# LEAD TOXICITY IN NEW ZEALAND BROWN TEAL (*ANAS CHLOROTIS*)

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## *Lead and lead toxicity*

Lead is one of the most toxic metals known to man and can cause disease in wild animals, domestic animals and humans worldwide (Fisher et al., 2006). Lead is a highly toxic non-essential heavy metal found naturally in the environment in small quantities however environmental contamination can occur due to anthropogenic use of lead based products (Hoffman et al., 2002; Pattee and Pain, 2003).

The most common routes of lead absorption by animals and humans are by ingestion, inhalation or absorption through the skin depending on the type of lead compound (Pattee and Pain, 2003). Lead is commonly used in domestic items such as shot, paint, batteries, plumbing material, solder, fishing sinkers, galvanised wire, foil, construction material, petrol among others (Dumonceaux and Harrison., 1994; Locke and Thomas, 1996).

The most common cause of lead toxicity encountered in wildlife is ingestion of lead shot, fishing sinkers and other sources of lead found in the environment (Hoffman et al., 2002; Davidson, 2006). The most commonly affected wild birds are waterfowl and birds of prey due to either direct ingestion of lead or indirect means such as via prey containing lead shot (Samour and Naldo, 2005; Davidson, 2006; Pain et al., 2009; Lambertucci et al., 2011). Other terrestrial birds and seabirds are also at risk due to the anthropogenic contamination of the environment with lead (Fisher et al., 2006; Pain et al., 2009).

## *Lead toxicosis effects and clinical signs*

Lead toxicity is caused by lead replacing cations, such as calcium and magnesium, throughout the body and clinical signs will vary dependent on the dose, the chronicity of exposure and the body system affected (Pattee and Pain, 2003). The effects of lead poisoning are non-specific and can affect several body systems (see Table 1 for effects of lead toxicity). Clinical signs vary depending on the level and duration of exposure, the diet of the species affected, age and reproductive status (Lauwerys and Hoet, 2001). The clinical signs of lead toxicity can be acute if lead is absorbed in large amounts over a short period of time or chronic if exposure levels are low over a prolonged period of time (Pain, 1996).

Once lead is ingested and moves into the ventriculus and proventriculus, the acidity of the gastrointestinal tract turns the lead soluble which is then actively absorbed into the blood. Once lead is absorbed into the bloodstream it is stored within red blood cells, soft tissue and bones (Pattee and Pain, 2003). Bird species with a muscular ventriculus absorb lead particles more efficiently; and bird species that regurgitate indigestible items can occasionally expel the metal particles in the cast (Pain 1996).

Clinical signs seen in birds are often vague or non-specific and can include lethargy, weakness, anorexia, weight loss, regurgitation, distended crop and proventriculus, polyuria, ataxia, circling, head tilt, blindness, convulsions, haemoglobinuria, anaemia and death (Dumonceaux and Harrison, 1994). The severity of the clinical signs does not always correlate with whole blood lead concentration (Pain, 1996).

## *Diagnosis*

Diagnosis of lead toxicity involves a thorough physical examination as well as a neurological exam, consideration of the medical history (if available), and ancillary tests such as blood biochemistry, blood lead levels, haematology and radiographs (Dumonceaux and Harrison, 1994; Richardson, 2006). The main tool in diagnosing and assessing the degree of lead exposure is a blood lead level however there is no universally agreed level associated with clinical signs across all bird species (Pain, 1996). Levels of 0.1mg/L are used as an indicator of potential toxicity in human medicine. For the purpose of this article we will be using the categories of lead levels of Stauber et al. (2010) (see Table 2).

Blood biochemistry and haematology parameters are not diagnostic of lead toxicity but can indicate organ damage, or demonstrate anaemia and basophilic stippling (common in mammals) (Lauwerys and Hoet, 2001).

Radiographs are helpful in assessing whether any radio opaque objects are present in the gastrointestinal tract. These may require removal using bulking agents, via endoscopy, or surgically depending on the size of the particles (Dumonceaux and Harrison 1994; Richardson, 2006).

