooting of live quarry, clays and other targets is popular in the UK and most of the ammunition used is lead. Two thirds of the rural land in the UK is reportedly managed by shooting providers for a combination of reasons including shooting; active shoot management is undertaken on 2 million hectares of this (12% of the UK's rural land) (PACEC 2006). Many areas not managed specifically for shooting activities, including farmland and the foreshore, are also shot over for sport shooting, subsistence hunting and/or the control of pest animals (*e.g.* pigeons and corvids).

Each lead shotgun cartridge may contain between 100 and 600 lead gunshot depending on gunshot size, with a typical 30 g load containing approximately 300 individual number 6 gunshot. As gunshot leave the barrel of the gun they spread out thus even if the target is hit, most gunshot will miss. Only a small proportion of the gunshot from a single shotgun cartridge may be retrieved within a killed animal (see *e.g.* Cromie *et al.* 2010, Pain *et al.* 2010). Most lead gunshot fired from shotguns falls into the environment.

The tonnage of lead ammunition deposited annually into the UK environment is not precisely known. There are no official estimates of numbers of shooting participants, numbers of birds or other animals shot, or cartridges fired. However, broad estimates can be made using some published (*e.g.* PACEC 2006) and unpublished (*e.g.* shooting media, web articles and social media) sources. An estimated 28 million birds (gamebirds, wildfowl and pigeons) are shot annually in the UK (based upon PACEC 2006 and Aebischer 2013). The majority of gunshot used to kill these birds is composed of lead. Although there are restrictions on the use of lead for shooting wildfowl and/or over wetlands in the UK countries there is poor compliance with the legislation, at least in England (Cromie *et al.* 2002, 2010, 2015). In addition, wildfowl form only a small proportion of gamebirds shot. Assuming an average of 3-8 shots per bird (based on shooting web articles and social media) and 30 g gunshot per cartridge this represents about 2,500 to 6,700 tonnes of lead gunshot fired at gamebirds annually, most of which will fall into the environment. This excludes the gunshot used on the hundreds of thousands of rabbits and hares (combined), and numerous animals shot as part of pest control activities.

For target shooting, including clay pigeon shooting, the vast majority of the ammunition used is likely to be lead, probably to conform with International Shooting Sports Federation (ISSF) rules (Thomas and Guitart 2013). In 1991, it was reported that 220 million clay pigeons were used in the UK with at least one shot fired at each (B Carter, Clay Pigeon Shooting Association, *pers. comm.*; cited in Mellor and McCartney 1994). With a 28 g load commonly used to shoot clays and a number 8 cartridge (containing approximately 400 gunshot) this represented a minimum annual release of 6,160 tonnes of lead gunshot (approximately 88 billion individual gunshot) at the time, with a predicted rise in the popularity of clay shooting.

This suggests that approximately 8,000-13,000 tonnes of lead gunshot are used in the UK each year. This estimate is not precise and depends upon the accuracy of the assumptions in the estimate. It has been suggested by knowledgeable sources from the shooting community that approximately 5,000 tonnes a year of gunshot is used for all shooting combined although we have been unable to source any published data to substantiate this. These two figures are broadly similar and irrespective of the precise figure, thousands of tonnes of lead gunshot are deposited, and accumulate, in the UK environment annually, representing tens of billions of individual pellets. Ammunition used for target shotgun shooting is concentrated in and around target shooting clubs. Ammunition used for live quarry shooting is distributed, to variable degrees, across large tracts of the countryside.

The tonnage of bullets used annually (excluding those used by the police and/or the military) is considerably smaller, probably in the range of a few hundred tonnes a year. In areas of intensive lead bullet usage (*e.g.* firing ranges), bullets or fragments thereof are found deposited within the environment (*e.g.* Vantelon *et al.* 2005, Lewis *et al.* 2001). The authors are not aware of UK studies investigating the density of bullets in the environment in areas of lower intensity of usage such as places in which other more "typical" UK live quarry shooting activities occurs. However, it is probable that bullets that either miss their targets or travel through their targets are deposited within the environment, most likely penetrating whatever substrate by a distance dependent on the density of the substrate they hit, and the velocity and mass of the projectile.

A large amount of lead gunshot, bullets, and fragments thereof, is deposited in the environment annually and accumulates over time. Much of this may be available for animals to ingest directly, probably mistakenly for food, for grit, or inadvertently along with soil or foodstuffs.

Gunshot densities in the environment tend to be highest in areas of intense and/or regular hunting/shooting pressure. They typically range from just a few to hundreds per square metre (*e.g.* Mudge 1984, Spray and Milne 1988, Mellor and McCartney 1994 for the UK, and Mateo 2009 for Europe) but thousands can be found per square metre in some situations. For example, O'Halloran *et al.* (1988) reported gunshot density in the vicinity of a clay pigeon shooting range in Lough Neagh, County Antrim, of 2,400 gunshot/m^2 in the upper 5 cm of shoreline in front of the range and with gunshot being retrieved on the lake bed up to 60 m from the shore.

Lead is a relatively stable metal under most conditions and remains as pellets of gunshot for considerable periods of time. It has been used in the UK for over two centuries and, indeed, the potential for a "historical legacy" of gunshot remaining available to wildlife is an important aspect of the epidemiology of lead poisoning in wildlife. Complete decomposition of particulate lead probably takes tens or hundreds of years under most conditions (Scheuhammer and Norris 1996, Rooney *et al.* 2007). Gunshot degradation is caused by a combination of physical erosion/abrasion, which is accelerated in coarse and gritty soils and/or those with marked levels of movement and chemical activity.

Densities of lead gunshot in the soil tend to increase over time if lead gunshot continues to be used. However, gunshot will generally sink slowly through the soil and new soil accumulates above the gunshot with rates of sinking affected by soil density and other characteristics. Hartikainen and Kerko (2009) found that on the coarse stony soil of a shooting range in southern Finland, lead gunshot migrated downwards relative to the surface at a rate of some 2-3 mm per year. Flint (1998) found in various wetland types to which gunshot was added experimentally that most gunshot was still within the top 4 cm of sediment three years after deposition. In the Camargue marshes (southern France), assuming a constant settlement rate, Tavecchia *et al.* (2001) estimated a half-life of gunshot in the first 0-6 cm, thus available to waterfowl, for 46 years, with complete settlement beyond this depth after 66 years. Flint and Schamber (2010) found that 10 years after seeding experimental plots on tundra wetlands with number 4 gunshot, about 10% remained in the top 6 cm and >50% in the top 10 cm. These authors predicted that it would probably require >25 years for spent lead pellets to exceed depths at which waterfowl forage. However, one would expect the proportion of pellets available to feeding waterfowl to decrease with time over this period. Lead gunshot may become less available when redistributed by cultivation (*e.g.* Thomas *et al.* 2001), and some farming practices could hypothetically make lead gunshot deposited decades ago more available (Chrastny *et al.* 2010, Rooney and McLaren 2000, Stansley *et al.* 1992, White Young Green Environmental 2006), as can the lowering of water levels (Spray and Milne 1988).

While a historical legacy of deposited gunshot exists, there is good evidence that the majority of gunshot ingested by wildfowl is that most recently deposited. Anderson *et al.* (2000) found that in the fifth and sixth years after a nationwide ban on the use of lead gunshot for shooting waterfowl in the USA, 75.5% of 3,175 gunshot ingested by a sample of 15,147 mallard on the Mississippi flyway were non-toxic.

LEAD AMMUNITION IN THE TISSUES OF GAME SPECIES THAT CAN BE CONSUMED BY PREDATORS AND SCAVENGERS (*i.e.* OF RELEVANCE TO EXPOSURE ROUTE 2)

Of the tens of millions of animals shot in the UK each year using lead ammunition, an unknown proportion of the carcasses is not recovered and hence is potentially available to scavengers. For many of the tens of thousands of red deer *Cervus elaphus* shot per year, the viscera are discarded in the field and they, and any remnants of lead ammunition within them, are potentially available to scavengers. A further additional set of animals are wounded by gunshot and bullets but survive and may carry remnants of lead ammunition in their bodies. These animals may be eaten by predators, perhaps selected as prey because of their weakened condition, or die later and be eaten by scavengers.

The tissues of game animals killed using shotgun cartridges usually contain some of the gunshot that struck the animal and killed it. Pain *et al.* (2010) performed X-radiography on 121 entire carcasses of wild red grouse *Lagopus lagopus*, red-legged partridge *Alectoris rufa*, pheasant *Phasianus colchicus*, mallard, woodpigeon *Columba palumbus* and woodcock *Scolopax rusticola* killed by shooting and obtained from retailers and shoots in the UK (16 – 26 individuals per species). Eighty seven percent of all birds examined had whole gunshot, large

fragments, small fragments or some combination of the three types detectable by X-radiography.

Substantial fragmentation of lead gunshot occurs when gamebirds and waterfowl are killed using gunshot. X-radiographic studies show that gamebirds and mammals shot either with lead gunshot or lead bullets often contained lead fragments which were small, numerous and widely dispersed in edible tissues away from the wound canals (*e.g.* Pain *et al.* 2007, 2010, Knott *et al.* 2010 for UK studies, and Dobrowolska and Hunt *et al.* 2006, 2009, Melosik 2008, Krone *et al.* 2009, Grund *et al.* 2010, for relevant studies elsewhere).

