Lead poisoning in Australian birds

Fact sheet

Introductory statement
Humans have found many uses for lead and have used it for many centuries. As a result, thousands of tons are mined and purified or recycled and used each year. As the result of its production and use, lead has and continues to enter the environment on a global and massive scale creating many opportunities for wildlife and human exposure. Lead has no physiological use in birds or other animals or plants. Exposure to high levels of lead is generally rapidly lethal, while exposure to low levels of lead can impact multiple body systems and cause chronic, typically subtle, effects. Mass mortality events in birds have been associated with the ingestion of spent lead shot and bullets and exposure to lead dust from industrial sources. Widespread environmental exposure to lead in lower concentrations is also occurring in a range of bird species. The impact on these species is more difficult to measure, but is likely to be significant.

Aetiology
Lead is a dense soft malleable metal with an atomic number of 82. It occurs naturally in deposits of galena (lead sulfide [PbS]), cerussite (lead carbonate [PbCO$_3$]), and anglesite (lead sulphate [PbSO$_4$]). Historically, lead has been used in pipes, paint, glass and as an additive to petrol. It still has many industrial and every day uses.

Natural hosts
Lead is toxic to all animals from microscopic plankton to birds and humans (Pokras and Kneeland 2009). Lead poisoning should be considered as a differential diagnosis in any mass bird mortality event and in individual birds exhibiting neurologic signs (DECWA 2007; Degernes 2008). Specific groups of birds, however, are more likely to become intoxicated with lead than are others. These include ducks, geese and swans that feed off the bottom of shallow water bodies, loons (Gavia spp.) (Degernes 2008; Porkras and Chaefel 1992; Schexnider et al. 2009) and birds of prey that feed on hunter-killed or injured waterfowl and other game species (Redig and Arent 2008; Guitart et al. 2009). Top order predators, for example seabirds, may also be at risk of exposure to lead. Lead concentrations in skeletal muscle of short-tailed shearwaters (mutton birds) was found to have lead concentrations that were 29% higher than considered safe for human consumption (Lavers and Bond 2013).
World distribution

Lead poisoning in birds is a problem throughout the world anywhere that lead ammunition is used for hunting, lead has been used in automobile fuel, lead is mined, lead is refined, and products containing lead enter the environment.

Distribution in Australia

Lead poisoning in waterfowl has been reported in Tasmania, Victoria, New South Wales, and the Northern Territory (Kingsford et al. 2012; Norman et al. 1993; Whitehead and Tschirner 1991; Wickson et al. 1992). Mass die offs of honeyeaters and purple-crowned lorikeets occurred in Esperance in 2006-2007 from lead intoxication (DECWA 2007). Elevated lead concentrations have been found in lorikeets in an urban area of Sydney (Rosenwax and Phalen 2009). The potential for lead poisoning in Australia is high at mine sites, along transportation corridors and transfer sites for lead ore, and refining sites for lead. It is also high in areas were hunters continue to use lead bullets or shot and in urban areas contaminated with lead from leaded fuel and paint.

Epidemiology

Sources of lead

There are predominately five sources of environmental lead from which birds, other wildlife, and humans can be exposed.

- Environmental contamination from mining sites, transportation of lead ore, and refining sites.
- Legacy/urban lead (lead present in soil and dust from the prior use of leaded petrol, lead balance weights on tires, and lead paint).
- Lead from run off from urban and other environments that accumulates in aquatic sediment and becomes increasingly concentrated in species that are higher in the food chain.
- Environmental contamination with spent lead shot from hunting waterfowl and other game birds.
- Lead shot and bullets in the tissues of waterfowl and larger game animals injured or killed by hunters.

