# Anthropogenic lead (Pb) exposure in populations of a wild parrot (kea *Nestor notabilis*)

Clio Reid[1], Kate McInnes[1,*], Jennifer M. McLelland[2] and Brett D. Gartrell[2]

[1]Research and Development Group, Department of Conservation, PO Box 10420, Wellington 6143, New Zealand
[2]New Zealand Wildlife Health Centre, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Private Bag 11222, Palmerston North 4442, New Zealand
[*]Author for correspondence (Email: kmcinnes@doc.govt.nz)

Published on-line: 9 December 2011

**Abstract:** Kea (*Nestor notabilis*), large parrots endemic to hill country areas of the South Island, New Zealand, are subject to anthropogenic lead (Pb) exposure in their environment. Between April 2006 and June 2009 kea were captured in various parts of their range and samples of their blood were taken for blood lead analysis. All kea ($n$ = 88) had been exposed to lead, with a range in blood lead concentrations of 0.014 – 16.55 μmol $L^{-1}$ (mean ± SE, 1.11 ± 0.220 μmol $L^{-1}$). A retrospective analysis of necropsy reports from 30 kea was also carried out. Of these, tissue lead levels were available for 20 birds, and 11 of those had liver and/or kidney lead levels reported to cause lead poisoning in other avian species. Blood lead levels for kea sampled in populated areas (with permanent human settlements) were significantly higher ($P < 0.001$) than those in remote areas. Sixty-four percent of kea sampled in populated areas had elevated blood lead levels (> 0.97 μmol $L^{-1}$, the level suggestive of lead poisoning in parrots), and 22% had levels > 1.93 μmol $L^{-1}$ – the level diagnostic of lead poisoning in parrots. No kea from remote areas had levels > 0.97 μmol $L^{-1}$. The kea is a long-lived, slow-reproducing species at a high risk of decline from even a small reduction in its survival rate. Based on our findings, we conclude there is an urgent need to implement lead abatement strategies in areas of the kea range that overlap with permanent human settlement.

**Keywords:** conservation; free-ranging; plumbism; Psittacidae; toxins

## Introduction

Kea (*Nestor notabilis*) are large parrots endemic to hill country areas of the South Island, New Zealand. They are noted for their highly intelligent, curious, and bold nature, which has repeatedly brought them into conflict with humans and endangered their survival (Diamond & Bond 1999). Kea's neophilic tendencies bring them into contact with many anthropogenic hazards, such as ingestion of foreign objects (e.g. pieces of closed cell foam and rubber) that can cause impactions of the crop and intestines, resulting in death (Brejaart 1994; Peat 1995; Jarrett 1998); drowning in containers such as water tanks (Jackson 1969; Elliott & Kemp 1999); cyanide (Peat 1995); chocolate (Gartrell & Reid 2007); and lead (Pb) (Jarrett 1998; McLelland et al. 2010). Although lead poisoning has been established as the cause of death of several wild kea within the past 15 years, lead exposure was recently discovered to be common in a single population of wild kea near an alpine village at Aoraki/Mt Cook National Park (McLelland et al. 2010). In that study, 38 wild kea were found to have been exposed to lead, and 26 of those birds had elevated (> 0.97 μmol $L^{-1}$) lead levels in their blood. The exposure of kea to lead in other parts of their range is to date unknown and such information is needed to prioritise the extent of lead abatement strategies.

The effects of lead on waterfowl, shorebirds, and raptors have been widely covered in the scientific literature, and are well known in captive parrots (Dumonceaux & Harrison 1994), but there are only a handful of reports of its occurrence in wild parrots. Kea are not the only wild New Zealand parrots affected by lead exposure. Several anecdotal cases have been reported of lead poisoning in wild kākā (*Nestor meridionalis*) (BDG, unpubl. data), and one case in a wild kākāpō (*Strigops habroptilus*) (J. Potter, Auckland Zoo, pers. comm.). In Esperance, Western Australia, exposure to lead carbonate from inappropriately transported lead dust resulted in mass bird deaths in 2006 and 2007, including of white-tailed black cockatoos (*Calyptorhynchus baudinii*) and purple-crowned lorikeets (*Glossopsitta porphyrocephala*) (Golder Associates 2008; Gulsen et al. 2009).

Lead is a highly toxic heavy metal that adversely affects the nervous, renal, gastrointestinal, and reproductive systems, and the biosynthesis of haeme (Verity 1997; Pattee & Pain 2003). Lead has also been shown to have teratogenic effects (i.e. it interferes with normal embryonic development resulting in abnormalities) and to cause death in bird embryos, which are sensitive even to relatively low doses of lead (Kertész et al. 2006). Lead has also been shown to cause a reduction in bone mineralisation (Gangoso et al. 2009), which could mean an increase in bone fragility (Fleming et al. 2000; Whitehead & Fleming 2000). Lead has been recorded to have effects on the peripheral and central nervous system even at very low exposures, and has been associated with defects in development, cognition and behaviour in both humans (Toscano & Guilarte 2005) and birds (Dey et al. 2000; Burger & Gochfeld 2005).

Lead poisoning may be acute or chronic with clinical signs dependent on the amount and surface area of the lead ingested (Platt 2006). Clinical signs in birds include behavioural changes, lethargy, anorexia, vomiting, diarrhoea, ataxia, limb paresis (nerve-related weakness) or paralysis, seizures, anaemia and emaciation (Platt 2006). Death may occur within 48 h of the first appearance of clinical signs (Platt 2006). Psittacines (parrots and their relatives) in particular fall victim to acute lead intoxication because of their curious nature and inclination to chew any object they may encounter (Gelis 2006).