In addition to studies of lead ammunition and fragments in dead animals and animal parts, numerous studies have used X-radiography to investigate proportions of live birds, predominantly wildfowl, carrying embedded gunshot in their tissues. The proportion of animals, or parts thereof, shot using lead ammunition which are potentially available to predators and scavengers is unknown for some species, but may be high for quarry species such as ducks and geese (see Table 1 for live wildfowl), and must represent hundreds of thousands of animals potentially contaminated with ammunition-derived lead per year entering the food supply of wild predators and scavengers.

Even in some protected species, such as swans, that cannot be legally shot, a high proportion may contain embedded gunshot (Newth *et al.* 2011, see Table 1).

Sales of non-lead ammunition in the UK are low. Use of lead ammunition is permitted throughout the UK for the majority of shooting. Restrictions on the use of lead gunshot apply to the shooting of wildfowl and coot *Fulica atra* and moorhen *Gallinula chloropus* anywhere in England and Wales, and also for any species over certain listed wetland areas in these countries. In Scotland and Northern Ireland restrictions apply to all shooting with gunshot of any animal over wetlands, although all species including wildfowl may be shot with lead ammunition away from wetland areas in these countries. Wildfowl comprise a small proportion of birds shot, and ammunition composed primarily of lead is used for the vast majority of shooting of game in non-wetland habitats. The use of lead ammunition to shoot wildfowl has not been lawful in England since 1999, but recent compliance studies (see Cromie *et al.* 2010, 2015) found between 68 - 77% of wild duck carcasses bought from game dealers in England had been shot using lead ammunition. Hence, it is clear that the vast majority of game animals shot in the UK are killed using lead ammunition.

AMMUNITION-DERIVED LEAD IN SOILS AND WATER (HAVING ORIGINATED FROM INTACT GUNSHOT OR BULLETS DEPOSITED IN THE ENVIRONMENT) (*i.e.* OF RELEVANCE TO EXPOSURE ROUTE 3)

Wildlife may be exposed, primarily *via* ingestion, to lead of ammunition origin that has moved from deposited lead ammunition into the soil and water. While elemental lead is very stable under neutral pH conditions, the surface of ammunition will be chemically transformed in the environment, and the lead compounds formed, which will vary with soil conditions, will play an important part in determining the mobility of lead. In water the solubility of different compounds is related to pH, amount of calcium, salinity and the presence of humic material.

Soils and sediments act as an environmental sink for lead. Lead in soil may occur in a variety of chemical forms (*e.g.* as carbonates, sulphides *etc.*) and fractions, *e.g.* including exchangeable, adsorbed and organic complexes. Lead is strongly adsorbed to soil organic matter, silicate clays, peat and iron oxides. Consequently, under most conditions the majority of lead that enters soils and is transformed into lead compounds is likely to be retained in the upper layers and not leach to any great extent into the subsoil and groundwater. However, although this is a general rule, the mobility of lead in soils is nonetheless highly variable in relation to environmental conditions and is thus site specific. Research into the degradation/transformation of metallic lead from gunshot or bullets, (*e.g.* see Cao *et al.* 2003, McLaren *et al.* 2009, Sanderson *et al.* 2012, Sullivan *et al.* 2012) illustrates the varied impacts temperature, moisture and soil chemistry have on the rate of degeneration/transformation of metallic lead gunshot or bullets, the transformation products, and the rate of passage of lead and its transformation products through the soil profile.

Under most environmental conditions gunshot degrades only slowly and in addition to the chemical processes described above, degradation may be influenced by physical erosion/abrasion, which is accelerated in coarse and gritty soils and/or those with considerable soil movement. Movement of lead through the soil may also be influenced by other factors, such as precipitation and snow melt.

The National Sports Shooting Foundation (the trade association for America's firearms industry) has produced a report on 'Lead Mobility at Shooting Ranges', a synthesis of which is given in its

Table 1: **Summary table of prevalence of embedded gunshot in live trapped wildfowl species.**

Species	Country	Embedded shot (%)	Reference
Bewick's swan *Cygnus columbianus bewickii*	UK	31.2	Newth *et al.* (2011)
Whooper swan *Cygnus cygnus*	UK	13.6	Newth *et al.* (2011)
Migratory wild geese	Germany	21	Krone *et al.* (2009)
Pink-footed goose *Anser brachyrhynchus*	Denmark (1990-92)	24.6 (juvs)-36.0 (adults)	Noer and Madsen (1996)
	Denmark (1998-2005)	9.2-22.2	Noer *et al.* (2007)
Greylag goose *Anser anser*	Spain, Doñana	44.4	Mateo *et al.* (2007)
Canada goose *Branta canadensis*	Canada (Maritimes)	32	CWS, unpublished data
	USA	42	Funk (1951)
Small Canada goose *Branta canadensis parvipes*	Canada	≥25	Macinnes *et al.* (1974)
Brant goose *Branta bernicla*	USA	20	Kirby *et al.* (1983)
Barnacle geese *Branta leucopsis*	Denmark	13	Holm and Madsen (2013)
Mallard *Anas platyrhynchos*	UK	17.6	WWT unpublished (1980s)
	France, Camargue	23.4	Tavecchia *et al.* (2001)
	Netherlands	1.4-3.4 †	Lumeij and Scholten (1989)
	Netherlands	22-68 ††	Lumeij and Scholten (1989)
	Canada	28	Elder (1950)
	USA	13	Funk (1951)
	USA	27	Murdy (1952)
Northern pintail *Anas acuta*	UK	27.1	WWT unpublished (1980s)
	USA	13	Funk (1951)
Northern shoveler *Anas clypeata*	UK	25.8	WWT unpublished (1980s)
Gadwall *Anas strepera*	UK	26.3	WWT unpublished (1980s)
American black duck *Anas rubripes*	Canada (Maritimes)	12–18	CWS, unpublished data
Common teal *Anas crecca*	France, Camargue	4.4-9.6	Guillemain *et al.* 2007
Pochard *Aythya ferina*	UK	25.0	WWT unpublished (1980s)
Tufted duck *Aythya fuligula*	UK	14.9	WWT unpublished (1980s)
Canvasback *Aythya valisineria*	USA	29	Perry and Geissler (1980)
Lesser scaup *Aythya affinis*	USA	10	Perry and Geissler (1980)
Redhead *Aythya americana*	USA	15	Perry and Geissler (1980)
Ring-necked duck *Aythya collaris*	USA	21	Perry and Geissler (1980)
Common eider *Somateria mollissima*	Canada (Maritimes)	20-35	CWS, unpublished data

†Gizzard wall †† Whole body and an estimate.

Table 2: **Soil lead concentrations in shooting and control areas in the UK**

Gunshot source	Soil lead (mg/kg) (average of 72 mg/kg in principal English topsoil)		Reference
	Shooting Site	Control Site	
Game shooting wood and pheasant rearing area	160 (wood) 68 (field)	60 (wood) 44 (field)	Sneddon *et al.* (2009)
Clay pigeon shoot (100-175 m from stands)	Mean of 3,038 Max. of 8,172	72	Clements (1997)
Clay pigeon fall out zone on acid peat bog	Mean of 306 Max. of 15,700	67	White Young Green Environmental (2006)
Clay pigeon shoot for 20 years (80-100 m from stands)	5,000 to 10,600	-	Mellor and McCartney (1994)

executive summary[1]. Adriano (1986) provides comprehensive information on the biogeochemistry and bioavailability of lead in the terrestrial environment.

In areas of lead ammunition deposition, soil lead concentrations can be extremely elevated, *e.g.* from a few to hundreds of times higher than in control soils; some examples from the UK are given in Table 2. The figures here can be compared with average soil lead concentration of 72 mg/kg in principal English topsoil (with English soils in the principal domain, covering 94% of the area of England, having 'Normal Background Concentrations' of up to 180 mg/kg - British Geological Survey, see Ander *et al.* 2013). A limited number of studies is available either measuring lead in water from sites contaminated with lead, or lead in biota exposed to water contaminated by lead from ammunition (*e.g.* Heier *et al.* 2009, Stromseng *et al.* 2009). These provide evidence that in some areas where shooting occurs regularly and/or at high intensity, and in and possibly close to the gunshot fallout areas, water lead concentrations can be elevated above those at control sites. The extent to which such contamination is likely to affect sites downstream of shooting areas is unknown, but the likelihood of broader watershed contamination appears low, and it seems likely that the majority of the water contamination will be relatively local.