Post mortem findings commonly reported in wild birds include muscle wasting, poor body condition, absent or reduced visceral fat, an enlarged gallbladder, impacted proventriculus with damaged ventricular lining, and lead shot or other particles present in the ventriculus. In suspected cases of lead toxicity liver, kidney and bone tissue should be collected and tested for lead levels. Post mortem radiographs are often useful in identifying radiopaque metallic foreign bodies in the gizzard, but this is not present in all cases of lead poisoning (Davidson, 2006).

## *Treatment*

Patients with lead toxicity should receive supportive care such as fluids, heat and adequate nutrition. Specific treatment includes the use of a binding or chelating agent. There are three chelating agents used for the treatment of lead toxicosis in avian species however Calcium EDTA is the drug of choice (see Table 3 for lead toxicity treatment drugs). Some clinicians advocate the use of antibiotics and antifungals during the course of treatment for lead toxicity, as well as vitamin supplements and iron (Dumonceaux and Harrison, 1994; Richardson, 2006).

If radiographs show that heavy metal particles are present then removing the particles is advisable. Several approaches can be taken depending on the size, shape and location, for example the use of lubricants, cathartics or bulking agents to allow the metal particles to be excreted, or removal by endoscopy, lavage or surgery. Radiographs should be repeated to assess whether all lead particles have been removed (Dumonceaux and Harrison., 1994; Richardson, 2006).

## *Introduction to Brown Teal*

The Brown Teal or Pateke (*Anas chlorotis*) is a species of small dabbling duck endemic to New Zealand (Marchant and Higgins, 1990). They were formerly widespread throughout New Zealand but have declined in numbers since the arrival of Maori and European settlers due to introduced carnivorous predators, excessive hunting, habitat and nest destruction, disease, competition for food and habitat with introduced species (Barrie and Robertson, 2005).

The Brown Teal are considered nationally vulnerable recovering under the Department of Conservation (DOC) classification system (O'Connor et al., 2007) and endangered under the IUCN (IUCN, 2009). It is New Zealand's rarest waterfowl species on the mainland with an estimated population of 2000 to 2500. A captive breeding program is run as part of the Brown Teal Recovery Plan involving zoological institutions and private aviculturist across New Zealand (O'Connor et al., 2007).

## *Brown Teal Case Study*

A breeding pair of Brown Teal, a 7 year old male and a 9 year old female, was presented to The Nest Te Kohanga (Veterinary Hospital) at Wellington Zoo for a health check. This pair was part of the captive breeding program and their holder was concerned about weight loss and lethargy in the male Brown Teal. The holder runs a rehabilitation centre for waterfowl in the outskirts of Wellington and holds another 9 Brown Teal.

A thorough physical examination was performed on the two birds on arrival. Both birds were bright, alert and responsive and no obvious abnormalities were detected on physical examination. The male Brown Teal (Fig. 1) was slightly underweight with a body condition score of 2/9 and weight of 497 grams (average weight 620-700), while the female Brown Teal had a body condition score of 6/9 and weight of 480 grams (average weight 530-600). As the birds had travelled for over an hour fluids were administered via crop tube, food was offered ad lib and they were left to settle overnight in preparation for further tests the next day.

**Figure 1** – Brown Teal being examined at the Nest.

***Differential diagnosis***

The clinical signs were vague as the main abnormality on physical examination was a poor body condition score on the male Brown Teal with no obvious abnormalities on physical examination. The initial differential diagnosis was weight loss due to parasitism, inflammation, infection, food competition or toxicity.

***Diagnostic test***

The following day the male Brown Teal was anaesthetised with oxygen and isoflurane. Radiographs and bloods were taken for biochemistry, haematology, lead and zinc levels. A pooled faecal sample was collected to check for parasites and a cloacal swab was cultured for *Salmonella* and *Yersinia* spp. Supportive care was continued in the form of oral fluids and supportive feeding until results were available. The next day the female Brown Teal was anaesthetised and the same tests were performed. Radiographic findings showed no abnormalities in the male Brown Teal (Fig. 2). The female Brown Teal had radiopaque particles in the ventriculus as well as evidence of skeletal changes associated with an old fracture mid radius in the right wing (Fig. 3)