The size of the overall kea population in the wild is unknown,

the most accurate estimate to date being approximately 3000 (Diamond & Bond 1999). Kea are currently listed by the Department of Conservation (DOC) as naturally uncommon (Miskelly et al. 2008) and by the IUCN as vulnerable and decreasing (BirdLife International 2008). Recent population surveys have indicated that kea numbers in the Nelson Lakes area have significantly declined since the 1990s (T. Orr-Walker, Kea Conservation Trust, pers. comm.). Kea numbers in Nelson Lakes in the 1990s indicated the population there was stable with high nesting success and adult survival rates (Elliott & Kemp 1999). An average of 10 fledglings per year were produced in an area of approximately 6000 ha (Elliott & Kemp 1999), whereas a production rate of two fledglings per year was observed in 2009 and 2010 over an area of 11 000–13 000 ha (T. Orr-Walker, Kea Conservation Trust, pers. comm.).

The aim of this study was to extend the findings of McLelland et al. (2010) and survey the extent of lead exposure in wild kea throughout their range, in both remote areas and those with permanent human settlements. We hypothesised that kea in areas that featured permanent human settlements would have significantly higher exposure to lead. By including the data from McLelland et al.'s study, and expanding on it with a further 50 samples from kea in different locations from throughout their geographical range, we have been able to provide a comparison of lead exposure in different habitats and highlight the problem of lead toxicity to the species as a whole. This study therefore makes a novel contribution to knowledge of anthropogenic hazards facing kea, and the field of wildlife health overall.

## Methods

A total of 88 kea were successfully sampled between April 2006 and June 2009. This paper includes blood lead results ($n = 38$) from kea at Aoraki/Mt Cook National Park and post-mortem tissue lead levels ($n = 6$) from various sites previously reported in McLelland et al. (2010). Blood samples from a further 50 kea and post-mortem tissue lead levels from a further 14 kea are included in this paper. The seven sampling sites throughout the South Island, New Zealand, included two areas with permanent human settlement – the Fox Glacier – Franz Josef area (43°S, 170°E) and Aoraki/Mt Cook National Park (43°S, 170°E) – and five remote areas – Golden Bay (40°S, 173°E), Hohonu Range, West Coast (42°S, 171°E), Arawhata Valley, Mt Aspiring National Park (44°S, 169°E), Rob Roy Valley, Mt Aspiring National Park (44°S, 168°E) and Treble Cone ski field (44°S, 168°E) (Table 1; Fig. 1). We classify Treble Cone ski field as remote because it is not a permanent human settlement. Furthermore, the buildings onsite were constructed relatively recently, and consequently have little (if any) lead building materials. Lead building materials appear to be the primary source of anthropogenic lead in the kea's environment. Kea presence in the area is limited to winter months, and the kea that visit the area are likely to live most of the year in Mt Aspiring National Park, which we classify as a remote area. The sample size, sex, and age of kea sampled are shown in Table 2. Kea were caught in 2006 at Aoraki/Mt Cook for a previous study (McLelland et al. 2010) and at Aoraki/Mt Cook and Mt Aspiring National Park in conjunction with another study (Reid 2008). Birds were caught at Treble Cone ski field