PATHWAYS OF EXPOSURE TO LEAD FROM AMMUNITION

DIRECT INGESTION OF AMMUNITION-DERIVED LEAD BY WILD BIRDS (EXPOSURE ROUTE 1)

The first published record of a bird poisoned following lead gunshot ingestion in the UK was for a pheasant, 139 years ago (Calvert 1876). Recognition of the direct ingestion of spent gunshot and subsequent mortality from lead poisoning in wildfowl and a range of other birds, primarily other waterbirds and terrestrial birds such as Galliformes, grew throughout the last century. This pathway of ingestion of lead gunshot has been extensively documented and reviewed (*e.g.* for global studies see Bellrose 1959, Franson and Pain 2011, and papers in Pain 1992 and Watson *et al.* 2009; for the UK see Olney 1960, 1968, Owen and Cadbury 1975, Thomas 1975, Thomas 1982, Brown *et al.* 1992, Thomas *et al.* 2009, Parslow *et al.* 1982, Mudge 1983, Street 1983, Spray and Milne 1988, Butler 2005, Butler *et al.* 2005, Potts 2005, O'Connell *et al.* 2008, Newth *et al.* 2012). Gunshot ingestion levels by wildfowl from UK studies and terrestrial birds from the UK and elsewhere are summarised in Tables 3 and 4 respectively.

Mateo (2009) provided a summary of historic prevalence of lead gunshot ingestion in 19 species of wildfowl from Europe including the UK (15 of which are species of swans, geese and ducks from northern Europe). Levels of gunshot ingestion varied among sites and species with an overall combined level

[1]Available online (see http://www.nssf.org/ranges/rangeresources/library/detail.cfm?filename=facility_mngmnt/environment/executive_summary.htm&CAT=Facility%20Management)

Table 3: **Summary of proportions of wildfowl with ingested gunshot from UK studies of hunter-shot birds and birds found dead.**

Species	Birds found dead All data from Newth *et al.* (2012) and WWT *post mortem* database.			Birds shot by hunters			References (birds shot by hunters only)
	N (sample size)	Number with ingested gunshot	% with ingested gunshot	N (sample size)	Number with ingested gunshot	% with ingested gunshot	
Mallard *Anas platyrhynchos*	479	15	3.1	2016	91	4.5	Olney (1960), Thomas (1975), Mudge (1983), Street (1983)
European wigeon *Anas penelope*	24	0	0	862	0	0	Olney (1960), Thomas (1975), Mudge (1983)
Common teal *Anas crecca*	68	1	1.5	1188	12	1	Olney (1960), Thomas (1975), Mudge (1983)
Northern shoveler *Anas clypeata*	16	0	0	133	3	2.3	Olney (1960), Thomas (1975), Mudge (1983)
Pochard *Aythya ferina*	72	12	16.7	130	11	8.5	Olney (1968), Thomas (1975), Mudge (1983)
Northern pintail *Anas acuta*	60	5	8.3	162	21	13	Thomas (1975), Mudge (1983)
Tufted duck *Aythya fuligula*	79	2	2.5	103	9	8.7	Thomas (1975), Mudge (1983)
Gadwall *Anas strepera*	65	0	0	42	2	4.8	Thomas (1975), Mudge (1983)
Goldeneye *Bucephala clangula*	1	0	0	15	1	6.7	Mudge (1983)
Pink-footed goose *Anser brachyrhynchus*	25	2	8	73	2	2.7	Mudge (1983)
White-fronted goose *Anser albifrons*	8	0	0	30	0	0	Mudge (1983)
Greylag goose *Anser anser*	133	9	6.8	42	3	7.1	Mudge (1983)
Barnacle goose *Branta leucopsis*	99	13	13.1	61	0	0	Mudge (1983)
Total	**1129**	**59**	**5.2**	**4857**	**155**	**3.2**	
Mute swan *Cygnus olor*	548	27	4.9	548*	16*	3.0*	*Swans are protected and numbers of 'shot' swans containing ingested lead are estimated from the ratio of gunshot in found dead to hunter-shot birds in the other species.
Whooper swan *Cygnus cygnus*	414	98	23.7	414*	60*	14.6*	
Bewick's swan *Cygnus columbianus bewickii*	99	13	13.1	99*	8*	8.1*	

Totals combined from all studies cited. The study of Olney (1960) did not cite the origin of the birds but wildfowlers were thanked in the acknowledgements for provision of birds and the text indicated that a small number of birds sent in for *post mortem* examination had been found to suffer from lead poisoning (although it did not state whether these were included). We have assumed that the birds in Olney's study were hunter shot. For the study of Thomas (1975) we have subtracted six mallard with gunshot in the gizzard as six birds had 'shot in' gunshot and it was unclear whether these had already been excluded from the results.

for mallard of 3.6%, pintail *Anas acuta* 5.4%, and pochard *Aythya ferina* 9.3% in northern European wetlands. The majority of studies summarised by Mateo (2009) appear to be of birds shot by hunters, though in some cases trapped birds were included. Studies conducted in the UK reported broadly similar levels of gunshot ingestion to those elsewhere in Europe, although they vary among sites and species (Table 3).

More recently, Newth *et al.* (2012) reported lead poisoning in wildfowl (between 1971 and 2010) in the UK where the majority of cases of birds dying of lead poisoning (75% of 251) still had lead gunshot in various stages of dissolution in their gizzards. The *post mortem* data used for this study revealed a small number (13) of lead poisoned birds with >40 pellets within the gizzard, three of which contained more than 100 pellets, including a Canada goose *Branta canadensis* whose gizzard contained 438 pellets, while Spray and Milne (1988) reported a mute swan with 844 pellets. Species suffering lead poisoning from ingested gunshot included those feeding in water and wetlands, as well as grazing species including geese and swans where a large proportion of time is spent feeding on agricultural land (Newth *et al.* 2012). Gunshot ingestion levels in birds found dead from this paper and WWT's database are given in Table 3. Of a subset of 104 whooper swans diagnosed as having died of lead poisoning, 86% contained shotgun pellets in the gizzard.

More studies on lead poisoning have been conducted on wildfowl than other taxa. However, where lead ingestion has been investigated in other taxa that feed in areas shot-over using lead gunshot it has generally been found. Table 4 summarises some of the studies that illustrate gunshot ingestion in a range of non-wildfowl waterbirds and in terrestrial birds. This is not comprehensive but illustrative of the range of different birds that can be affected.

Several methods have been used to estimate the proportion of wild birds with ingested gunshot in the gizzard or digestive tract and various biases may be associated with them. Hunter-shot birds will be subject to the biases involved in hunting, *e.g.* young birds are often over-represented in hunting bags. Also ingestion of lead may remove many poisoned individuals from a population (*via* lead-related morbidity and mortality) or conversely lead ingestion may disable birds sufficiently to make them more likely to be harvested (*e.g.* Bellrose 1959, Heitmeyer *et al.* 1993, Demendi and Petrie 2006). In field experiments Bellrose (1959) found that mallard dosed with lead gunshot were more vulnerable to being shot than undosed controls – by 1.5 times, 1.9 times and 2.1 times for birds dosed with one, two and four No. 6 gunshot respectively. Trapping may potentially introduce biases, but little information exists. Ingestion levels in birds found dead may also be subject to confounding factors. Firstly, "found dead" studies are biased towards those species most likely to be visible to humans *e.g.* large, white or close to human habitation. The nature of lead poisoning as a debilitating condition may make affected individuals more prone to disappearing into vegetation and to scavenging and predation (Sanderson and Bellrose 1986, Pain 1991). Moreover, gunshot may be ground down or dissolved in the bird's alimentary canal and thus not be apparent on radiographs or at *post mortem* examination. While proportions of birds found dead with ingested gunshot in the gizzard may not accurately reflect the situation in the wild population, finding gunshot in found dead birds obviously illustrates the pathway of ingestion.

Despite these biases and confounding factors, any one or all of these methods can be used to compare the prevalence of ingestion across space and time. Studies from across the world have shown that levels of gunshot ingestion are influenced by factors including species' feeding habits, gunshot density and availability (influenced by substrate type and shooting intensity, duration and season) and grit availability (*e.g.* Bellrose 1959, Flint 1998, Mudge 1983, Thomas *et al.* 2001, Demendi and Petrie 2006 – see also reviews cited above).

Several means can be used to establish or infer the provenance of elevated tissue lead concentrations in birds. Ratios of stable lead isotopes in materials vary according to the geological origin of the lead. Lead isotope ratios can therefore be compared between animal tissue samples, lead from ammunition and the other potential sources that exist in the area where the animal lived and this can help to identify or exclude some potential sources of the lead. Lead isotope studies have linked gunshot ingestion with elevated tissue lead concentrations in a range of wild birds in a number of studies from around the world. These studies support ammunition-derived lead as the major contributor to widespread elevated tissue lead concentrations in wild birds (*e.g.* Scheuhammer and Templeton 1998, Scheuhammer *et al.* 2003, Svanberg *et al.* 2006, Martinez-Haro *et al.* 2011).