Lead mining and processing: Australia has substantial deposits of lead that have been exploited for commercial use since 1841. The major currently operational mines in Australia are in Broken Hill, NSW, Mt. Isa, QLD and surrounds, and McArthur River, NT. Lead is also mined in Elura, NSW, Hellyer and Rosebery, TAS, Browns, NT and Goongewa, Gossan Hill, Pillara, and Scuddles, WA (Gulson et al. 2004; Mackay et al. 2013). Lead ore is processed to varying degrees of purity at mine sites and at Cockle Creek in Newcastle, NSW. Much of this lead is then further purified at Port Pirie, SA (Taylor 2011). Lead contamination of soil and increased blood lead levels in children have been documented in communities adjacent to the larger mines, some of the smaller mines (Gulson et al., 1994a, Gulson et al., 1994b, Gulson et al., 1994c; Gulson et al. 2004; Mackay et al. 2013; Munksgaard et al. 2010; Taylor et al. 2010a; Taylor and Schniering 2010) and at Mt. Isa, NSW, Newcastle, NSW and Port Pirie, SA where lead is refined (Baghurst et al. 1992; Dalton and Bates 2005; MCPHR 1999; Mizon et al. 2004; Taylor 2011; Taylor 2012; Taylor et al. 2013; Young et al. 1992). Run off from mines is also associated with contamination of the environment with lead and other metals (Mackay and Taylor 2013). The transfer of lead carbonate to ships in Esperance resulted in at least two episodes where lead containing dust was released into the marine sediment and into the surrounding town (Heyworth and Mullan 2009). The resulting environmental contamination caused the death of thousands of nectivorous birds.
(honey eaters and purple-crowned lorikeets) in two separate events and increased blood lead levels in children in the community (DECWA 2007; Gulson et al. 2009).

Legacy/urban lead: From 1932 until it was completely banned in 2001, in Australia, tetraethyl lead was added to automobile fuel (Kayhanian 2012; reviewed in The Lead Group 2011). Lead released in exhaust bound to dust particles and contaminated the soil adjacent to roads. Lead concentrations in soils immediately adjacent to these roads are high, but decline exponentially as one moves away from the road and by 50 metres minimal lead can be detected. In large and smaller urban areas road dust containing lead blows up against buildings where it settles, contaminating the soil around some of these buildings (Chattopadhyay, 2003; reviewed in Laidlaw and Taylor 2011). Paint containing lead has been used for many decades to paint the interior and exterior of homes until 1997, when the maximum amount of lead in paints sold in Australia was reduced to less than 0.1%. Weathered paint and paint scraped off older buildings has also been a source of soil contamination (reviewed in Laidlaw and Taylor 2011; reviewed in Wong et al. 2006). The impact of legacy lead on urban birds is not known. Ground feeding birds would be thought to be the most likely to be exposed to lead, but a study has found that urban rainbow lorikeets (Rosenwax and Phalen 2009), birds that do not come to the ground, often have high concentrations of blood and tissue lead. This suggests these birds are either being exposed by lead in dust, are consuming lead used in structures, such as solder, or that the plants that they consume contain lead (Kayhanian 2012; reviewed in Peralta-Videa, 2009).

Lead from runoff: Run off from roads and urban areas has resulted in significant amounts of lead entering water bodies. Most lead is found in the sediment but some enters the food chain. It has significant impacts on shellfish and phytoplankton (Kayhanian 2011). Concentrations increase the higher the animal is in the food chain and maximum concentrations are found in top order predators such as pelagic species of birds and marine mammals (reviewed in Wong et al. 2006).

Spent shot: Lead shot has been used to hunt waterfowl and other game birds for over 100 years in Australia and around the world, resulting in massive amounts entering the environment (Newth et al. 2013). Lead shot is consumed by water birds, predominately swans, geese, and ducks, when they are feeding in shallow water bodies. The acid in the bird’s stomach solubilizes the lead and allows it to be absorbed into the body. Lead weights used for fishing also are sources of ingested lead, particularly in loons in the Northern hemisphere (Porkras and Chaefel 1992). It is no longer legal to use lead shot to hunt ducks in New South Wales, Victoria, South Australia or Tasmania and duck hunting is banned in Queensland and Western Australia. Lead shot can still be used, however, to hunt quail. Even though the use of lead shot has been banned in many parts of the world, spent shot still remains in water bodies frequented by birds. Drought brings water levels down allowing access to lead shot and in some countries it is very common for outbreaks of lead poisoning in water fowl to occur under these conditions (Degernes 2008).