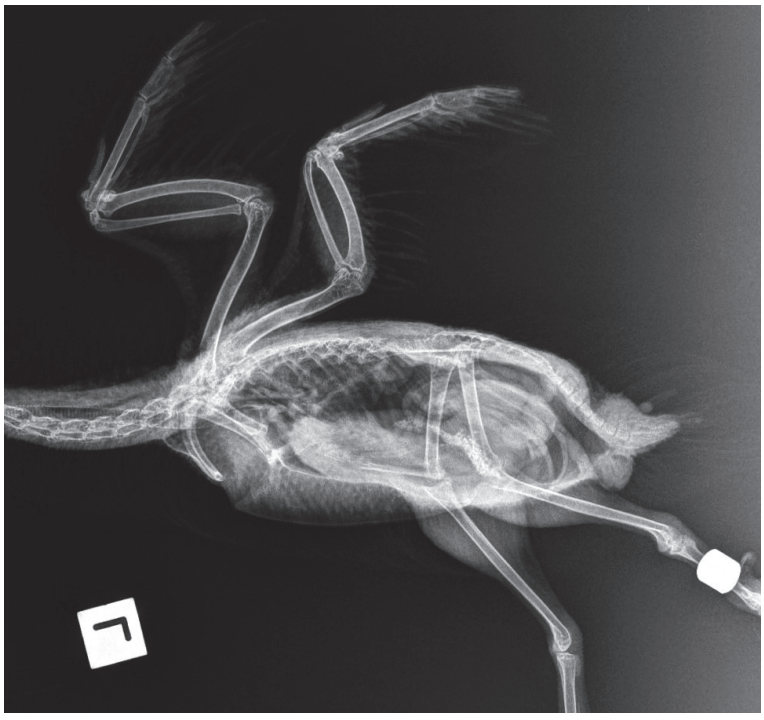

**Figure 2** – Lateral radiograph of male Brown Teal. No obvious abnormalities detected.

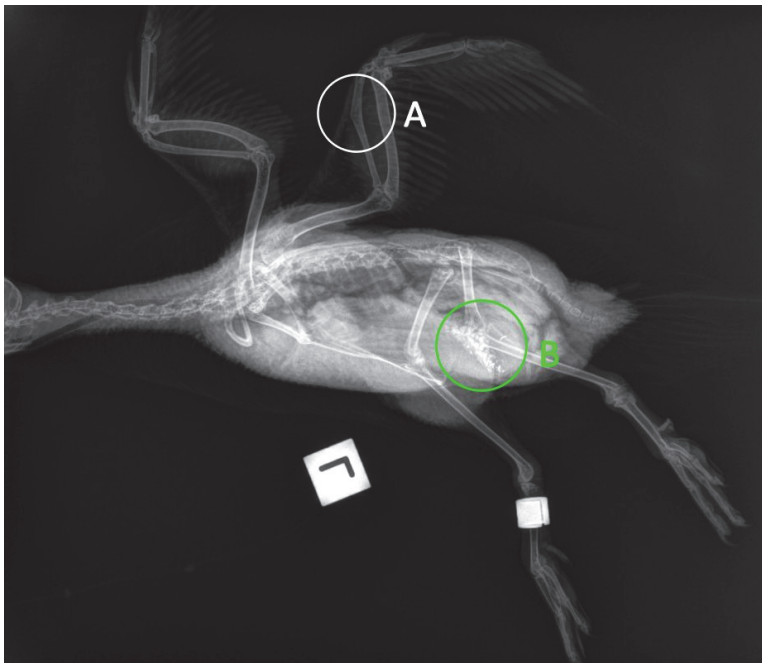


**Figure 3:** Lateral view of female Brown Teal. A - old fracture of the right radius; B - small radiopaque particles and wire in the ventriculus.

The blood biochemistry panel was run on an in-house Vetscan biochemistry analyser and was within normal limits for all measured parameters. The haematology results showed a low level anaemia in the male Brown Teal with signs of regeneration. See Table 4 for results.