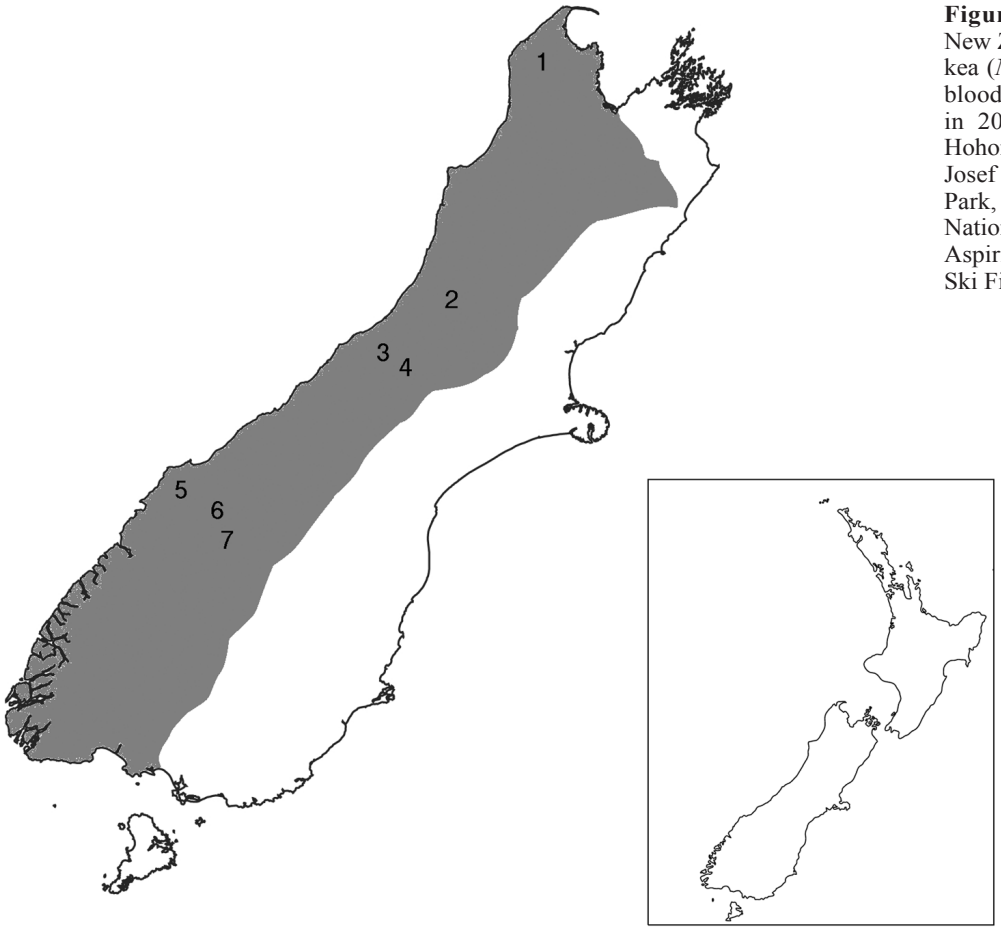


**Figure 1.** Map of the South Island, New Zealand, showing the locations where kea (*Nestor notabilis*) were captured and blood-sampled for blood lead analysis in 2006–2009 (1 = Golden Bay, 2 = Hohonu Range, 3 = Fox Glacier – Franz Josef area, 4 = Aoraki/Mt Cook National Park, 5 = Arawhata Valley, Mt Aspiring National Park, 6 = Rob Roy Valley, Mt Aspiring National Park, 7 = Treble Cone Ski Field).

**Table 1.** Areas of the South Island, New Zealand, where kea (*Nestor notabilis*) were blood-sampled for blood lead analysis.

| Area | Area type | *n* | Females | Males | Unknown |
|---|---|---|---|---|---|
| Golden Bay | Remote | 1 | ? | ? | 1 |
| Hohonu Range (West Coast) | Remote | 2 | 0 | 2 | 0 |
| Fox – Franz Josef area | Populated | 13 | 2 | 10 | 1 |
| Aoraki/Mt Cook National Park | Populated | 42 | 5 | 37 | 0 |
| Arawhata Valley (Mt Aspiring National Park) | Remote | 6 | 2 | 4 | 0 |
| Rob Roy Valley (Mt Aspiring National Park) | Remote | 19 | 2 | 17 | 0 |
| Treble Cone Ski Field | Remote | 5 | 1 | 4 | 0 |

**Table 2.** Blood lead (Pb) levels for the five age groups of kea (*Nestor notabilis*) sampled from remote and populated areas of the South Island, New Zealand. Mean blood lead level (μmol $L^{-1}$)* ± SE.

| Age group | Area type | *n* | Blood lead level | Range |
|---|---|---|---|---|
| Nestling | Populated | 2 | 1.81 ± 0.232 | 1.58 – 2.05 |
| | Remote | 0 | - | - |
| Fledgling | Populated | 24 | 2.01 ± 0.341 | 0.34 – 8.20 |
| | Remote | 7 | 0.10 ± 0.011 | 0.058 – 0.14 |
| Juvenile | Populated | 16 | 2.20 ± 0.974 | 0.33 – 16.55 |
| | Remote | 4 | 0.12 ± 0.027 | 0.07 – 0.20 |
| Subadult | Populated | 5 | 1.04 ± 0.272 | 0.23 – 1.78 |
| | Remote | 11 | 0.13 ± 0.027 | 0.014 – 0.31 |
| Adult | Populated | 8 | 0.19 ± 0.038 | 0.058 – 0.43 |
| | Remote | 11 | 0.12 ± 0.012 | 0.07 – 0.20 |
| All birds | Populated | 55 | 1.70 ± 0.328 | 0.058 – 16.55 |
| | Remote | 33 | 0.12 ± 0.010 | 0.014 – 0.31 |

*To convert values from μmol $L^{-1}$ to μg $dl^{-1}$, multiply by the conversion factor 20.72.

specifically for this study, and in the remaining areas were sampled opportunistically for this study by researchers and DOC staff working on other projects.

Birds were captured and blood-sampled from the left or right ulnar vein (under the wing) and/or in a few cases, the medial metatarsal vein (on the leg). Blood was placed in 0.4-ml lithium heparin microtainers (BD Biosciences, Franklin Lakes, NJ, USA). A clinical examination was carried out on birds at Aoraki/Mt Cook, Rob Roy Valley (Mt Aspiring National Park), and Treble Cone ski field. A combination of bill length, eye, cere, bill and plumage colouration, moult stage, and weight (where measured) were used to identify the age-class and sex of the unbanded kea (Bond et al. 1991; Diamond & Bond 1999). All previously unbanded birds were banded before release. Blood was analysed for lead content using a portable lead analyser (LeadCare, ESA Inc., Chelmsford, MA, USA). Blood samples were placed in buffer solution and either directly analysed for lead content (i.e. when the portable lead analyser was available for use in the field) or cooled within the time frame recommended by the manufacturer until they could be submitted to the New Zealand Wildlife Health Centre at Massey University for blood lead analysis. The analyser uses anodic stripping voltammetry to measure blood lead levels, as described in Wang (2000), and has a detection range of 0.0 – 3.14 μmol $L^{-1}$ and an analytical reporting range of 0.068 – 3.14 μmol $L^{-1}$ (ESA Biosciences Inc. 2005). Levels greater than 3.14 μmol $L^{-1}$, i.e. above the upper limit of the analyser, are expressed as 'HI'. Where this occurred, a 1:10 dilution with saline was used to give a quantitative measure. The interpretation of an elevated blood lead concentration was made using the level suggestive of lead poisoning (> 0.97 μmol $L^{-1}$) (Platt 2006). Blood lead concentrations are reported in SI units (μmol $L^{-1}$). Measurements in μg $dl^{-1}$ were converted to μmol $L^{-1}$ by multiplying by the conversion factor of 0.04826.