Lead shot and bullet fragments in tissues of game animals: Not all animals shot with lead pellets or bullets die or are retrieved by hunters. Some are crippled and these are often killed and eaten by birds of prey and those that die and are not picked up by the hunters can be scavenged. These birds then ingest the shot or fragments of it and become lead poisoned (Redig and Arent 2008; Nadjafzadeh et al. 2013). In larger species that are hunted in Australia such as pigs, deer, and kangaroos, lead bullets are used. When these bullets strike the animal they typically break into many fragments. Animals may be wounded and subsequently die but not be found by the hunter or parts of the animal containing lead can be left in the field after the killed animal has been field dressed. These then can be consumed by predators and the predators can develop lead toxicity. Ravens and crows, wedge-tailed eagles and kites have all been seen to consume tissues from field dressed kangaroos (Read and Wilson 2004).
Impact of internalised lead

Lead can enter the body by ingestion, inhalation, or through contact with the skin depending on its chemical formulation. It has impacts on multiple systems including the central and the peripheral nervous system, kidneys, the integrity of the vascular system, the synthesis of haemoglobin and cellular energy production. It can also cause abortions in both humans and animals. For a more detailed discussion of the biochemical impacts of lead the readers are referred to Godwin (2001), Taylor et al. (2010b) and Toscano and Guilarte (2005).

Clinical signs

In many cases where there is widespread environmental contamination the first indication of lead intoxication is the presence of many dead birds (DECWA 2007; Degernes 2008).

The most common signs of lead intoxication in all species are caused by its effect on the nervous system. The impact on the central nervous system results in altered mentation, changes in responsiveness to environmental stimuli, loss of balance, the inability to stand and seizures that may be brought on with stimulation. Damage to the peripheral nerves results in weakness, especially of the legs (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008; Toscano and Gu¨ilarte 2005).

Damage to the nerves of the digestive tract stops its motility and as a result birds stop eating and may vomit. Faeces become dark green because they only contain bile pigments and not food. Damage to the kidney interferes with the bird’s ability to concentrate urine and as a result, droppings may be watery (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008). Ducks, geese, and swans can develop swelling (oedema) of the subcutaneous tissues of the head and neck (Degernes 2008).

Lead damages red blood cells causing their premature destruction resulting in increased pigment (biliverdin) release into the urine staining the urates green or yellow (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008). In at least one native Australian species, the galah (Lightfoot and Yaeger 2008), haemoglobin released from the breakdown of red blood cells can be found in the urine and will stain the urates light red to pink. When red blood cell destruction is severe, birds will be anaemic and thus appear pale (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008).

Birds exposed to high concentrations of lead will develop signs quickly and may still be in good body condition. Other birds exposed to lower concentrations will present with severe weight loss and muscle atrophy. Excellent reviews of signs of lead poisoning in waterfowl (Degernes 2008), birds of prey (Redig and Arent 2008) and pet birds (Lightfoot and Yaeger 2008) are available for more detail on this topic.

A syndrome seen in birds of prey (McLelland et al. 2012) around the world and in rainbow lorikeets in Australia (Rosenwax and Phalen 2009) is characterized by clenching of the feet and inability to extend the hocks. A study in New Zealand in Marsh Harriers (McLelland et al. 2012) found that these birds had elevated blood and tissue lead. However, other Marsh Harriers not exhibiting these signs also had elevated lead levels and affected birds did not respond to treatment for lead poisoning and microscopic lesions for lead poisoning were not found. Similarly, elevated blood lead and tissue concentrations have been found in lorikeets with this syndrome, but also in birds not showing signs and these lorikeets with clench-claw syndrome do not respond to treatment for lead poisoning. Given current knowledge of clench-claw syndrome, it is unlikely that it is caused by lead poisoning alone or possibly at all.
Imaging

Finding ingested lead or lead containing objects in the grinding stomach (ventriculus) of a bird with signs consistent with lead poisoning is diagnostic. The most common lead object found in wild waterfowl and birds of prey is lead shot (Degernes 2009). The absence of lead in the stomach, however, cannot be used to rule out lead intoxication. Impaction of the proventriculus can be seen radiographically in some waterfowl with lead poisoning. The absence of motility of the digestive tract is manifest in some cases by the presence of gas in the digestive tract and dilation of the intestines.

Clinical pathology findings

Complete blood count: Anaemia is a hallmark change in birds with lead poisoning but will be variable in degree depending on the duration of the intoxication. Birds that are suddenly exposed to high concentrations of lead may not be anaemic. Birds that survive a few days will have a mild anaemia with a disproportionately large number of immature red blood cells in circulation. Birds that have chronic lead poisoning will be severely anaemic and have a low blood total protein. Increases in inflammatory cells and toxic changes in them may also be seen (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008).

