

Kea (*Nestor notabilis*) and Lead

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Introduction

The kea (*Nestor notabilis*) is the world's only sub-alpine parrot. It is endemic to the high country of the South Island, however the fossil record indicates it was once found in the North Island (Heather & Robertson, 1996). The size of the population is unknown, although it is estimated to be approximately 1000-5000 birds (Anonymous, 2006) and they are listed as vulnerable by the IUCN (International Union for the Conservation of Nature and Natural Resources or World Conservation Union) red list. Threats to kea include predation, competition (with introduced animals and habitat use) and pollution (IUCN, 2006). Keas are primarily herbivores, although they are opportunistic and will sample any available food source. This foraging behaviour and their particularly inquisitive nature has lead to many incidences of toxicity as kea sample items from tourists, rubbish dumps, buildings, board walks and cars just to name a few. Many of rubbish or construction items contain lead such as lead flashing and lead-head nails. Young kea may be at higher risk of developing lead toxicosis as manipulation and chewing objects is thought to be a part of their development process. One previous study looking at hazards to kea at rubbish dumps found that of those kea sampled, young birds were much more likely to have elevated lead levels (Jarrett, 1998).

This study has involved a review of kea entered into the New Zealand wildlife post-mortem database 'Huia', analysis of lead in archived tissues and a survey of blood lead levels in wild kea.

Review of Post Mortem Database

Since 1991, there have been thirty kea entries in the wildlife post-mortem database "Huia". Twenty-eight of these were necropsies. Of these, seventeen were captive birds, ten wild and one unknown (assumed captive). Information regarding the cause or suspected cause of death of these birds was available for all wild birds and twelve of the captive birds.

Primary cause of death	Number of wild birds	Number of captive birds	Total
Lead poisoning	4	1	5
Aspergillus	2	1	3
Possible toxicity	1	2	3
Other toxicity	2		2
Other	1	4	5
Gastrointestinal disease		4	4
TOTAL	10	12	22

Gastrointestinal disease includes, a polyp causing oesophageal obstruction, intestinal carcinoma and ventriculitis. Other includes renal disease, starvation, salpingitis/metritis and parasitism.

Only the wild birds will be discussed further.

The ten wild keas entered into the database were found dead or debilitated (dying soon after capture). Six of these birds died due to a toxic insult (lead, theobromine and possible cyanide). The four other birds died from conditions commonly associated with debilitation such as emaciation and aspergillosis. None of these four were tested for lead at the time of necropsy. Three of the ten birds were less than one year old and two of these had confirmed lead poisoning. One bird of unrecorded age was confirmed with lead poisoning and another was suspicious. One of the five adult birds had confirmed lead poisoning.

Most native wildlife that passes through the post-mortem room at Massey University have their preserved tissues archived. Of the ten wild keas, liver tissue from eight has been submitted for lead analysis. These results are not available at the time of writing.

Due to the small population size of kea and inhospitable habitat, it is likely that many kea that die with possible lead toxicosis, or other conditions and have not presented for necropsy.

Blood lead survey of wild kea

Capture Methods

Twelve previously uncaught juvenile male keas were captured outside the Hermitage in Mt Cook/Aoraki village over six days in early 2006. The keas natural curiosity causing them to investigate new objects was utilised to capture them. A snare was set (a light bamboo pole with string attached and a noose at the end), raised on small rocks to prevent snagging on the ground. A rock with butter was placed in the middle. Once a bird stepped into the snare with both feet, the noose was pulled tight around one or both of the legs and the bird was suspended then restrained for examination and blood collection. Other methods of capture included using a lure (orange dog bone smeared with butter, attached to a string) and hoop net. Birds were lured into the proximity of the hoop net and the net then dropped over them. An individual bird was very curious about the net and came close enough to be caught without a lure. Playing hide and seek with one bird enabled hand catching when it got too close.

Sample collection

Immediately after capture the birds were blood sampled from the left or right ulna and/or medial metatarsal vein. The ulna vein was found to be preferable due to the ability to collect larger volumes of blood. Blood was collected into 1ml syringes using a 25ga needle. Two smears were made with fresh blood and the remaining blood was placed into 0.4ml lithium heparin microtainers and mixed.

Clinical examination

A clinical examination was carried out on each bird. The condition of the bird was estimated using the prominence of the keel and the pectoral muscles. Each bird was examined by the same person to ensure consistency. The mouth, eyes, ears and cloaca were visually examined. The abdomen was palpated and the flight feathers examined for condition, stress barring and parasites.

Banding

All birds were banded following sample collection. Coloured bands were placed on the right tarsometatarsus to enable visual identification without capture. Steel bands with identification numbers were placed on the left tarsometatarsus. This ensured we did not sample birds twice.

Analysis

The blood was analysed for lead content using a portable leadcare machine. All blood samples were analysed for lead content within three hours of collection.

Blood in lithium heparin microtainers were submitted to a commercial laboratory between three and four days post collection. The time delay was due to the remote location. Prior to submission, samples were kept refrigerated at four degrees Celsius or insulated in bubble wrap with ice packs.