Data from the Huia (New Zealand wildlife pathology) and Massey University pathology databases were searched and records retrieved for all kea entered between 1991 and 2010. Records for captive kea were excluded. Pathological information from the records was examined to determine the number of kea that had died of lead poisoning. Tissue lead concentrations (liver and kidney) for kea submitted to Massey between 1991 and 2007 have already been published in McLelland et al. 2010 ($n$ = 6). Tissue lead levels for kea submitted between 2007 and 2010 have been added here ($n$ = 14) to the data already published.

### Statistical analyses

Kea were designated as being either from populated or remote areas. Populated areas were defined by the presence of one or more permanent human settlements such as villages or townships (Aoraki/Mt Cook, Fox Glacier – Franz Josef area) with older buildings known or suspected to contain lead roofing materials. Kea sampled from populated areas were considered likely to have visited nearby permanent human settlements. Remote areas were defined by the absence of permanent human settlements for a radius of at least 5 km, and based on behavioural observations and recent historical records kea sampled from these areas were considered unlikely to have visited such settlements. Differences in blood lead levels (μmol $L^{-1}$) between kea sampled from populated and remote areas were compared with a Wilcoxon's rank sum test.

Results from samples that were clotted, <50-µl volume, or were not tested within the portable lead analyser manufacturer's recommended time frame were not included in the statistical analyses. All statistical analyses were conducted using the Rcmdr (R Commander) package version 1.5-2 (Fox 2009) of R version 2.9.2 (R Development Core Team 2009).

## Results

All kea included in the analysis ($n$ = 88) had been exposed to lead (Table 2; mean ± SE, 1.11 ± 0.220 µmol $L^{-1}$; range, 0.014 – 16.55 µmol $L^{-1}$). Kea from remote areas had significantly lower (Wilcoxon rank sum; W = 1755.5, $P < 0.0001$) blood lead levels (0.12 ± 0.010 µmol $L^{-1}$) than those from populated areas (1.70 ± 0.328 µmol $L^{-1}$). Sample sizes were not large enough to statistically test the differences between age groups or sexes.

None of the kea that were sampled in the field showed obvious clinical signs of lead poisoning (such as regurgitation or neurological disease) at the time of capture; however, one bird was seen to be ataxic (showing uncoordinated movements) and clumsy, with a wide-based stance the day after sampling, possibly due to lead exposure (Platt 2006; McLelland et al. 2010). One subadult female was found in Aoraki/Mt Cook Village and showed severe ataxia (to the point that she was unable to fly), and other abnormal behaviours such as failing to exhibit shading behaviour (i.e. staying out in full sun at a time of day when kea would normally seek shade in the beech forest), and making abnormal vocalisations (Reid 2008). She was sent to a veterinary clinic and treated for lead poisoning, but subsequently died. Her blood lead level and necropsy results revealed that she died of lead poisoning.

Of 30 wild kea submitted to Massey University for necropsy between 1991 and 2010, tissue lead levels were available for 20 birds (Table 3), and 11 of these (55%) were diagnosed with lead poisoning (McLelland et al. 2010; BDG unpubl. data).

## Discussion

Blood lead levels in the 88 kea included in analysis convincingly indicate that where kea population ranges overlap with permanent human settlements (e.g. Aoraki/Mt Cook National Park, Fox Glacier – Franz Josef areas) kea are commonly exposed to lead. In remote areas, where kea populations do not overlap with these types of settlements, blood lead levels in kea are lower, indicating that lead exposure is less common.

Establishing what level of lead exposure results in clinical effects is problematic in wild species. Some authors report that concentrations of >0.97 µmol $L^{-1}$ lead in whole blood are suggestive of lead poisoning in psittacines, and concentrations of >1.93 – 2.90 µmol $L^{-1}$ are diagnostic of lead poisoning (Platt 2006). Some birds, however, have exhibited clinical signs and responded to therapy with blood lead levels as low as 0.48 µmol $L^{-1}$, and others have shown no clinical signs with much greater levels (Dumonceaux & Harrison 1994). Although the absence of obvious clinical signs in the live wild kea sampled from populated areas in this study may suggest tolerance to high blood lead concentrations, this does not preclude the diverse subclinical effects of low-level lead exposure (McLelland et al. 2010). In chickens, exposure to even low levels of lead (< 0.48 µmol $L^{-1}$) has been shown to damage the central nervous system (Lurie et al. 2006). In humans it has been suggested that blood lead levels > 0.097 µmol $L^{-1}$ should be regarded as elevated due to the effects of even very low lead levels on biological processes, mainly the nervous system (Gilbert & Weiss 2006). A suggested limit for blood lead levels in Hispaniolan Amazon parrots was < 0.097 µmol $L^{-1}$ (Osofsky et al. 2001).

Kea sampled in populated areas had considerably higher blood lead levels than those in remote areas (Table 1; Fig. 2). The majority of kea sampled in populated areas were above the 0.97 µmol $L^{-1}$ threshold suggestive of lead poisoning in parrots (Platt 2006), and only one bird was below the threshold of 0.097 µmol $L^{-1}$ suggested by Gilbert and Weiss (2006)

**Table 3.** Liver and kidney lead concentrations (µmol $kg^{-1}$ wet weight*) in fresh-frozen and formalin-fixed (FF) tissue from wild kea (*Nestor notabilis*) ($n$ = 20) from database records.