Biochemistry: Blood biochemical changes are not specific. Uric acid levels may be increased if the birds are in kidney failure. Liver and muscle enzymes will be increased if there is liver and muscle cell injury (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008). Electrolytes (sodium, potassium, and chloride) are expected to be low as the result of decreased food intake, but could also be elevated in a dehydrated bird.

Blood lead levels: Detecting lead in the blood is the definitive way of diagnosing lead exposure and poisoning in birds. Lead should not be present in the blood of birds. Birds exhibiting signs of lead poisoning with have blood lead concentrations in excess of 0.5 ppm, but lead poisoning should be suspected in birds with blood values in excess of 0.2 ppm (Lightfoot and Yaeger 2008).

Identification of the source of lead: In Australia, the source of the lead in a bird or in the environment can be determined by stable isotope analysis (Gulson 1985).

Pathology

Gross lesions: Gross lesions in birds that die of lead poisoning are generally not specific or are absent completely. If the dose of lead is high, birds may die quickly and still be well muscled with adequate body fat. Those that die more slowly will be thin and have little or no body fat. Waterfowl may have oedema of the head and neck and/or dilation of the end of the oesophagus and the first part of the stomach (proventriculus) (Degernes 2009). The stomach may be impacted with food. In all species that die with kidney failure secondary to lead poisoning, the kidneys may be white and moderately swollen because of diffuse deposition of uric acid. Pale streaking (degeneration) of the heart muscle may occur. Finding ingested lead, most commonly spent ammunition, or materials containing lead in the stomach is highly suggestive of lead intoxication in any species of bird (Degernes 2008; Redig and Arent 2008).

Microscopic lesions: Microscopic lesions may not be present in birds that are intoxicated with high dosages of lead and die quickly. Birds that die more slowly may exhibit death of portions of the kidney (proximal tubules). A special stain, known as the acid fast stain, can be used to identify intra nuclear inclusions in proximal tubule cells, however, these inclusions are often not present in lead poisoned birds. If the bird is in kidney failure, crystalline deposits of uric acid may also be found in the kidney. As lead poisoning causes an
increased rate of red blood cell destruction, there are often increased amounts of iron pigment in the phagocytic cells in the spleen and liver. Multiple focal areas of cell death can also occur in the liver. Death of the cells of small blood vessels is sometimes seen in the heart and in other organs. In some waterfowl this causes bleeding into the intestinal tract. When it occurs in the heart it results in death of surrounding heart muscle fibres. Changes in the nervous system can also be found. Cells (Schwann cells) surrounding the cell process (axons) that connect nerves are damaged and the insulating material that they produce (myelin) breaks down with resultant death of the axons (Schmidt et al. 2003).

**Differential diagnosis**

Nutritional diseases such as thiamine, vitamin E, and calcium deficiencies can also cause neurological signs that could resemble acute lead intoxication in an individual bird (Klasing 2008).

Several intoxicants, including arsenic, mercury, zinc, botulism toxins C and E, organochlorines, organophosphates, and carbamates can also cause similar signs to lead poisoning, or cause significant mortality events (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008).

Infectious agents that diffusely infect the brain or spinal cord including the avian influenza, Exotic Newcastle Disease, and West Nile viruses can also cause similar neurologic signs. In North American Canada geese (*Branta canadensis*), trumpeter (*Cygnus buccinator*) and mute (*Cygnus buccinator*) swans, snow (*Chen caerulescens*) and Ross’s geese (*Chen rossii*) and rarely in birds of prey, avian Bornavirus can cause both central nervous system signs and gut stasis that could readily be mistaken for lead poisoning (Delnatte et al. 2013). There are also numerous insect-borne viruses found in Australia and other parts of the world that have the potential to cause neurologic signs in birds. Salmonella DT 160 often causes neurologic signs in house sparrows (*Passer domesticus*) (Alley et al. 2002) as does systemic salmonellosis in pigeons (Schmidt et al. 2003). The rat lung worm (*Angiostrongulus cantonensis*) can also cause neurologic signs, but infection is not seen in species typically impacted by lead poisoning (Monks et al. 2005).

Traumatic injuries to the brain and spinal cord can also result in central nervous system signs.