The blood sample, faeces and culture swab were sent to a commercial diagnostic laboratory for analysis. No parasites were identified in the faecal sample and the culture swab was negative for Salmonella and Yersinia for both birds. Blood lead levels were measured on a portable blood lead analyser (LeadCareH, ESA Inc., Chelmsford, Massachusetts, USA). Lead levels in the male Brown Teal were 0.11mg/L and 0.7mg/L in the female Brown Teal. These levels are considered to be consistent with lead toxicity (Stauber et al., 2010).

***Treatment***

Upon diagnoses of lead toxicity both birds were started on Ca-EDTA 20% chelation therapy (35mg/kg IM q12h; Provet NZ Pty Ltd, Christchurch) for 5 consecutive days with a 3 day rest period. Symptomatic and supportive care was given in the way of oral fluids at a 1.5x maintenance rate and Harrison recovery diet 20ml PO BID. Repeat blood lead level tests were taken to assess treatment response during the rest period. The female Brown Teal was also given psyllium (Metamucil PO SID; Procter and Gamble) orally once a day as an aid to help pass the metal particles. No antibiotics or antifungals were used.

### *Case Progression and further diagnostics*

After the first round of chelation the lead levels of the male Brown Teal decreased to 0.05mg/L which is considered a subclinical level so further treatment was not required. Within a week of treatment the male Brown Teal had gained approximately 200 grams.

The female Brown Teal required a total of three courses of chelation with Ca-EDTA to reduce lead levels to below 0.09mg/L. At Wildbase levels below 0.1mg/L are considered subclinical, (personal communication). Radiographs were repeated under general anaesthetic to ensure the metal particles had been expelled. As there was no further evidence of metal particles in the ventriculus and clinically the Brown Teal was gaining weight and behaving normally the chelation therapy was stopped.

### *Flock and aviary investigations*

Once the two Brown Teal were diagnosed with lead toxicity their holder was contacted to ask about the facility layout, water supply, soil supply, material of the aviaries and how many other birds were at risk. His Brown Teal collection consisted of 11 birds in total (including the birds currently being treated). There were 2 breeding pairs, 2 adult females and 5 juvenile birds. None of the remaining animals at the facility were showing any signs of disease or ill-thrift. The decision was made to test the 9 remainder Brown Teal for lead toxicity at the property and the recommendation was made to test water and soil samples of the four aviaries housing the birds.

The remaining 9 birds showed levels of lead exposure despite not showing clinical signs of lead toxicity at the time of the sampling. On physical examination they appeared healthy and their weights were within normal limits. Five birds had lead levels less than 0.1mg/L, three birds had lead levels of 0.1mg/L and one bird had levels of 0.2mg/L. As lead has no biological function a safe exposure limit for most species has not been determined. As the birds were not showing clinical signs of toxicity arrangements were made to move the birds to different aviaries and monitor them for any change in behaviour. Only the Brown Teal with a lead level of 0.2mg/L was treated.

The owner of the waterfowl rehabilitation centre was asked to collect water and soil samples from each aviary for lead testing. All water samples were negative while the soil sample measured 612 mg/kg. The diagnosis of lead toxicity and evidence of lead exposure in all birds in the 4 aviaries suggested that contaminated soil was the source of the lead. The Brown Teal likely ingested lead particles when foraging for food and grit on the ground or ate lead contaminated prey such as worms or insects. A metal detector identified large metal particles in the soil in the aviaries. The owner planned to remove a large layer of the top soil before the birds were returned to the aviaries. The rest of the Brown Teal were moved to different aviaries and recommendations were made to re-test the blood lead levels a few weeks later unless the teal showed signs of disease.

## *Discussion*

This report describes the clinical course, diagnostics and pathological findings in a pair of Brown Teal with lead toxicity. The male Brown Teal presented with non-specific clinical signs of disease. A full physical examination followed by ancillary diagnostic tests on both birds came to the diagnosis of lead toxicity. The treatment with CaEDTA resolved the clinical signs and the blood lead levels came down to subclinical values.