| Kea # | Fresh liver | FF liver | Fresh kidney | FF kidney | Diagnosis of lead poisoning |
|---|---|---|---|---|---|
| 1 | 0.11 | - | - | - | No |
| 2 | 0.19 | - | 0.32 | - | No |
| 3 | 0.92 | 1.06 | - | - | No |
| 4 | 1.93 | - | - | - | No |
| 5 | 2.90 | - | - | - | No |
| 6 | 3.04 | - | 10.13 | - | No |
| 7 | 3.38 | - | 2.41 | - | No |
| 8 | 3.67 | - | 4.83 | - | No |
| 9 | 4.73 | - | 0.48 | - | No |
| 10 | 48.26 | 32.82 | - | - | Yes |
| 11 | 53.57 | - | - | - | Yes |
| 12 | 64.67 | - | 73.36 | - | Yes |
| 13 | 69.49 | - | - | - | Yes |
| 14 | 72.39 | - | 130.30 | - | Yes |
| 15 | 84.94 | - |  | - | Yes |
| 16 | 135.13 | - | - | - | Yes |
| 17 | 136.09 | 390.91 | - | 1109.98 | Yes |
| 18 | 276.05 | 386.08 | - | - | Yes |
| 19 | - | 260.60 | - | 772.16 | Yes |
| 20 | - | - | 59.36 | - | Yes |

*To convert values from µmol $kg^{-1}$ to mg $kg^{-1}$, multiply by the conversion factor 0.2072.

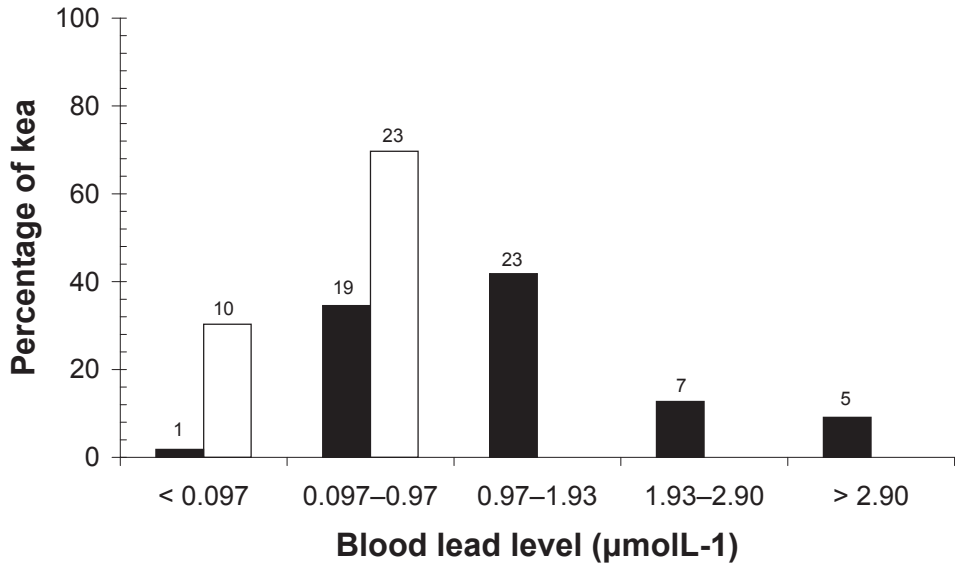


**Figure 2.** Blood lead levels of kea sampled in populated (black bars) and remote (white bars) areas of the South Island, New Zealand, 2006–2009. Numbers above bars represent sample size of kea.

(Fig. 2). In contrast, no birds sampled in remote areas were above the 0.97 µmol $L^{-1}$ threshold, whereas 30% were below the 0.097 µmol $L^{-1}$ threshold. Although Jarrett (1998) had a smaller sample size of kea tested for blood lead levels ($n$ = 11), 82% had blood lead levels above 0.97 µmol $L^{-1}$, and no kea were below the 0.097 µmol $L^{-1}$ threshold level suggested by Gilbert and Weiss (2006). The kea tested in Jarrett's study were from the Arthur's Pass area where there was a permanent human settlement with buildings containing lead materials (Peat 1995), which fits our definition of a populated area.

There is no truly minimal safe level for lead (Pain et al. 2009); for example, even at extremely low concentrations lead affects blood enzyme activity essential for haemoglobin production (Redig et al. 1991; Grasman & Scanlon 1995). Subclinical lead poisoning in chickens has been shown to cause immunosuppression, which disrupts the immune system (Lee et al. 2001; Dietert et al. 2004), and to increase susceptibility to infectious diseases (Youssef et al. 1996). Experimental dosage with low levels of lead (resulting in feather lead levels (mean ± SE) of 0.83 ± 0.09 µmol $kg^{-1}$ (172 ± 18 ng $g^{-1}$ dry weight); Burger & Gochfeld 1990) has been found to affect locomotion, food begging, learning, thermoregulation, and individual recognition in herring gulls (*Larus argentatus*) in the laboratory and in the wild (Burger & Gochfeld 2005). Lead affected chicks' success at more complicated tasks, such as learning the location of hidden food, shading behaviour, and recognising caretakers or siblings (Burger & Gochfeld 2005). Lead-exposed chicks had lowered survival rates in the wild due to abnormal behaviour (Burger & Gochfeld 1994), and the conclusion was that lead profoundly affects neurobehavioural development, and that environmentally relevant lead levels can significantly affect survival (Burger & Gochfeld 2005). Gorissen et al. (2005) found that exposure to heavy metals, including lead, diminished the singing behaviour of wild great tits, which may affect breeding behaviour and be a useful indicator of environmental stress at the population level.

Sources of lead accessible to wild kea include roofing materials such as flashings and lead-head nails, and rubbish items (Peat 1995; Jarrett 1998). These materials are available in many places where kea live (CR & KM, pers. obs.). Young kea congregate in built-up areas where human and kea habitats overlap, such as in alpine townships and ski fields. These groups have been observed spending time on roofs of buildings investigating fixtures (CR, pers. obs.). Young kea, similar to human children, investigate objects and materials via oral exploration. Lead is malleable and is reported to have a sweet taste (Locke & Thomas 1996) that is thought to be attractive to captive psittacines (Lightfoot & Yeager 2008) and these may be factors in the exposure of wild kea to lead. Signs of damage were apparent on lead-head nails and roof flashing of buildings in Aoraki/Mt Cook National Park that were typical of marks left by the bills of chewing parrots (CR, pers. obs.). In a previous study kea were commonly observed chewing on lead-head nails and lead flashing (Brejaart 1994).