Results

Number	Lead ug/dl	Lead mg/L	PCV
10	8.7	0.087	Not done
9	13.5	0.135	0.56
2	14.8	0.148	0.51
12	16.2	0.162	0.54
1	21.3	0.213	0.58
5	21.6	0.216	0.48
3	23.7	0.237	0.47
6	24.7	0.247	0.45
4	26.7	0.267	0.49
7	29.2	0.292	Not done
11	32.8	0.328	0.5
8	69.0*	0.69*	0.45

Table 2: Lead levels and packed cell volumes of 12 wild juvenile male kea from the Mt Cook/Aoraki Village. *The original measurement was "Hi" (upper limit of the analyser is >65ug/dL. A 1:10 dilution with saline gave us this result.

The results indicate that all the birds sampled have been exposed to lead. Results ranged from 8.7µg/dL to 69.0 µg/dL with an average level of 25.2µg/dL. There appears to be a trend of decreasing PCV with increasing lead levels (with the exception of occasional birds) however all of these are within what we would consider a normal range for a sub-alpine bird. These results have not been statistically analysed at the time of writing.

Limitations

All the kea sampled were juvenile males that were captured in a human inhabited area. Young female birds are more cautious than male birds and are less likely to be caught. Adult birds are less likely to congregate in groups around human habitation and are often more difficult to catch. These birds were often caught as juveniles and remember the capture methods. Due to the difficulties in capturing birds that do not frequent areas where there are humans, a control population has not been obtained.

Discussion

None of the keas tested showed overt clinical signs at the time of sampling. Bird 6 was observed to be ataxic and clumsy with a wide-based stance the day after sampling, however we were unable to recapture him, and he was not seen again during the rest of the field trip.

In humans, lead levels greater than 10ug/dL indicate potential poisoning and levels above 65ug/dL should be treated as an emergency (Anonymous, 2005). In parrots, it has been suggested that blood

lead levels greater than 20ug/dL are suggestive of lead poisoning and if accompanied by clinical signs, levels greater than 40-60 ug/dL are diagnostic (Dumonceaux & Harrison, 1994). However, some birds have shown clinical signs and responded to therapy with blood lead levels as low as 10ug/dL, and others have had no clinical signs with much higher levels (Dumonceaux & Harrison, 1994).

As mentioned above, it is recommended that blood lead levels be interpreted in conjunction with the observation of clinical signs. This however does not take into account the sub-clinical effects of lead or the effects that chronic lead exposure may have. In humans, lead is known to cause impaired learning ability, neuromuscular defects and altered haeme metabolism among other things (Silbergeld & Goldberg, 1980). Young rats also appear to absorb and retain significantly more lead per dose than adults (Momcilov & Kostial, 1974). The kea sampled were all young birds so it may be that lead has a more significant effect. During their first two years, keas spend considerable time developing motor and coordination skills as well as feeding and foraging skills (Temple, 1996). The skills developed are very important for survival in a sub-alpine environment (Temple, 1996). When chronically exposed to lead, accumulations occur in the bone. Bone-bound lead has a long half life and is considered inert(Needleman, 1980). However, the lead is mobilised when bone turn over is increased, such as in osteoporosis in humans, potentially causing recrudescence of clinical disease(Needleman, 1980). This may be a problem for female birds when they begin laying. The effects of lead on impaired learning ability, as well as other body systems may be detrimental to the survival of the species.

Further work

It is hoped that more wild keas will be sampled this season to increase the sample size. All Kea submitted for necropsy will have liver lead analysis performed.

References

- Anonymous. (2005). *LeadCare Blood Lead Testing System Users Guide*: ESA.
- Anonymous. (2006). *Kea (New Zealand's mountain parrot*. Retrieved 11/08/2006, from <http://www.doc.govt.nz/Conservation/001~Plants-and-Animals/001~Native-Animals/Kea.asp>
- Dumonceaux, G., & Harrison, G. J. (1994). *Toxins*. In Ritchie BW, Harrison GJ & Harrison LH (Eds.), *Avian Medicine: Principles and Applications* (pp. 1034-1038). Florida: Wingers Publishing.
- Heather, B. D., & Robertson, H. A. (1996). *The field guide to the birds of New Zealand*. Auckland,: Viking.
- IUCN. (2006). *The IUCN Red List of Threatened Species: Nestor notabilis*. Retrieved 11/08/2006, from <http://www.iucnredlist.org/search/details.php/14729/summ>
- Jarrett, M. I. (1998). *Hazards to kea (Nestor notabilis) at rubbish dumps : a thesis submitted in partial fulfilment of the requirements for the degree of Master of Applied Science at Lincoln University*. Lincoln University.
- Momcilov, B., & Kostial, K. (1974). Kinetics of Lead Retention and Distribution in Suckling and Adult Rats. *Environmental Research*, 8(2), 214-220.
- Needleman, H. L. (1980). Human Lead Exposure: Difficulties and Strategies in the Assessment of Neuropsychological Impact. In R. L. Singhal & J. A. Thomas (Eds.), *Lead Toxicity* (pp. 15-16). Baltimore: Urban & Schwarzenberg.

- Silbergeld, E. K., & Goldberg, A. M. (1980). Problems in Experimental Studies of Lead Poisoning.
In R. L. Singhal & J. A. Thomas (Eds.), *Lead toxicity* (pp. 37). Baltimore: Urban &
Schwarzenberg.
- Temple, P. (1996). *The book of the kea*. Auckland, N.Z.: Hodder Moa Beckett.