**Laboratory diagnostic specimens**

**Live bird:** Whole blood (200 µl or more) should be collected into a heparinised plastic container. Multiple commercial diagnostic laboratories provide blood lead testing in animals. If there is a need for testing humans, a physician or the state Department of Public Health should be contacted. Blood aminolevulinic acid is also increased in birds with lead poisoning and it has also been used as a marker for lead poisoning (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008). Hair has been used to trace previous exposure to lead (Gulson 2008).

**Dead bird:** Liver, kidney, and bone are the three most commonly used tissues to determine if a bird has been exposed to lead. In cases of acute lead poisoning concentrations will be higher in soft tissues and concentrations will be higher in kidney than in liver. With chronic poisoning, the highest concentrations will be found in bone (Gulson 2008). Skeletal muscle concentrations can also be determined to verify that meat is safe to eat. Skeletal muscle concentrations will me much less than those found in liver, kidney and bone (Lavers and Bond 2013).
Treatment

Treatment can be divided into three elements, binding (chelating) lead in the body so that it can be excreted, supporting the bird until it has recovered from signs caused by lead intoxication, and eliminating lead from the digestive tract, if it is present, to prevent additional uptake.

Chelating lead: Chelators are compounds that tightly bind lead allowing it to be excreted from the body. Calcium ethylenediaminetetraacetic acid (EDTA) is an injectable drug that is the treatment of choice in the initial phases of treatment of lead poisoning when oral medications cannot be administered because of paralysis of the digestive tract. A dosage rate of 50 mg/kg given by intramuscular injection every 12 hours is recommended. Some authors recommend treating for 5 days and then stopping treatment for 3 days and repeating the five day treatment in cycles until blood lead levels are below toxic concentrations. Dimercaptosuccinic acid (DMSA) is a chelating agent that can be given orally and can substitute for EDTA once the motility of the digestive tract has been re-established. It is given at 25 to 35 mg/kg, twice daily for 5 days followed by 2 days off and the cycle repeated for 3 to 5 weeks. DMSA treatment has been combined with EDTA treatment and found to result in a more rapid drop in blood lead levels (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008). Another chelating agent is D-penicillimine. Though less frequently used, protocols for water-based treatments using D-penicillimine to follow up initial EDTA treatments have been developed and these are useful when multiple birds are being treated or it is difficult or too stressful for the bird to administer treatment orally or by repeated injection.

Supportive care: Birds presenting with lead poisoning are usually dehydrated and generally are unable to drink, so they must be given appropriate fluids either intravenously or subcutaneously until their digestive system is functioning again. Tube feeding is often necessary for a few days once the digestive begins to function as these birds may not eat on their own.

Removing lead from the ventriculus: Aggressive oral treatment with psyllium, paraffin oil, or peanut butter may help to push pieces of lead out of the ventriculus and allow it to be passed. The success of this treatment may depend on the species of bird being treated and not all authors have found these treatments to be effective (Degernes 2008; Lightfoot and Yaeger 2008; Redig and Arent 2008). Gastric lavage has also been used to remove lead in waterfowl (Degernes 2008).

Prevention and control

Strict rules are in place to prevent lead contaminated dust from mining, transportation, and refining operations from entering the surrounding environment and monitoring of these environments is done (Elias and Gulsom 2003). Closure of smelting plants has also resulted in reduced rates of lead exposure to children (Dalton and Bates 2005). A review of remediation practices that are available for lead contaminated environments is available (Elias and Gulsom 2003).

Leaded fuels are no longer used and lead concentrations in the soil around roads have decreased dramatically (Kayhanian 2012). Lead has been eliminated from household paint and this is reducing lead release into the environment and opportunities for children to eat flaking lead paint. Guidelines are available on how to safely prevent lead contamination of the environment if one is working with interior or exterior surfaces that are were painted with lead-based paint (EPA NSW 2003).

The ban on using lead shot for the hunting of waterfowl in North America and other countries has resulted in a marked decrease in lead poisoned waterfowl and also lead poisoned birds of prey in some countries, but
not in others (Newth et al. 2013). It is likely that it is having a similar impact in Australia. There is a strong push to replace the use of lead bullets used to hunt larger species of animals in North America and in other countries. An example of a replacement for the lead bullet is the copper expanding bullets made by Barnes Bullet Company (Lindon, Utah, USA). These bullets are available from multiple distributors in Australia (Field and Game Australia 2007).