However over the last few years we have seen an increase in cases of lead exposure and toxicity in native and endangered species both in captivity and in the wild. Affected species include waterfowl such as Brown Teal and raptors such as Australasian Harriers (*Circus approximans*) (MacLelland et al., 2010) as well as other terrestrial birds such as Gruiformes Takahē (*Porphyrio hochstetteri*) (Youl, 2009), psittacine birds such as Kea (*Nestor notabilis*) (MacLelland et al., 2010) and Kaka (*Nestor meridionalis*), ratites such as North Island Brown Kiwi (*Apteryx mantelli*), Sphenisciformes such as Yellow Eyed Penguin (*Megadyptes antipodes*) and other seabirds (personal communication).

Despite attempts to reduce the use of lead based product and the ban of certain lead shot use in the vicinity of water bodies and wetlands the environmental contamination of New Zealand seems to involve a large range of habitats (Youl, 2009). The anthropogenic lead contamination poses a health risk for all the wild animals in both natural environment and captivity.

## *References*


Anderson A, Pueschel S and Linakis J, 1996. Pathophysiology of lead poisoning. Lead poisoning in children. Baltimore (MD), PH Brookes, 75-96.

Barrie H and Robertson H, 2005. The Field guide to the Birds of New Zealand (Revised Edition). Viking, pp 270-271.

Davidson, WR, 2006. Field manual of wildlife diseases in the southeastern United States. Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, GA.

Dumonceaux G and Harrison GJ,1994. Chapter 37: Toxins. In BW Ritchie, GJ Harrison, and LH Harrison (eds). Avian Medicine: Principles and Applications. Wingers Publishing, Lake Worth, Florida, pp. 1034-1038.

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

Hoffman DJ, 2003. Chapter 4:Wildlife toxicity testing. In DJ Hoffman, BA Rattner, GA Burton Jr and J Cairns Jr (eds),Handbook of ecotoxicology. CRC press, 75-110.

International Union for Conservation of Nature IUCN, 2009. Red List of Threatened Species, 2009.

Lambertucci SA, Donázar JA, Huertas AD, Jiménez B, Sáez M, Sanchez-Zapata JA and Hiraldo F, 2011. Widening the problem of lead poisoning to a South-American top scavenger: Lead concentrations in feathers of wild Andean condors. Biological Conservation 144, 1464-1471.

Lauwerys RR and Hoet P, 2001. Industrial chemical exposure: guidelines for biological monitoring. CRC Press.

Locke L and Thomas N. 1996. Lead poisoning of waterfowl and raptors. Noninfectious diseases of wildlife, 2, pp 108-117.

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

O'Connor SM, Maloney R and Pierce RJ, 2007. Pateke (Anas chlorotis) recovery plan, 2005-10. Science & Technical Pub., Department of Conservation.

Pain DJ, 1996. Lead in waterfowl. In WN Beyer, GH Heinz and AW Redmon-Norwood (eds), Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations.Lewis Publishers, CRC Press, Boca Raton, 251-264.

Pain DJ, Fisher I and Thomas VG, 2009. A global update of lead poisoning in terrestrial birds from ammunition sources. Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans, The Peregrine Fund, Boise, Idaho, 99-118.

Parton K, Bruere AN and Chambers J, 2001. Veterinary clinical toxicology. Veterinary Continuing Education, Massey University.

Pattee OH and Pain DJ,2003. Chapter 15: Lead in the environment. In DJ Hoffman, BA Rattner, GA Burton Jr and J Cairns Jr (eds), Handbook of Ecotoxicology. Lewis Publishers, Boca Raton, 373-408.

Richardson JA, 2006. Chapter 31: Implications of toxic substances in clinical disorders. In G Harrison and T Lightfoot (eds), Clinical Avian Medicine, vol. 2. Spix Publishing, Inc Palm Beach, Florida, 714-715.

Samour J and Naldo JL, 2005.Lead toxicosis in falcons: a method for lead retrieval. Seminars in Avian and Exotic Pet Medicine. Elsevier, 143-148.

Stauber E, Finch N, Talcott PA and Gay JM, 2010. Lead Poisoning of Bald (Haliaeetus leucocephalus) and Golden (Aquila chrysaetos) Eagles in the US Inland Pacific Northwest Region-An 18-year Retrospective Study: 1991-2008. Journal of Avian Medicine and Surgery 24, 279-287.