Temporal or spatial correlations between elevated tissue lead levels in birds and hunting activities can also help establish the primary source(s) of lead exposure. Studies have compared

Table 4: **A selection of non-wildfowl avian species reported as ingesting lead gunshot from the environment.**

Species	Countries	References
Galliformes (largely terrestrial habitats)		
Chukar *Alectoris chukar*	USA	Hanspeter and Kerry (2003)
Grey partridge *Perdix perdix*	Denmark, UK	Clausen and Wolstrup (1979), Keymer and Stebbings (1987), Potts (2005)
Red-legged partridge *Alectoris rufa*	UK	Butler (2005)
Common pheasant *Phasianus colchicus*	Denmark, UK, USA	Calvert (1876), Elder (1955), Clausen and Wolstrup (1979), NWHL (1985), Dutton and Bolen (2000), Butler *et al.* (2005)
Wild turkey *Meleagris gallopavo*	USA	Stone and Butkas (1978)
Scaled quail *Callipepla squamata*	USA	Campbell (1950)
Northern bobwhite quail *Colinus virginianus*	USA	Stoddard (1931), Keel *et al.* (2002)
Ruffed grouse *Bonasa umbellus*	Canada	Rodrigue *et al.* (2005)
Helmeted guineafowl *Numida meleagris*	Canada	Hunter and Haigh. (1978)
Columbiformes (largely terrestrial habitats)		
Rock pigeon *Columba livia*	USA , Belgium	DeMent *et al.* (1987), Tavernier *et al.* (2004).
Mourning dove *Zenaida macroura*	USA	Locke and Bagley (1967), Lewis and Legler (1968), Best *et al.* (1992), Schulz *et al.* (2002).
Gruiformes (largely wetland habitats)		
Sandhill crane *Grus canadensis*	USA	Windingstad *et al.* (1984), NWHL (1985)
Clapper rail *Rallus longirostris*	USA	Jones (1939)
King rail *R. elegans*	USA	Jones (1939)
Virginia rail *R. limicola*	USA	Jones (1939)
Common moorhen *Gallinula chloropus*	Europe, UK, USA	Jones (1939), Locke and Friend (1992), Thomas (1975)
Common coot *Fulica atra*	France	Pain (1990)
American coot *F. americana*	USA	Jones (1939)
Charadriiformes (largely wetland habitats)		
American woodcock *Scolopax minor*	Canada	Scheuhammer *et al.* (1999, 2003)
Black-necked stilt *Himantopus mexicanus*	USA	Hall and Fisher (1985)
Long-billed dowitcher *Limnodromus scolopaceus*	USA	Hall and Fisher (1985)
Common snipe *Gallinago gallinago*	France, UK	Beck and Granval (1997), Thomas 1975
Jack snipe *Lymnocryptes minimus*	France	Beck and Granval (1997)
Dunlin *Calidris alpina*	Canada	Kaiser *et al.* (1980)
Ciconiiformes (largely wetland habitats)		
White-faced ibis *Plegadis chihi*	USA	Hall and Fisher (1985)
Ciconiiformes (largely wetland habitats)		
Caribbean flamingo *Phoenicopeterus ruber ruber*	Mexico	Schmitz *et al.* (1990)

See also Kimmel and Tranel (2007) for examples.

tissue lead levels in wildfowl before and after bans on the use of lead gunshot for wildfowl hunting (*e.g.* Samuel and Bowers 2000, Stevenson *et al.* 2005), or in areas where lead gunshot may be used *vs* areas where only non-toxic gunshot may be used (*e.g.* Franson *et al.* 2009). Scheuhammer and Dickson (1996) investigated the geographical pattern of elevated lead concentrations in several thousand wing bones from young-of-the-year ducks collected in Canada to investigate their relationship with activities known to cause environmental lead contamination, *i.e.* waterfowl hunting, non-ferrous metal mining/smelting, and urban/industrial development. Ingestion of spent-lead gunshot was the likely primary source of elevated lead exposure for wild ducks in Canada. In areas of significant waterfowl hunting, a widespread pattern of elevated bone-lead was found, rather than few small local sites of high lead exposure. However, lead contamination of bones of young ducks was significantly correlated with proximity to metal mining sites; this accounted for about a quarter of the total area characterised by a high incidence of elevated lead exposure.

These studies support ammunition-derived lead as the major source of widespread lead exposure.

INGESTION OF AMMUNITION DERIVED LEAD IN THE TISSUES OF DEAD OR LIVE GAME SPECIES (EXPOSURE ROUTE 2)

Many bird species worldwide, including New and Old World vultures, eagles, kites, buzzards, caracaras, gulls and corvids, frequently scavenge tissue from carcasses of dead vertebrates and parts of their bodies discarded by hunters. Predatory birds that may consume, and perhaps select, wounded animals carrying ammunition include species from the same taxonomic groups, but also include owls, falcons and a wider range of accipitrid raptors. In the UK, red kite *Milvus milvus*, golden eagle *Aquila chrysaetos*, white-tailed eagle *Haliaeetus albicilla*, buzzard *Buteo buteo*, raven *Corvus corax*, carrion crow *C. corone*, hooded crow *C. cornix* and magpie *Pica pica* are the bird species most likely to scavenge from carcasses or discarded viscera of game animals. All species of raptors and owls could potentially kill and feed upon a game animal with fragments of lead ammunition shot into its tissues. Because they frequently prey upon waterfowl which may be contaminated with embedded lead gunshot (Table 1), western marsh harriers *Circus aeruginosus* and peregrine falcons *Falco peregrinus* (which prey upon a wide range of medium sized birds) are the raptor species which might be expected to be most exposed to ammunition-derived lead *via* this route.

Ingestion of lead ammunition or ammunition fragments by predatory and scavenging birds has been reported for decades. Some of the earliest studies involved the poisoning of bald eagles *Haliaeetus leucocephalus*, which frequently feed on wildfowl in the USA (Kaiser *et al.* 1979, Feierabend and Myers 1984, Reichel *et al.* 1984), golden eagles (Craig *et al.* 1990) and the California condor *Gymnogyps californianus*, a Critically Endangered species whose remaining small population in the wild was almost driven to extinction by lead poisoning caused by scavenging upon discarded viscera and carcasses of unretrieved large game animals such as deer (Rideout *et al.* 2012). Numerous studies have reported ingested ammunition-derived lead in white-tailed eagles (*e.g.* Kenntner *et al.* 2001 in Germany and Austria, Helander *et al.* 2009 in Sweden), and in a proportion of the carcasses of both this species and of Steller's sea eagles *Haliaeetus pelagicus* and mountain hawk eagles *Spizaetus nipalensis* in Hokkaido, Japan (Saito 2009).

Examination of regurgitated birds' food pellets provides additional information on the frequency of ingestion of remnants of lead ammunition. X-radiographs of regurgitated food pellets from a roost site of red kites in the English Midlands found that a minimum of 2% contained lead gunshot (Pain *et al.* 2007). Since the study area included estates on which partridges and pheasants were shot with lead gunshot, scavenging of unrecovered shot birds or of wounded birds that died later could have been a route by which the red kites obtained the lead gunshot. Other studies have found that the frequency of occurrence of gunshot in regurgitated pellets is higher during than outside the hunting season. These include studies of western marsh harriers in France (Pain *et al.* 1997), eastern marsh harriers *Circus spilonotus* in Japan (Hirano *et al.* 2004) and white-tailed eagles in Sweden (Helander 1983).

Mateo *et al.* (2013) reviewed information on lead gunshot ingestion and lead poisoning in Spain, and reported the presence of lead gunshot in regurgitated pellets from red kites (in central Spain and Doñana), Egyptian vultures (in the Canary Islands), western marsh harriers (from the Ebro delta and Doñana), Spanish imperial eagles *Aquila adalberti* (from central Spain, Castilla-La Mancha and Doñana) and peregrine falcons (in Doñana). These authors reported that the incidence of ingestion of lead gunshot by the Spanish imperial eagle in Doñana varied between years in relation to goose hunting pressure, which in turn varies with water levels in the protected areas. For additional information on lead gunshot ingestion and poisoning of raptors in Spain see Mateo *et al.* (2007), and also Cerradelo *et al.* (1992),

Mateo *et al.* (2001), González and Hiraldo (1988), Castaño López (2005), Mateo *et al.* (1999), Gonzalez (1991), Garcia and Viñuela 1999, Donázar *et al.* (2002).

Krone *et al.* (2009) performed experiments on white-tailed eagles in which iron nuts of various sizes were inserted into carcasses or discarded viscera form which they fed. The eagles always avoided ingesting nuts of 7.7 mm diameter or larger, but ingested some of the nuts smaller than this (2.7 – 6.0 mm). For the smallest size of nuts used in the experiment (2.7 mm), 80% of the nuts presented were eaten. These nuts were considerably larger than most of the fragments of ammunition-derived metal seen in X-radiographs of deer carcasses and discarded viscera. Knott *et al.* (2010) found that 83% by weight of the radio-dense fragments they found in deer viscera had a diameter less than 1 mm and the largest fragment seen on the radiographs was only slightly larger than the smallest nuts used in the experiment. Hence, this experiment suggests that in a similar situation in the wild, were fragments from lead ammunition to be present in a carcass, many of these could be readily ingested whilst scavenging on the remains of game animals.