Two nestling kea were sampled from a populated area (Aoraki/Mt Cook) in this study and both showed elevated blood lead levels. The nestlings may have been exposed to lead both in the egg and from contaminated food delivered by their parents (McLelland et al. 2010). In other avian species, metal concentrations in fledglings represent in part metals sequestered in the egg by females and accumulation from food brought back to chicks by parents (Burger & Gochfeld 1993). Concentrations of heavy metals in eggs represent not only recent exposure of females but mobilisation of stored metals from past intake (Burger 1994; Burger & Gochfeld 1996). There is a potential for toxic minerals such as lead in the egg to affect later-stage embryos, including critical strength of the embryo needed for hatching success (Mora 2003). If kea are being exposed to lead before fledging, this expands the problem of lead exposure to birds that are not yet visiting anthropogenic sources of lead themselves. In other words, the presence of anthropogenic lead sources in the environment not only affects kea directly accessing those sources, but also their progeny.

The mean blood lead levels for fledglings, juveniles, and subadults sampled from populated areas were above the level suggestive of lead poisoning (Table 2). The high levels seen in younger birds may be related to the fact that kea begin as inefficient foragers, increasing their skill and decreasing their manipulative behaviour as they age (Brejaart 1994; Diamond & Bond 1999). Young kea spend much of their time exploring and manipulating objects, including inedible ones, unlike

adults, which are adept foragers (Diamond & Bond 1991, 1999). The low blood lead levels seen in adults may be due to increased foraging efficiency with age (Diamond & Bond 1999), and less inclination to investigate lead sources such as roofing fixtures (Reid 2008). Blood lead levels indicate recent lead exposure (Pattee & Pain 2003) and lead, once absorbed, is rapidly deposited in a range of tissues, primarily liver, kidney and bone (Pain et al. 2005). Therefore blood lead analysis may not reveal prior lead exposure in adult kea, for example during the early life stages.

Although kea sampled from remote areas were found to have low lead levels, they are still at some risk of lead exposure. Kea disperse as juveniles and can travel considerable distances, e.g. up to 80 km (Diamond & Bond 1999). Kea that originate in populated areas and are exposed to lead there before dispersal may disperse into remote areas and expose their offspring to lead when they reproduce. Also, remote areas inhabited by kea, although lacking permanent human settlements, are not devoid of anthropogenic lead sources (KM & CR pers. obs.). Kea sampled from remote areas in this study may have had access to huts with lead roofing materials, and hunter-killed game carcasses containing lead shot or bullets. Necropsy revealed a partially digested lead shot pellet in the stomach of a juvenile male kea found dead at St Arnaud in October 2000, and tissue lead levels confirmed lead poisoning (R. Norman unpubl. necropsy report). Kea have also been observed feeding on hunter-killed game carcasses (Jackson 1960; Schwing 2010). Lead shot pellets and bullet fragments have exposed several avian species to lead due to ingestion from hunter-killed animal carcasses (Fisher et al. 2006). However, the primary anthropogenic sources of lead in areas inhabited by kea are likely to be older buildings constructed with lead materials.

In light of recent findings (McLelland et al. 2010) and those of this study, accounts of previous kea studies (e.g. Jackson 1969) indicate that lead poisoning may have been more common than was realised at the time (Reid 2008). Behaviours that Jackson (1969) described as normal for wild kea in Arthur's Pass during the 1960s appear abnormal in comparison with recent observations of wild kea in other areas (Reid 2008) and clinical and pathological findings in his report were consistent with lead poisoning (Youl 2009). Jarrett's study of kea in the same area in the 1990s revealed that kea sampled there had been exposed to lead, and that younger birds were more likely to have elevated blood lead levels than older birds (Jarrett 1998). Lead was also implicated as a cause of death in kea (Jarrett 1998). Subsequent studies of kea behaviour and cognition have also been carried out in the areas of Arthur's Pass (e.g. Diamond & Bond 1999), Aoraki/Mt Cook and Fox Glacier (e.g. Gajdon et al. 2006) – areas in which kea have been shown to be exposed to lead. It is likely that kea suffer the same ill-effects from lead exposure as other avian species and, although kea may not show overt clinical signs, their behaviour and cognitive abilities may be affected (Reid 2008). Studies of kea in populated areas may not reflect the normal range of kea behaviours and capabilities of those in remote areas, therefore studies of kea behaviour in remote areas with a lower risk of lead exposure may provide a useful comparison for existing ones (Reid 2008).

Based on our findings, we conclude there is an urgent need to minimise as much as possible the amount of available lead in kea habitat. Lead abatement strategies are currently being carried out to this end, including the removal of lead from buildings such as back-country huts in areas inhabited by kea (P. Gaze and R. Suggate, Department of Conservation, pers. comm.).

