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# Lead Levels in Backyard Chickens

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## Introduction

There has been an increase in the number of chickens (*Gallus gallus domesticus*) kept in the inner city urban areas of Sydney. These chickens are kept both for their egg production and as pets. They are commonly presented to Bird & Exotics Veterinarian Green Square for diagnosis and treatment as individual pets rather than on a flock basis.

High environmental lead has been reported to be present in Sydney urban areas built before 1970. Chickens from these urban areas form the majority of the chickens presented to Bird & Exotics Veterinarian. The source of the lead contaminating the soil of older urban developments comes from lead paint, car batteries, sump oil and leaded petrol (Mira et al., 1996). Chickens scratch in the soil as part of their normal foraging behaviour and therefore will consume the lead-contaminated soil.

## Methodology

Twenty-one chickens had blood lead levels measured over a two year period at Bird & Exotics Veterinarian. A positive result consisted of a blood lead level over 10µg/dl (0.483 umol/L). Blood samples were taken in lithium heparin and lead levels run on a Leadcare 2 blood lead analysis machine immediately after collection. The medical records of the chickens were examined retrospectively and the blood lead results were plotted on a map of Sydney. The map was then compared to a lead soil map of Sydney (Birch et al., 2010).

## Results

Of the 21 chickens tested, 18 had blood lead levels above 20µg/dl (0.966 umol/L) and three had levels below 10µg/dl. The majority of chickens with blood lead levels greater than 20µg/dl presented with non-specific signs of lethargy (10/18) or simply appeared quieter than normal. Four chickens with blood levels over 20µg/dl had the neurological (paralysis or abnormal gait, n=3) or gastrointestinal signs (vomiting, n=1) commonly associated with lead poisoning in parrots (Labonde, 1995).

One chicken had mild anorexia and one chicken presented for reproductive issues (laying soft eggs). Two chickens with no clinical signs were tested. One as its companion chicken had high blood lead levels and the other with a skin lesion was tested as the owner was concerned that there were high soil lead levels on their property.

The results showed that 100% of tested chickens that lived within 7 km of the centre of the City of Sydney and presented to Bird & Exotics Veterinarian, had very high blood lead levels (greater than 30µg/dl). All of these chickens lived in urban areas that had been built prior to 1940.

Three chickens resided 8-9 km away from the centre of Sydney. Two of the chickens came from a suburb that was built after 1940. One had the lowest of the positive blood lead results recorded (24µg/dl) and the other was negative. The third chicken in this group had a high positive (>70µg/dl) and was from a suburb built prior to 1940.

The three negative blood lead chickens came from suburbs outside this 10 km ring from the centre of Sydney and resided in suburbs that were developed after the 1960s.

The sample of chickens tested was representative of the geographical spread of all chickens seen at the veterinary clinic. Of the 21 chickens tested, five (29%) lived outside the 7 km ring. This is in comparison to the 88 new chicken patients seen during this two year period, of which 35% lived outside of this 7 km ring of suburbs.

Radiograph results were not included in the study as only a minority were radiographed.

Eight of the 18 blood lead positive chickens died or were euthanased within one month of diagnosis. In three cases clients elected for in hospital treatment with twice daily 40mg/kg CaEDTA injections. The majority (15/18) of clients elected to have their chickens treated as outpatients. All outpatient clients elected for once daily Ca EDTA injections at 80mg/kg, for convenience reasons, rather than

the recommended twice daily injections. The treatment failure rate in the outpatients was 53%.

## Discussion

Outdoor pet chickens living in particular Sydney suburbs require blood lead testing unrelated to the reason for presentation to the veterinarian. A chicken that lives within 7 km of the central business district of Sydney and presents to a veterinarian with non-specific signs of illness may have lead toxicosis. All these inner city suburbs were built prior to 1940 and all the chickens tested in these areas had high blood lead levels. The high blood lead levels in this area correlates with soil studies for many parts of this area which showed high soil lead levels (>400ppm) (Birch et al., 2010). It is also possible that chickens living in urban areas in other parts of Australia built before 1940 will have increased blood lead levels, and may, in some instances, have non-specific signs caused by lead poisoning.

Client health is an important consideration in cases of high blood lead levels in backyard urban chickens used for egg production. The ingested lead is absorbed in the intestinal mucosa and then stored in the liver, kidney and bone (Bakilli et al., 1995). For domestic layer hens the lead will also be preferentially deposited in the egg shell and yolk. Blood lead levels in chickens directly correlate with lead levels measured in the egg yolk and are also increased in the shell (Trampel et al., 2003). This correlation does not necessarily mean that ingested eggs will be toxic to humans, as that would depend on the number of eggs consumed and the weight of the owners. When high blood lead levels are present in chickens it is important to advise clients, especially those with children, of their possible exposure to lead. Exposure may be from their young children playing and eating the same soil as the chickens scratch in as well as the lesser possibility of exposure from eating contaminated eggs.

Lead affects multiple body systems leading to variable clinical signs. The absorbed lead competes with calcium causing neurological problems by blocking neuromuscular junctions. It may also result in axonal demyelenation. Lead decreases erythrocyte production and increases erythrocyte destruction leading to anaemia (Fudge, 2000). The classical signs of lead poisoning in birds are neurological and gastrointestinal, with some showing signs of lethargy (Dumonceaux and Harrison, 1994). However, the clinical signs seen in the chickens in this study were generally non-specific. Very few in the study showed the common indicators seen in parrots of either neurological or gastrointestinal signs. The results suggest that any chicken presented to a veterinarian with non-specific signs of illness that is kept in urban areas developed prior to 1940 should be assumed to have some form of lead poisoning, whether or not it is the primary cause of the illness for which the chicken is presented for.