Several methods exist to infer the origin of elevated tissue lead concentrations and lead poisoning in predatory and scavenging birds. The most detailed isotopic studies have been conducted on California condors and they indicate that elevated lead exposure in free-living condors is mostly consistent with lead from ammunition rather than other sources (Church *et al.* 2006, Finkelstein *et al*. 2010, 2012, Rideout *et al*. 2012). Departure of blood lead isotope signature from the background pattern in free-living birds increased progressively as the total blood lead concentration increased, moving towards the isotopic signature of lead ammunition and bullet fragments retrieved from lead poisoned condors (Church *et al.* 2006, Finkelstein *et al.* 2012). Isotopic analysis also illustrates that ammunition-derived lead is the likely provenance of elevated tissue lead concentrations in a number of Steller's sea eagles and white-tailed eagles in Hokkaido, Japan (Saito 2009 – with rifle ammunition implicated). Legagneux *et al.* (2014) found that blood lead concentrations in the raven, a scavenging species, increased over the moose *Alces alces* hunting season in eastern Quebec, Canada, and that birds with elevated blood lead levels had isotopic signatures that tended towards those of ammunition. Several other studies, including on red kites in England (Pain *et al.* 2007) and white-tailed eagles in Sweden (Helander *et al.* 2009) show isotopic signals consistent with ammunition sources in birds with elevated tissue lead, although they do not exclude all possible non-ammunition sources of lead. Lead concentrations in the livers of a sample of red kites and sparrowhawks *Accipiter nisus* found dead in Britain were not elevated and lead isotope signatures were distinct from that of leaded petrol, marginally overlapped with that for coal, and overlapped more with those for lead ammunition (Walker *et al.* 2012). The isotopic signatures in this study may reflect the fact that liver concentrations were low and could have resulted from multiple diffuse sources.

A number of studies of scavenging and predatory birds have investigated the relationship between tissue (generally blood) lead levels and spatial and temporal variation in exposure to food contaminated with ammunition-derived lead. Green *et al.* (2008) showed that blood lead concentrations in California condors tended to rise rapidly when satellite-tagged condors spent time during the autumn deer-hunting season in areas with high levels of deer hunting, but that visits to these same areas outside the hunting season, and visits to other areas with low levels of deer hunting at any time of year were not associated with rises in blood lead levels. Craighead and Bedrosian (2008) found that 47% of blood samples in ravens in the USA collected during the large game (mainly deer) hunting season had elevated blood lead (>10 µg/dl), compared with 2% outside the hunting season; these results were consistent with those of Legagneux *et al.* (2014) cited above. Kelly *et al.* (2011) compared blood lead concentrations in golden eagles and turkey vultures *Cathartes aura* prior to and one year following implementation of a ban (in 2008) on the use of lead ammunition for most hunting activities in the range of the California condor in California; lead exposure in both species declined significantly after the ban. Similarly, Pain *et al.* (1997) found that geometric mean blood lead levels were 3-4 times higher in free-flying live-trapped western marsh harriers during the hunting season in France than outside the hunting season. Kelly and Johnson (2011) found that the blood lead concentrations of turkey vultures in California were significantly higher during the large game hunting season than outside it. Gangoso *et al.* (2009) found that the geometric mean concentration of lead in the blood of Egyptian vultures in the Canary Islands was about four times higher during the hunting season than outside it. While these studies show consistent results, it is nonetheless worth noting that most studies which contrast the blood lead concentration of birds within and outside the hunting season underestimate the underlying difference in exposure to lead. This arises because blood lead remains high for some time, often several weeks, after the ingestion of lead has ceased. Consequently, some blood samples obtained in the

early part of the non-hunting season will still contain appreciable amounts of lead acquired during the hunting season.

Widely available reference works summarise observations of the principal food sources of mammals and it is apparent that many mammal species worldwide frequently scavenge tissue from carcasses of dead vertebrates and parts of their bodies discarded by hunters (*e.g.* see Legagneux *et al.* 2014). Badger *Meles meles*, red fox *Vulpes vulpes* and pine marten *Martes martes* are the mammal species in the UK most likely to scavenge from these sources. We are not aware of direct observations of ingestion of ammunition-derived lead fragments by scavenging or predatory mammals in the UK. However, it seems probable from the feeding behaviour of many species, in which large chunks of meat and some bone fragments are swallowed, that some ingestion of remnants of ammunition occurs.

INGESTION OF CONTAMINATED SOIL, WATER OR BIOTA (EXPOSURE ROUTE 3)

Field studies provide evidence that where lead levels of soil, water and/or biota are elevated as a result of the degradation of lead from ammunition, there is likely to be uptake of lead by certain invertebrate and vertebrate animals, with higher tissue lead concentrations in animals from contaminated than control sites (Ma 1989, Stansley and Roscoe 1996, Vyas *et al.* 2000, Hui 2002, Migliorini *et al.* 2004, Labare *et al.* 2004, Heier *et al.* 2009, Bianchi *et al.* 2011). Few studies have been conducted in the UK, but Sneddon *et al.* (2009) found that tissues of earthworms (washed and retained until their bowel was empty before assaying) from a shooting woodland in Cheshire were significantly higher in lead (111.79 mg/kg) than in those from the control woodland (5.49 mg/kg). Mixed washed and unwashed small mammal hair showed no significant variations in lead levels between these sites.

ESTIMATED IMPACTS OF LEAD FROM AMMUNITION ON WILD BIRDS AND OTHER WILDLIFE IN THE UK

ESTIMATED ANNUAL MORTALITY IN WILDFOWL AND TERRESTRIAL GAMEBIRDS IN THE UK FOLLOWING DIRECT GUNSHOT INGESTION (EXPOSURE ROUTE 1)

The physiological effects of lead in wild birds and pathways by which ammunition-derived lead reaches them are described in foregoing sections. Here we estimate, broadly, the numbers of wildfowl and terrestrial gamebirds in the UK likely to suffer morbidity and welfare effects and to die from poisoning by ammunition-derived lead.

WILDFOWL

Data are sufficient to allow us to make rough estimates of annual mortality in wintering wildfowl in the UK, although with relatively low precision.

To do this, we used the average proportions of birds with ingested gunshot provided in Table 3 for the UK, and only estimated mortality for the species with data presented in this Table. The incidence of gunshot ingestion in swans cannot be estimated from hunter-shot birds because they are protected species, but data do exist for found-dead swans. To estimate an expected value for hunter-shot swans, we used data for all of the non-swan species (Table 3), and calculated the average percentages of hunter-shot and found-dead birds with ingested gunshot. For hunter shot birds this was 3.2% (155 of 4,857 birds) and for found-dead birds was 5.2% (59 of 1,129 birds). We then used the ratio of these (3.2/5.2; 0.62) to estimate what might reasonably be expected as the incidence of ingested gunshot in swans, had they been 'hunter-shot' (this was 3% for mute swan, 8.1% for Bewick's swan and 14.6% for whooper swan – Table 3).

British wintering population estimates for the species in Table 3 were taken from Musgrove *et al.* (2011), *i.e.* 2,356,100 birds. By multiplying the incidence of ingestion by species population sizes we estimate that 82,313 birds (3.5%) would have ingested gunshot at any one time, assuming that proportions are similar to those given for hunter-shot birds in Table 3. We used the method of Bellrose (1959) to estimate mortality from the incidence of ingested gunshot. We assumed the proportions of birds with different numbers of ingested gunshot (*i.e.*, 1, 2, 3 *etc.*) to be similar to that reported by Mudge (1983) in the UK. Mudge reported numbers of gunshot ingested by 12 of the 16 species in Table 3, and we have averaged these for our calculation, *i.e.* 54% of those birds with ingested gunshot had just one gunshot, 15% had 2 gunshot and so on (Table 5). We adjusted the proportions of birds with each number of ingested gunshot using Bellrose's estimates of hunting bias, because birds that have ingested lead gunshot are more likely to be shot by hunters, presumably due to their weakened state. We used the same hunting bias corrections as Bellrose, based upon his experimental work on mallard (Table 5). We also used Bellrose's method to correct for the effects of turnover. Bellrose

Table 5: Estimate of numbers of 16 species of wildfowl listed in Table 3 dying of lead gunshot ingestion annually during winter.