## Acknowledgements

This research was carried out in collaboration with the Department of Conservation, Massey University, and Victoria University of Wellington. Work by CR and JMM was carried out with the approval of the Victoria University of Wellington Animal Ethics Committee and Massey University Animal Ethics Committee, respectively. All work was carried out with the approval of DOC. We thank Andrew Hill and all the team at the New Zealand Wildlife Health Centre at Massey University for their assistance with blood lead analysis in the lab. We thank Gyula Gajdon, Anja Meduna, Josh Kemp, Paul Van Klink, and Mike Ogle for collecting and submitting kea blood samples for analysis, and Stuart Thorne and Florence Gaud for assistance in kea capture. We also thank Wayne Linklater for his support as supervisor to CR at the Victoria University of Wellington. Funding for this work was provided by DOC and in part by an IVABS, Massey University postgraduate research grant for JMM, and an internal research grant from Victoria University for CR. We thank Judy Diamond and an anonymous reviewer for their comments on the manuscript.

## References

BirdLife International 2008. *Nestor notabilis*. In: IUCN 2010. IUCN Red List of Threatened Species. Version 2010.1. www.iucnredlist.org. Downloaded 17 May 2010.

Bond AB, Wilson K-J, Diamond J 1991. Sexual dimorphism in the kea *Nestor notabilis*. Emu 91: 12–19.

Brejaart R 1994. Aspects of the ecology of kea, *Nestor notabilis* (Gould), at Arthur's Pass and Craigieburn Valley. Unpublished MApplSc thesis, Lincoln University, Lincoln, New Zealand. 92 p.

Burger J 1994. Heavy metals in avian eggshells: another excretion method. Journal of Toxicology and Environmental Health 41: 207–220.

Burger J, Gochfeld M 1990. Tissue levels of lead in experimentally exposed herring gull (*Larus argentatus*) chicks. Journal of Toxicology and Environmental Health 29: 219–233.

Burger J, Gochfeld M 1993. Lead and cadmium accumulation in eggs and fledgling seabirds in the New York bight. Environmental Toxicology and Chemistry 12: 261–267.

Burger J, Gochfeld M 1994. Behavioral impairments of lead-injected young herring gulls in nature. Fundamental and Applied Toxicology 23: 553–561.

Burger J, Gochfeld M 1996. Heavy metal and selenium levels in Franklin's Gull (*Larus pipixcan*) parents and their eggs. Archives of Environmental Contamination and Toxicology 30: 487–491.

Burger J, Gochfeld M 2005. Effects of lead on learning in herring gulls: an avian wildlife model for neurobehavioral deficits. Neurotoxicology 26: 615–624.

Dey PM, Burger J, Gochfeld M, Reuhl KR 2000. Developmental lead exposure disturbs expression of synaptic neural cell adhesion molecules in herring gull brains. Toxicology 146: 137–147.

Diamond J, Bond AB 1991. Social behavior and the ontogeny of foraging in the kea (*Nestor notabilis*). Ethology 88: 128–144.

Diamond J, Bond AB 1999. Kea, bird of paradox: the evolution and behavior of a New Zealand parrot. University of California Press, Berkeley, California. 230 p.

Dietert RR, Lee J-E, Hussain I, Piepenbrink M 2004. Developmental immunotoxicology of lead. Toxicology and Applied Pharmacology 198: 86–94.

Dumonceaux G, Harrison GJ 1994. Toxins. In: Ritchie BW, Harrison GJ, Harrison LR eds Avian medicine: principles and applications. Lake Worth, FL, Wingers. Pp. 1030–1052.

Elliott G, Kemp J 1999. Conservation ecology of kea (*Nestor notabilis*). WWF-NZ final report. New Zealand, World Wide Fund for Nature. 64 p.

ESA Biosciences Inc. 2005. LeadCare® blood lead testing system user's guide. ESA Biosciences Inc., 22 Alpha Rd., Chelmsford 01824, MA, USA.

Fisher IJ, Pain DJ, Thomas VG 2006. A review of lead poisoning from ammunition sources in terrestrial birds. Biological Conservation 131: 421–432.

Fleming RH, McCormack HA, Whitehead CC 2000. Prediction of breaking strength in osteoporotic avian bone using digitized fluoroscopy, a low cost radiographic technique. Calcified Tissue International 67: 309–313.

Fox J 2009. Rcmdr: R Commander version 1.5-2. R package. Vienna, Austria, R Foundation for Statistical Computing. http://www.r-project.org

Gajdon GK, Fijn N, Huber L 2006. Limited spread of innovation in a wild parrot, the kea (*Nestor notabilis*). Animal Cognition 9: 173–181.

Gangoso L, Álvarez-Lloret P, Rodríguez-Navarro AAB, Mateo R, Hiraldo F, Donázar JA 2009. Long-term effects of lead poisoning on bone mineralization in vultures exposed to ammunition sources. Environmental Pollution 157: 569–574.

Gartrell BD, Reid C 2007. Death by chocolate: A fatal problem for an inquisitive wild parrot. New Zealand Veterinary Journal 55: 149–151.

Gelis S 2006. Evaluating and treating the gastrointestinal system. In: Harrison GJ, Lightfoot TL eds Clinical avian medicine. Vol. 1. Palm Beach, FL, Spix. Pp. 411–440.

Gilbert SG, Weiss B 2006. A rationale for lowering the blood lead action level from 10 to 2 μg/dL. NeuroToxicology 27: 693–701.

Golder Associates 2008. Esperance - data gap analysis and conceptual site model: stage 1. Report No. 07764343414-R01-Rev1. Unpublished paper submitted to the Department of Environment and Conservation. Golder Associated Pty Ltd., West Perth. 265 p.

Gorissen L, Snoeijs T, Van Duyse E, Eens M 2005. Heavy metal pollution affects dawn singing behaviour in a small passerine bird. Oecologia 145: 504–509.

Grasman KA, Scanlon PF 1995. Effects of acute lead ingestion and diet on antibody and T-cell-mediated immunity in Japanese quail. Archives of Environmental Contamination and Toxicology 28: 161–167.