The diagnosis of lead exposure or toxicoses in chickens is made on blood lead levels or kidney/liver tissue lead levels. Blood lead levels over 10µg/dl suggest a significant exposure to lead and is likely to be the primary cause of clinical signs in both humans and animals (Needleman and Landrigan, 2004). Levels above 14.5ug/dl and certainly over 25ug/dl in areas of high lead exposure confirm toxicoses in humans and animals. Radiology is non-specific and is unlikely to be useful as the sole method of diagnosing lead poisoning in chickens. Chickens, unlike parrots, are unlikely to break off small pieces of metal or paint from the house walls or cage wire that may be seen as radiodense particles on radiography. They are more likely to accumulate lead through foraging in the contaminated soil. This will not be seen as radiodense particles on radiographs. They may however, while foraging, peck and ingest non-specific radiodense rocks and non-heavy metal metallic objects. These non-toxic objects may be difficult to distinguish on radiographs from heavy metal fragments in the ventriculus. Clinical pathology is also non-specific. In some cases haematology may show a hypochromic, regenerative anemia and possible erythrocyte “ballooning”. Uric acid may also be increased on biochemistry (Fudge, 2000).

Treatment involves chelation with CaEDTA twice daily intramuscularly for a minimum of five days at 40mg/kg (Dumonceaux and Harrison, 1994). Hospitalisation and fluid therapy are often required. There was a poor response to treatment on chickens treated as outpatients on once daily 80mg/kg CaEDTA. This once daily treatment was often used to improve client compliance at home. Failure to respond to treatment may also be related to the continued exposure to contaminated soil during and after the treatment period and other contributing illnesses present aside from lead exposure. The use of CaEDTA or any other medication in individual pet chickens used for personal egg production, as with any medication in food producing animals, is regulated differently in the various states in Australia. Similarly, the use of CaEDTA on other chickens within the flock without examination of each individual chicken is also regulated and may be problematic, depending on the interpretation of the various states'' and territories legislation.

Prevention of further contamination by either removing the chickens from the contaminated soil or soil remediation is necessary. The addition of calcium to the diet has been shown to decrease the severity of clinical signs in chickens as calcium competes with the lead. Calcium additives cannot be used as the sole treatment method but are speculated to be useful as part of a long term amelioration of the clinical signs of lead exposure in a flock of chickens (Bakalli et al., 1995)

## Conclusion: Recommendations for chickens being kept in urban areas built prior to 1940

It is recommended that the soil in suburbs built before 1940 is tested for lead prior to chickens being introduced. If levels exceed 400ppm soil remediation should be performed. This may involve, at minimum, the removal of a layer of the topsoil and replacing it with uncontaminated soil.

Other general recommendations for high soil lead include:

- Test the chickens or their eggs for lead levels to assess the levels of exposure.
- Advise clients, especially those with young children, to seek medical advice on the risk of exposure to lead from both eating the eggs and contact with contaminated soil.
- Increase the calcium content of the chickens' diet by in-water or in-feed additives of calcium to decrease the clinical signs and absorption of lead.
- Assume all unwell chickens that live in areas of high soil lead have at minimum some exposure to lead when formulating treatment plans.

Chickens with high lead levels and clinical signs should be treated in hospital with fluid therapy and twice daily calsenate injections at 40mg/kg. Treatment may need to be continued at this level or possibly less frequently until chickens can be removed from the contaminated soil. Consult state authorities on the use of medication in production animals kept as individual pets.

A further study is recommended to assess the general blood lead level of both healthy and unwell chickens in inner city urban areas to determine the overall risk to pet chickens in older urban areas.

## References


Bakalli RI, Pesti GM, Ragland WL. 1995. The magnitude of lead toxicity in broiler chickens. Veterinary and Human Toxicology 37, 15-19.

Birch G F, Vanderheyden M, Olmos M. 2010. The nature and distribution of metals in soils of the Sydney estuary catchment (Australia). Water, air and soil pollution, DOI: 10.1007/s11270-010-0555-1

Dumonceaux G and Harrison GJ (1994) Toxins. In: Ritchie BW, Harrison GJ, Harrison LR, editors. Avian Medicine: Principles and Application. Lake Worth, Fl: Wingers pp 1030-1052

Fudge AM. 2000 Disorders of Avian Erythrocytes. In: Fudge Laboratory Medicine Avian and Exotic Pets pp 28-34.

LaBonde J. 1995. Toxicity in pet avian patients. Seminars in Avian and Exotic Pet Medicine 4, 23-31.

Mira M, Bawden-Smith J, Causer J, Alperstein U, et al. 1996. Blood lead concentrations of pre school children in Central and Southern Sydney. Med Janet 164, 399-402.

Needleman HL and Landrigan PJ. 2004. What level of lead in blood is toxic for a child? American Journal of Public Health 1, 8-9.

Trampel DW, Imerman P, Carson TL et al. 2003. Lead contamination of chicken eggs and tissues from a small farm flock. Journal of Veterinary Diagnostic Investigation 15, 418-422