Number of gunshot ingested	% hunter-shot birds with ingested gunshot[1]	Hunting bias correction[2]	% with ingested gunshot after correction for hunting bias	% with ingested gunshot corrected for turnover[3]	Additional mortality rate (annual probability of death)[4]	% of the population estimated as dying of lead poisoning[5]	Number of birds estimated as dying[6]
1	1.89	1.5	1.26	9.45	0.09	0.85	20,039
2	0.525	1.9	0.276	2.07	0.23	0.48	11,230
3	0.081	2	0.041	0.30	0.3	0.09	2,147
4	0.207	2.1	0.098	0.74	0.36	0.27	6,255
5	0.207	2.2	0.094	0.70	0.43	0.30	7,132
6 or more	0.578	2.35	0.246	1.84	0.62	1.14	26,947
Totals	**3.487**		**2.015**	**15.11**		**3.13**	**73,750**

[1] Assuming incidences from Mudge (1983) for 12 of the 16 species in Table 3; [2] Correction factor based upon the increased likelihood of hunters to shoot wildfowl that have ingested lead gunshot (Bellrose 1959); [3] Assuming a 150 day hunting season (Britain) and an average 20 day residence time of gunshot in the gizzard – turnover of 150/20 = 7.5 (see Bellrose 1959); [4] Mortality level is the increase in mortality in mallard caused by ingestion of set numbers of lead gunshot (see Bellrose 1959) – we assume that the mortality level would be similar in all species; [5] % with ingested gunshot corrected for hunting bias and turnover multiplied by mortality level; [6] Using wintering wildfowl estimates from Musgrove *et al.* (2011).

took the average retention time of gunshot in the gizzards of mallard from experimental studies to be 20 days. He then divided the length of the hunting season by this 20 day period to give a turnover correction factor to account for the numbers of birds ingesting gunshot throughout the season. In the UK the wildfowl hunting season is at least 153 days (1 Sept until end of January inland – longer below the high water mark in all but Northern Ireland). We therefore used a correction factor of 7.5 (150/20) to account for turnover. By analysing ring recoveries, Bellrose calculated the absolute difference in the annual mortality rate of wild mallards in the USA between ringed ducks dosed experimentally with various numbers of gunshot and control ducks that were ringed but not given gunshot. This difference was for the year immediately following ringing and dosing (detailed in Bellrose 1959, Table 27 and pages 274-276). We assumed that these additional annual mortality rates would be broadly similar for all wildfowl. From these calculations, presented in Table 5, we estimate that 73,750 birds of the 16 species presented in Table 3 might die every winter in Britain from lead poisoning following gunshot ingestion (this figure would be slightly higher for the UK at c. 75,000, using data from Musgrove *et al.* 2013).

This may underestimate mortality for several reasons. It does not include species of wildfowl for which UK data on the incidence of gunshot ingestion is not available (*e.g.* some of the goose species), and does not include mortality caused by gunshot ingested in the UK outside of the hunting season (which will occur but likely with a reduced incidence). It also excludes the sub-lethal effects of lead which can also influence mortality. These three factors would result in our estimate of mortality being too low. A few factors could potentially result in our estimate being too high. We assume that mortality levels given ingestion of a specific number of gunshot will be similar in all species to those used by Bellrose (1959) for mallard, while these may be higher in some species and lower in others. It is possible that mortality levels could be lower in the geese and swans ingesting small numbers of gunshot

(as they have larger body size), although they can ingest very large numbers of gunshot (Newth *et al.* 2012). As lead poisoning was the diagnosed cause of death in a quarter of migratory swans found dead (Newth *et al.* 2012), overestimation in these species seems unlikely. Had there been widespread compliance with regulations banning the use of lead gunshot for shooting wildfowl (and over certain wetlands) in England, this estimate, based upon data from before the ban, might overestimate the numbers affected currently. However, compliance with English regulations banning the use of lead gunshot for shooting wildfowl has been shown to be low (Cromie *et al.* 2010, 2015) with some 70% or more of ducks shot in England and purchased through game outlets being illegally shot with lead ammunition. Hence, legal and illegal deposition of lead gunshot in wetland and terrestrial wildfowl feeding habitats is likely to have continued at broadly similar levels to the period before the ban. Similarly, Newth *et al.* (2012) found that the proportion of birds dying from lead poisoning in England did not change significantly after the introduction of legislation. For this reason the estimate of number affected is likely to be approximately correct.

While there are various assumptions and uncertainties in this calculation for wildfowl, we suggest that the true value is likely to be in the high tens of thousands and probably lie within the range 50,000-100,000 individuals. More precise estimates cannot readily be made at this time.

Many more birds are likely to suffer welfare effects from lead ingestion than die. If all wildfowl predicted to ingest gunshot suffer welfare effects, this would result in about 15% (Table 5) of birds, *i.e.* c. 353,000 suffering welfare effects every winter (and more throughout the year). We therefore estimate that 74,000 – 353,000 individual wildfowl suffer welfare effects every winter.

While it is possible to broadly estimate mortality from lead poisoning, determining impacts at a population level is not straightforward. This is especially the case for wildfowl in the UK, as the majority are migratory and thus subject to pressures across their ranges. The only reasonably robust way of doing this is to model and compare alternative population trajectories for a species based upon demographic rates estimated when effects of ammunition-derived lead are present and absent. The long-term and complicated nature of collecting such information means that for most species, an accurate assessment of the extent of mortality, and possible population level effects from lead ingestion, whatever the source, is currently not possible for most species.

When detailed information on demographic rates is not available, it is legitimate to adopt a comparative approach to the detection of effects of external drivers on population trends (Green 1995). This involves comparing population trends across species or populations with differing levels of exposure to ammunition-derived lead. A negative correlation between population trend and exposure may be suggestive of population-level effects. At a European level, Mateo (2009) correlated population trends in a set of 15 taxonomically similar European wildfowl species with broadly comparable life-history characteristics with reported prevalence levels of shot ingestion. There was a statistically significant tendency for species with high levels of shot ingestion to have more negative population trends than species with low shot ingestion levels. As was pointed out by Mateo, correlation is not causation and effects of some unidentified factor might have led to a spurious correlation. Nonetheless, this analysis is suggestive of an effect of lead contamination on population trend and indicates that it is worth looking further at the effects of lead, especially for species with high shot ingestion levels.

TERRESTRIAL GAMEBIRDS

Less information is available for the UK on levels of gunshot ingestion by terrestrial birds than by wildfowl and it would not be appropriate to extrapolate levels of mortality in terrestrial gamebirds from the studies on wildfowl.

However, data on the proportion of terrestrial birds with ingested gunshot are available for several species in the UK, *i.e.* hunter-shot red-legged partridge (1.4%, Butler 2005[2]), hunter-shot pheasant (3%, Butler *et al.* 2005) and grey partridge found dead (4.5% average for adults and juveniles, Potts 2005), so order of magnitude estimates of mortality can be made. To do this we took breeding population estimates (from Musgrove *et al.* 2013) of these species and the other most numerous gamebirds (red grouse) potentially susceptible to gunshot ingestion and added the numbers of pheasant and partridge raised in captivity which are subsequently released for shooting each year (*i.e.* 35 million pheasants and 6.5 million partridges – released in 2004 (PACEC 2006)). To obtain numbers of individuals from Musgrove *et al.*'s (2013) estimates, we doubled the numbers of territories of red-legged and grey partridges. For pheasant, we assumed that the ratio of males to females was 1:4.6 (after the shooting season (Cramp and Simmons 1980)). We ignored the many young wild-bred birds hatched in the previous breeding season that are

present during the shooting season because good estimates of the immature population were not readily available. Our estimate of numbers of terrestrial gamebirds birds that may potentially ingest gunshot is therefore an underestimate. We also omitted other potentially susceptible game species. We assumed that hunter-shot grey and red-legged partridges would have similar levels of gunshot ingestion (1.4%) because grey partridges found dead would be expected to have higher levels of gunshot ingestion if some had died of lead poisoning, and we assumed a low 1% level of gunshot ingestion in red grouse and used the 3% reported for pheasant. We then assumed that shooters are twice as likely to kill birds that have ingested lead gunshot (due to their weakened state) than to kill birds that had not ingested gunshot, and corrected for this (this is the correction factor for mallards that have ingested 3 shot - see Table 5 and Bellrose 1959). We then calculated the number of birds in the population likely to have ingested gunshot at any one time (c. 615,000). Given that we only have estimates for the proportion of gamebirds with ingested gunshot at the time they were killed, and gunshot has a residence time in the alimentary tract that rarely exceeds 30 days (on average about 20 in wildfowl (Bellrose 1959)), the number of birds likely to ingest gunshot at some time during the winter shooting season will be several times higher than this. All birds that ingest lead gunshot may suffer some welfare effect, and a proportion of them, perhaps of the order of hundreds of thousands, are likely to die from lead poisoning. We do not think that it is valid to give more precise estimates for terrestrial birds as studies of hunting bias and shot residence times in the intestine have not been conducted, and fewer studies are available on levels of shot ingestion.

EFFECTS ON PREDATORY AND SCAVENGING BIRDS AND OTHER WILDLIFE FOLLOWING INGESTION OF AMMUNITION-DERIVED LEAD IN THE TISSUES OF DEAD OR LIVE GAME SPECIES (EXPOSURE ROUTE 2)

Measurements of lead concentrations in tissue samples from carcasses of dead predatory and scavenging birds have been used, together with *post mortem* examinations, to assign the cause of death to lead poisoning and other causes. Such studies in the USA, Canada and Europe reported proportions of deaths caused by lead in species likely to be at risk of ingesting ammunition-derived lead ranging from 3% of deaths to 35% of deaths (Elliott *et al.* 1992, Wayland and Bollinger 1999, Wayland *et al.* 1999, Clark and Scheuhammer 2003, Finkelstein *et al.* 2012, Rideout *et al.* 2012). In Europe the bird species with the most consistently high proportions of deaths attributed to lead poisoning is the white-tailed eagle (14 – 28% of deaths attributed to effects of lead) (Elliott *et al.* 1992, Kenntner *et al.* 2001, Krone *et al.* 2006, Helander *et al.* 2009).