Gulsen B, Korsch M, Matisons M, Douglas C, Gillam L, McLaughlin V 2009. Windblown lead carbonate as the main source of lead in blood of children from a seaside community: An example of local birds as "canaries in the mine". Environmental Health Perspectives 117: 148–154.

Jackson JR 1960. Keas at Arthur's Pass. Notornis 9: 39–58.

Jackson JR 1969. What do Keas die of? Notornis 16: 33–44.

Jarrett M 1998. Hazards to kea (*Nestor notabilis*) at rubbish dumps. Unpublished MApplSc thesis, Lincoln University, Lincoln, New Zealand. 103 p.

Kertész V, Bakonyi G, Farkas B 2006. Water pollution by Cu and Pb can adversely affect mallard embryonic development. Ecotoxicology and Environmental Safety 65: 67–73.

Lee J-E, Chen S, Golemboski KA, Parsons PJ, Dietert RR 2001. Developmental windows of differential lead-induced immunotoxicity in chickens. Toxicology 156: 161–170.

Lightfoot TL, Yeager JM 2008. Pet bird toxicity and related environmental concerns. Veterinary Clinics of North America: Exotic Animal Practice 11: 229–259.

Locke LN, Thomas NJ 1996. Lead poisoning of waterfowl and raptors. In: Fairbrother A, Locke LN, Hoff GL eds Noninfectious diseases of wildlife. 2nd edn. Ames, IA, Iowa State University Press. Pp. 108–117.

Lurie D, Brooks DM, Gray LC 2006. The effect of lead on the avian auditory brainstem. NeuroToxicology 27: 108–117.

McLelland JM, Reid C, McInnes K, Roe WD, Gartrell BD 2010. Evidence of lead exposure in a free-ranging population of kea (*Nestor notabilis*). Journal of Wildlife Diseases 46: 532–540.

Miskelly CM, Dowding JE, Elliott GP, Hitchmough RA, Powlesland RG, Robertson HA, Sagar PM, Scofield RP, Taylor GA 2008. Conservation status of New Zealand birds. Notornis 55: 117–135.

Mora MA 2003. Heavy metals and metalloids in egg contents and eggshells of passerine birds from Arizona. Environmental Pollution 125: 393–400.

Osofsky A, Jowett PLH, Hosgood G, Tully TN 2001. Determination of normal blood concentrations of lead, zinc, copper, and iron in Hispaniolan Amazon parrots (*Amazona ventralis*). Journal of Avian Medicine and Surgery 15: 31–36.

Pain DJ, Meharg AA, Ferrer M, Taggart M, Penteriani V 2005. Lead concentrations in bones and feathers of the globally threatened Spanish imperial eagle. Biological Conservation 121: 603–610.

Pain DJ, Fisher IJ, Thomas VG 2009. A global update of lead poisoning in terrestrial birds from ammunition sources. In: Watson RT, Fuller M, Pokras M, Hunt WG eds Ingestion of lead from spent ammunition: implications for wildlife and humans. Boise, ID, The Peregrine Fund. Pp. 99–118.

Pattee OH, Pain DJ 2003. Lead in the environment. In: Hoffman DJ, Rattner BA, Burton GA Jr, Cairns J Jr eds Handbook of ecotoxicology. 2nd edn. Boca Raton, FL, Lewis. Pp. 373–408.

Peat N 1995. Kea advocacy strategy: towards resolving conflicts between kea and people. Miscellaneous Report Series 28. Dunedin, Otago Conservancy, Department of Conservation. 37 p.

Platt SR 2006. Evaluating and treating the nervous system. In: Harrison GJ, Lightfoot TL eds Clinical avian medicine. Vol. 2. Palm Beach, FL, Spix. Pp. 493–515.

R Development Core Team 2009. R 2.9.2: A language and environment for statistical computing. Vienna, Austria, R Foundation for Statistical Computing. http://www.r-project.org

Redig PT, Lawler EM, Schwartz S, Dunnette JL, Stephenson B, Duke GE 1991. Effects of chronic exposure to sublethal concentrations of lead acetate on heme synthesis and immune function in red-tailed hawks. Archives of Environmental Contamination and Toxicology 21: 72–77.

Reid C 2008. Exploration-avoidance and an anthropogenic toxin (lead Pb) in a wild parrot (kea: *Nestor notabilis*).

Unpublished MSc thesis, Victoria University of Wellington, Wellington, New Zealand. 95 p.
Schwing R 2010. Scavenging behaviour of kea. Notornis 57: 98–99.
Toscano CD, Guilarte TR 2005. Lead neurotoxicity: From exposure to molecular effects. Brain Research Reviews 49: 529–554.
Verity MA 1997. Toxic disorders. In: Graham DI, Lantos PL eds Greenfield's neuropathology. 6th edn. Vol. 1. Arnold, London. Pp. 755–811.
Wang J 2000 Analytical electrochemistry. 2nd edn. New York, John Wiley. 209 p.
Whitehead CC, Fleming RH 2000. Osteoporosis in cage layers. Poultry Science 79: 1033–1041.
Youl JM 2009. Lead exposure in free-ranging kea (*Nestor notabilis*), takahe (*Porphyrio hochstetteri*) and Australasian harriers (*Circus approximans*) in New Zealand. Unpublished MVSc thesis, Massey University, Palmerston North, New Zealand. 152 p.
Youssef SAH, El-Sanousi AA, Afifi NA, El-Brawy AMA 1996. Effect of subclinical lead toxicity on the immune response of chickens to Newcastle disease virus vaccine. Research in Veterinary Science 60: 13–16.

Editorial Board member: Joanne Hoare
Received 11 February 2011; accepted 31 May 2011