System, I. S. I. 2013. Physiological Data Reference Values. International Species Inventory System Apple Valley, Minnesota.

Youl JM, 2009. Lead exposure in free-ranging kea (Nestor notabilis), takahe (Porphyrio hochstetteri) and Australasian harriers (Circus approximans) in New Zealand: a thesis presented in partial fulfillment of the requirements for the degree of Masters of Veterinary Science in Wildlife Health at Massey University, Palmerston North, New Zealand.

**Table 1. Effects of lead toxicity in physiological systems**

| Physiological system | Effects of lead toxicity | Reference |
|---|---|---|
| Neurological | Cell death of neurons, demyelination and delaying development of CNS in young animals | Anderson 1996 |
| Renal | Renal tubular cell damage and fibrosis, can lead to renal dysfunction and insufficience | Anderson 1996 |
| Circulatory | Interferes with haeme synthesis, reduce red cell production, increased red cell fragility | Patte and Pain 2003 |
| Immune | Reduces and modifies the number of antibodies present, can predispose animals to infectious disease | Parton et al 2001 |
| Gastrointestinal | Increase necrosis of the GIT mucosa, liver parenchyma damage | Patte and Pain 2003 |
| Reproductive | Decrease fertility, can cause developmental abnormalities | Hoffman 2003 |
| Skeletal | Bone cell dysfunction | Patte and Pain 2003 |

**Table 2. Categories for blood lead level (Stauber et al., 2010)**

| Blood lead level | Significance |
|---|---|
| 0.02mg/L | Background exposure, no clinical |
| 0.02-0.05mg/L | Subclinical |
| 0.051-0.1mg/L | Clinical lead toxicity |
| <0.1mg/L | Severe clinical poisoning |

**Table 3. Lead toxicity treatment drugs**

| Drug | Dose | Side effect |
|---|---|---|
| CaEDTA | 30-35mg/kg IM/SC BID for 5 days, 3 days rest | Nephrotoxic |
| Succimer | 20-40mg/kg PO BID for 7 days | Gastrointestinal disturbances |
| D-Penicillamine | 30mg/kg PO BID x 7 days | Gastrointestinal disturbances |

**Table 4. Biochemistry and haematology results. Reference values from ISIS**

| Test | Brown Teal male | Brown Teal female | Reference interval | Units |
|---|---|---|---|---|
| Aspartate aminotranferase AST | 16 | 17 | 0-85 | U/L |
| Bile Acid BA | <35 | <35 | n/a | Umol/L |
| Creatinine kinase CK | 434 | 825 | 0-1130 | U/L |
| Uric acid UA | 136 | 189 | 0-755 | Umol/L |
| Glucose GLU | 11.3 | 12.9 | 6.35-14.98 | mmol/L |
| Calcium Ca2+ | 2.76 | 2.76 | 2.12-2.93 | mmol/L |
| Phosphorus Phos | 1.36 | 0.79 | 0-1.82 | mmol/L |
| Total Protein TP | 52 | 53 | 35-64 | g/L |
| Albumin ALB | 24 | 21 | 10-29 | g/L |
| Globulins GLOB | 28 | 33 | 19-41 | g/L |
| Potassium K+ | 3.3 | 4.6 | 0.4-6.6 | mmol/L |
| Sodium NA+ | 139 | 141 | 132-164 | mmol/L |
| Packed Cell Volume PCV | 31 | 38 | 35.9-58.1 | % |
| Total Protein TP (refractometer) | 50 | 50 | 35-64 | g/L |
| White Blood Cell Count WBCC | 19.5 | 10.6 | 0-23.65 | $10^9$ cells/L |
| Heterophils | 15.6 | 6.04 | 0-10.03 | $10^9$ cells/L |
| Lymphocytes | 3.1 | 4.24 | 0-13.24 | $10^9$ cells/L |
| Monocytes | 0 | 0 | 0-868 | $10^6$ cells/L |
| Eosinophils | 80 | 0 | 0-589 | $10^6$ cells/L |
| Basophils | 0 | 0 | 0-678 | $10^6$ cells/L |
| Polychromasia | <10 | <10 | <5 | % |