In the UK, Pain *et al.* (2007) reported lead concentrations from tissue samples from carcasses of 44 red kites found dead or that were captured sick and died subsequently in England between 1995 and 2003. Elevated liver lead concentrations (>15 mg/kg dw in these birds)[3] and *post mortem* examination analyses indicated that four (9%) of the birds had probably died from lead poisoning; several others had elevated liver lead but were diagnosed as dying of other causes. Walker *et al.* (2012, 2013) reported liver lead concentrations for another sample of 38 carcasses of red kites collected in England in 2010 and 2011 and found no cases with elevated liver lead concentrations.

Pain *et al.* (1995) reported lead concentrations from the livers of 424 individuals of 16 raptor species found dead in Britain and sent for analysis to the Institute of Terrestrial Ecology, Monks Wood, from the early 1980s to the early 1990s. There were eight species for which ten or more carcasses were analysed: short-eared owl *Asio flammeus*, buzzard, little owl *Athene noctua*, kestrel *Falco tinnunculus*, sparrowhawk, peregrine falcon, merlin *Falco columbarius* and long-eared owl *Asio otus*. The other eight species with fewer than ten carcasses included three of the species most likely on the grounds of diet to consume carrion contaminated with ammunition-derived lead (red kite (6 carcasses), golden eagle (5), white-tailed eagle (1)), and one species especially likely to prey upon waterfowl with shot-in or ingested shotgun pellet-derived lead in their tissues (western marsh harrier (1)). Of the species with 10 or more carcasses, feeding ecology would suggest that peregrine falcon and buzzard would be susceptible to preying upon or scavenging (in the case of buzzards) game species. Elevated lead concentrations in liver (>20 mg/kg dw)[3], within the range associated with lead poisoning mortality in raptors, were recorded in one peregrine falcon (4% of species sample)

[2] Earlier data for red-legged partridges (1933-1992) were excluded as Butler (2005) considered it possible that cases of lead ingestion were missed by the pathologists and considered it unlikely that a detailed search was part of all *post mortem* examinations, particularly when no clinical signs of lead poisoning were evident.

[3] A review by Franson and Pain (2011) suggested that birds with no history of lead poisoning usually have liver lead concentrations of <2 mg/kg wet weight (c. 6.3ppm dry weight) and frequently of <1 mg/kg ww (c. 3.1 ppm dw). In falconiformes, these authors suggested a liver lead range for sub-clinical poisoning of 2<6 ppm ww [6.3-18.6 ppm dw] with clinical poisoning associated with liver lead concentrations exceeding >6ppm ww. 'Elevated' liver lead could be considered as above background, *i.e.* 6.3 ppm dw with clinical poisoning occurring at levels above approximately 18.6 ppm dw. These figures can vary somewhat as there is no absolute wet weight to dry weight conversion factor for bird livers (1ppm ww was converted to 3.1 ppm dw by Franson and Pain (2011)).

and one buzzard (2% of species sample). Another one each of these species had liver concentrations of 15-20 mg/kg dw. No individuals of any other species had >15 mg/kg dw, although some had elevated liver lead concentrations in the range of 6-15 mg/kg dw.

Walker *et al.* (2012, 2013) reported liver lead concentrations for a sample of 30 carcasses of sparrowhawks collected in Britain in 2010 and 30 in 2011. Although one sample had a lead concentration of 12.6 mg/kg dw which is close to the threshold for clinical effects, the concentrations in all of the others were <2 mg/kg. It is unlikely that sparrowhawks will be frequently exposed to lead gunshot in their prey; it is possible that occasional exposure may occur in large females that could feed on pigeons that have been shot and wounded but survive.

While some data are available as described above, the necessary measurements of tissue lead concentration have not been reported from sufficient numbers of carcasses of several species potentially at risk to draw any reliable conclusions about the proportion of predatory and scavenging birds dying from lead poisoning in the UK. In particular, sufficient observations are lacking for white-tailed eagle, golden eagle and western marsh harrier. It should also be noted that the geographical distribution within the UK of the locations from which carcasses of scavenging and predatory birds were collected and sent for analysis is likely to be atypical of the distribution of the species as a whole for some of the species with potentially high risks of exposure to ammunition-derived lead. In particular, the collection of carcasses of buzzard, golden eagle and white-tailed eagle from areas in which large numbers of red deer are culled and viscera discarded is probably infrequent relative to the proportion of the population of these species in such areas. Carcasses are usually collected by members of the public, and areas with high levels of culling of deer tend to be remote from human populations.

There is strong evidence that a sometimes substantial proportion of predatory and scavenging birds die from lead poisoning from studies in North America and Europe (see earlier sections of this paper). The small numbers of samples of raptor carcasses collected from largely lowland England suggest that exposure is likely in a small proportion of individuals of those species that would be predicted to be at risk from their feeding ecology. Studies on red kites show that risks may vary locally. There has been little research in the UK on some of the potentially most at risk species (*e.g.* white-tailed and golden eagles, and marsh harriers) and in those areas (*e.g.* upland deer shooting areas and coastal areas) where the risks are likely to be most significant. However, source, pathway, receptor links clearly exists for these species and further research is required.

Few studies have been conducted on the possible impacts of ammunition derived lead in carnivorous mammals, but those that have show little evidence for direct poisoning. Rogers *et al.* (2012) reported that blood lead levels of grizzly bears *Ursus arctos* in the Greater Yellowstone Ecosystem, USA, were not appreciably higher during the hunting season, despite the presence of carcasses and discarded viscera of deer during the hunting season. In addition, they found that lead concentrations in blood and tissues of wolves *Canis lupus* and mountain lions *Puma concolor* in the region were low. Hence, in this region there was no evidence that ingestion of lead from hunter-killed carcasses or viscera was leading to the absorption of lead by these mammalian carnivores. Similarly, Millán *et al.* (2008) found relatively low levels of lead in liver, muscle and bone in five species of carnivorous mammals in Spain.

EFFECTS OF AMMUNITION DERIVED LEAD ON WILDLIFE FOLLOWING INGESTION OF LEAD CONTAMINATED SOIL, WATER AND BIOTA (EXPOSURE ROUTE 3)

There appear to be substantial inter-specific differences in the tolerance of invertebrates to lead of ammunition origin in soils and water. At a cast-off shooting range in Finland, Rantalainen *et al.* (2006) found microbes and enchytraeid worms to be negatively affected by the contamination while soil-dwelling nematodes and microarthropods appeared unaffected. Migliorini *et al.* (2004) found the abundance of Collembola, Protura and Diplura to be positively correlated with major detected contaminants (lead and antimony) in soils from a clay pigeon shooting range, while Symphyla showed a negative correlation with these pollutants. Concentrations of lead in the saprophagous *Armadillidium sordidum* (Isopoda) and the predatory *Ocypus olens* (Coleoptera) increased with the soluble lead fraction in soil, showing that a significant portion of metallic lead from spent pellets is bioavailable in the soil and can be bioaccumulated by soil organisms. Reid and Watson (2005) found soil levels of 6,410 +/- 2,250 and 296 +/- 98 mg(Pb)/kg dw, respectively at a clay-pigeon shooting site soil and an un-shot control site. At 6.1 +/- 1.2 mg(Pb)/g dw, shooting site body burdens of earthworms *Aporrectodea rosea* were almost 1,000 times higher than those from the control site

(7.1 +/- 9.0 µg(Pb)/g dw). An experiment in which earthworms collected from both sites were exposed to soil that had been artificially augmented with lead found a decrease in condition of earthworms from the control site, but not of those from the shooting site, suggesting the development of high tolerance to lead in the shooting site worms.

Exposure to lead from ammunition sources in areas of high shooting intensity has been reported to have impacts on small mammals and amphibians. White-footed mice *Peromyscus leucopus* and green frogs *Rana clamitans* sampled within the shot-fall area of a shooting range with high pellet density had depressed ALAD enzyme levels (Stansley and Roscoe 1996), a recognised indicator of sub-clinical lead toxicosis in mammals, and the mice also had reduced haemoglobin levels. Stansley *et al.* (1997) exposed eggs of pickerel frogs *Rana palustris* and bullfrogs *R. catesbeiana* to 0, 25, 50, 75 and 100% lead-contaminated surface water from a trap and skeet range. Total lead concentrations in 100% range water treatments varied from 840–3,150 µg/l, with the filterable form accounting for approximately 4–5% of the total. Hatching was not affected in either species but there was highly significant mortality (100% and 98%) in pickerel frog tadpoles after 10 days of exposure to 100 and 75% range water; mortality was not significantly increased in bullfrogs.

It has been shown experimentally that pigeons *Columbia livia* dosed with soil contaminated with lead from a shooting range absorbed lead in a dose-response manner as reflected in blood, tissues, feathers and erythrocyte protoporphyrin, a biomarker of lead effect (Bannon *et al.* 2011). In the field, Vyas *et al.* (2000) found elevated erythrocyte protoporphyrin levels in some ground foraging passerines held in aviaries in the vicinity of a clay pigeon shoot in Maryland, USA, relative to controls. The authors could not determine whether this was from ingestion of one or a combination of shot directly, degraded shot in soil (soil can be an important routes of exposure to lead in some bird species and situations (Beyer *et al*. 1998)) or other lead-contaminated dietary components. A case of lead poisoning has also been described in a grey squirrel *Sciurus carolinensis* in the vicinity of a law enforcement firing range in Georgia, USA (Lewis *et al.* 2001).

These studies, and those cited in preceding sections, show that where invertebrate and vertebrate animals are exposed to elevated levels of lead of ammunition origin, irrespective of the exposure route (*i.e.* ammunition fragments, soil, water or biota) it can exert sub-lethal negative effects on animal physiology (*i.e.* both welfare and individual survival) in many species, and in some animals may cause mortality. Effects are related to exposure levels and amounts absorbed, thus animals (*e.g.* birds) ingesting ammunition fragments directly are at particularly high risk as described in preceding sections. Nonetheless, local effects on a range of wildlife in areas of intensive ammunition use appear likely in many exposed species. While some inter-specific differences in susceptibility to the effects of lead occur across many taxa, the few studies available suggest that this may particularly be the case in invertebrates, with the possibility that this may be acquired (for one species studied). Insufficient data exist to be able to evaluate numbers of animals potentially affected *via* routes other than direct ingestion of ammunition fragments by birds.

CONCLUSIONS

The toxic effects of lead on humans and other vertebrates have long been known and most uses of lead causing elevated exposure to humans and wildlife have been phased out or heavily regulated across most of the world (Stroud 2015). Lead derived from ammunition now appears to be the most significant geographically widespread and common source of unregulated environmental lead contamination to which wildlife is exposed. Lead from ammunition has primarily been studied in birds, with the two main exposure pathways being direct ingestion of spent gunshot (*e.g.* by wildfowl and terrestrial gamebirds), and ingestion by predators and scavengers of lead gunshot, bullets, or fragments from these, in the flesh of their prey. Thousands of tonnes of lead ammunition, primarily gunshot, is deposited and accumulates in the UK environment every year. Lead ammunition degrades very slowly, and while deposited gunshot settles through soils and sediments it may take several decades to become unavailable to feeding animals. Predators and scavengers can be exposed to lead in dead and unretrieved game, discarded viscera from shot deer, and in the flesh of prey that have been wounded but survived. Studies on a variety of species/populations of live wildfowl have shown that a high proportion individuals (an average of >20% across 22 species) carry gunshot in their flesh.

Studies of exposure to, and poisoning by, lead from ammunition in birds have included: experimental dosing studies, *post mortem* examinations of birds, X-radiography studies of live birds for incidence of ingested ammunition or fragments, examination of regurgitated pellets for ammunition or ammunition fragments,

investigations of temporal and spatial relationships between ammunition ingestion/poisoning and shooting seasons or intensity, isotopic studies to investigate the source of elevated tissue lead concentrations; studies of changes in survival in wild birds with different levels of gunshot ingestion, and others. These studies have been conducted in many countries across the world, primarily over the last 65 years and have shown that lead poisoning from ammunition sources is geographically widespread and causes substantial suffering and mortality in many avian taxa.

Lead from ammunition is known to affect a wide range of biological and physiological systems in birds and other vertebrates, and birds can die rapidly after ingesting lead from ammunition (acute poisoning), or gradually following lower levels of exposure or absorption, or repeated exposure (chronic poisoning). Lead poisoning from ammunition lead was first recorded in the UK well over a century ago (Calvert 1876) and reports of lead poisoned birds grew rapidly from the 1950s onwards in the UK and globally. Wildfowl are the best studied taxa, but where lead ingestion has been investigated in other taxa that feed in shot-over areas, including terrestrial gamebirds, it has generally been found. Lead poisoning in predatory and scavenging birds, primarily raptors, has also been widely reported, although relatively few studies have been conducted in the UK.

In the UK, we can broadly estimate the numbers of birds from certain avian taxa that are likely to die as a direct result of ingesting lead gunshot every winter. These estimates are based upon published gunshot ingestion incidence in different species, and corrected for hunting bias (*i.e.* that hunters are more likely to shoot lead poisoned birds), turnover of gunshot in the alimentary canal, and increases in mortality as a result of ingesting different numbers of gunshot. These estimates suggest that 50,000-100,000 wildfowl are likely to die each winter (*i.e.* during the shooting season) as a direct result of lead poisoning. Wildfowl that die outside of the shooting season will be additional, as will birds dying from the indirect results of lead poisoning. Several hundred thousand wildfowl may suffer welfare effects.

Estimates of mortality for terrestrial gamebirds in the UK are likely to be less accurate and precise due to fewer studies, but we estimate that about 600,000 terrestrial gamebirds are likely to have ingested gunshot at any one time and many times more throughout the shooting season. All birds that ingest lead gunshot may suffer some welfare effect, and a proportion of them, perhaps of the order of hundreds of thousands, are likely to die from lead poisoning each year.

There is strong evidence from studies in North America and elsewhere that a sometimes substantial proportion of predatory and scavenging birds die also from lead poisoning. A few studies from the UK have reported lead poisoning in certain raptor species, and the source and pathways exists for a wider range of species to be affected, but further research on this is needed.

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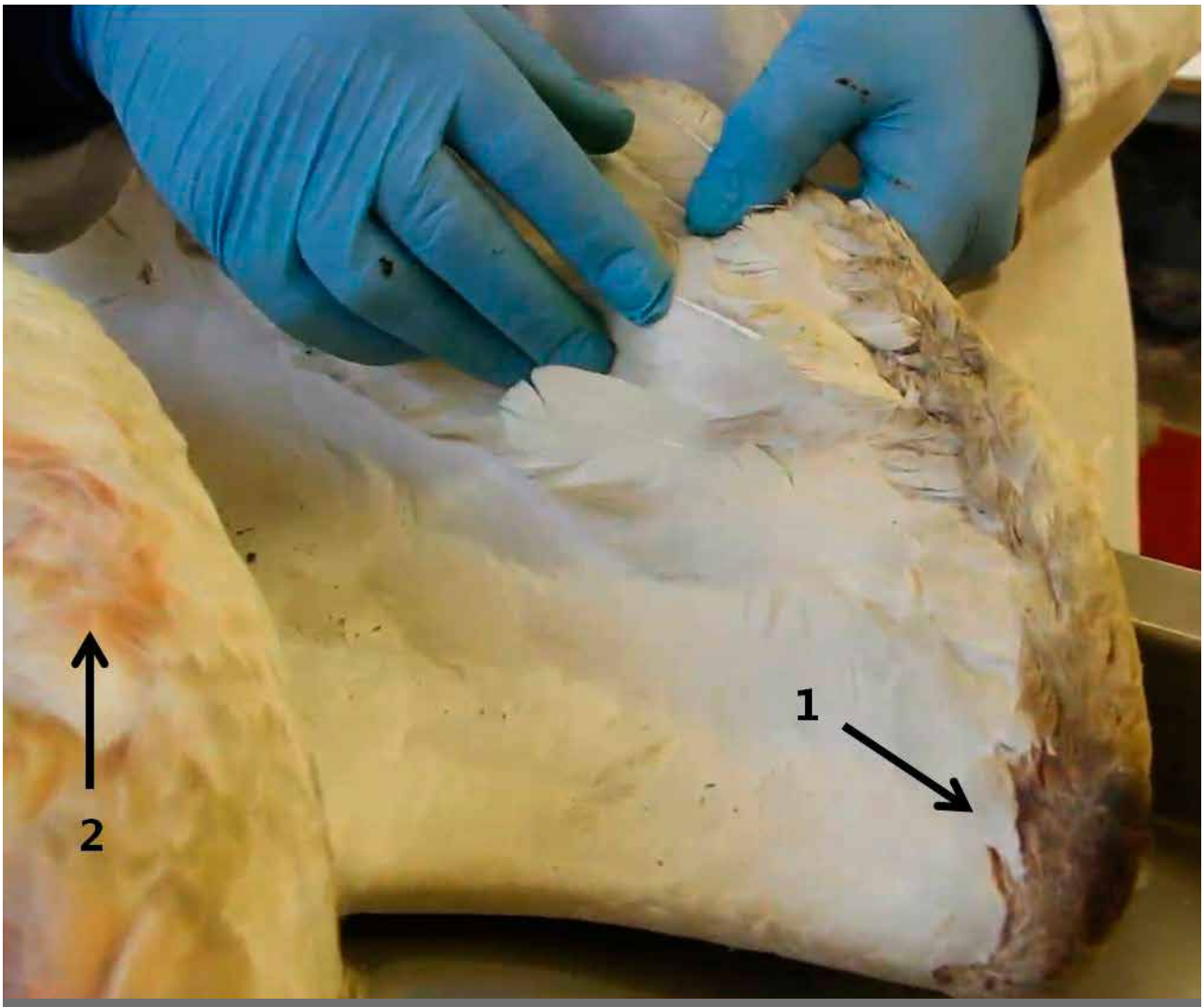

Muddy and bloody carpel joint of a lead poisoned whooper swan *Cygnus cygnus* (1): due to paralysis of the bird's legs it had been using its wings to propel itself on land prior to death. The blood staining on the bird's breast (2) illustrates that the abrasions have been bleeding.

Photo Credit: WWT