Fishing remains a popular sport in Australia and contamination of water bodies with lead sinkers continues. Alternate sinkers including those made with tin, bismuth, tungsten, and clay have been developed but are yet to be accepted widely and are not readily available in Australia.

**Surveillance and management**

There is an increasing body of knowledge about the distribution of lead contamination in Australia and where the problem is worst, children in these communities are routinely tested for lead exposure and treated as needed (Taylor 2011; Taylor et al. 2012). Recent studies, however, suggest that lead exposure, particularly in urban areas, may be more widespread than previously thought and there has been a call for increased environmental and human screening (reviewed in Laidlaw and Taylor 2011; reviewed in Wong et al. 2006). The Esperance example suggests that sampling birds may also prove to be a useful means of monitoring environmental lead levels (DECWA 2007).

**Statistics**

Northern Territory: Whitehead and Tschirner (1995) report that 21.4% of magpie geese (Anseranas semipalmata) sampled (n = 103) had ingested lead shot and 45.2% (n = 65) had elevated concentrations of lead in their livers.

New South Wales: Kingsford et al. (1994). Found elevated concentrations of lead in a Pacific black duck, four grey teal, and an Australian shelduck (Tadorna tadornoides) out of 326 ducks sampled at multiple sites across New South Wales.

Victoria: Wickson et al. (1992) found 21% of Pacific black ducks (Anas superciliosa) collected to have evidence of lead exposure. In a second study by Norman et al. (1993) in Victoria elevated lead levels were found in 2.2% of bone samples from Maned ducks (Chenonetta jubata), Hardhead (Aythya australis), 5.6% of liver samples from Black swans, 1.1% of bone samples from Australian shelducks, 7.5% of samples from bone and 4.6% of samples from liver from Pacific black ducks, 24% of bone samples and 23.1% of liver samples from blue-billed ducks (Oxyura australis) and 50% of bone and 30.4% of liver samples from musk ducks (Biziura lobata).

Tasmania: Smith et al. (1995) found elevated blood concentrations in 17% of black swans and elevated liver and bone concentrations in approximately 50% of liver and bones samples examined.

Western Australia: In December 2006, an estimated 4000 birds died as the result of widespread lead carbonate contamination of the town of Esperance. The majority of which were nectivorous species (honeyeaters, miners and wattlebirds) but other passerine species were also affected. The contamination occurred during the transfer of the lead carbonate from train cars to ships. In March 2007, an estimated 200 purple-crowned lorikeets also died from lead carbonate exposure when another contamination event occurred in Esperance (DECWA 2007).
Wildlife disease surveillance in Australia is coordinated by Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia's States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au.

Research

The impact of lead poisoning in Australian birds is really not known. Studies on population numbers and tissue levels of lead in native birds need to be done in all locations where lead is mined, smelted or transferred from one transportation system to another. The finding of lead in rainbow lorikeets in the city of Sydney also suggests that other species of birds in urban environments may also be exposed and merits additional investigation (Rosenwax and Phalen, 2009).

Human health implications

Lead poisoning is a health hazard for Australians and people around the world. It is of particular concern in children because they are most likely to ingest lead by contact with contaminated dust, to ingest lead paint and because of their increased metabolic rate (Baghurst et al., 1992, Hunt et al., 2006, Jarup 2003 Layton and Beamer, 2009; Laidlaw and Filippelli, 2008, Moya et al. 2004; Weiss 2000). Children are also the most susceptible to the impacts of lead poisoning. There is no safe level of lead and even very low lead levels may have some negative impacts on the foetus and growing animals and children (Landrigan 2000; de Burbure et al. 2006). Most work has been done in people, where it has been shown that low level lead intoxication causes a decrease in IQ and is associated with an increased probability of developing behavioural problems, mental illness and mental retardation in children (Bellinger 2003, Bellinger 2008, Braun et al. 2006, Jusko et al. 2008; Jedrychowski et al. 2008, Jedrychowski et al. 2009; 2000; Lanphear et al. 2005; Liu et al. 2010; Taylor et al. 2012).

In adults, low levels of lead intoxication have been associated with increased risks for stroke and heart attack, decrease in fertility, acceleration of cognitive decline in the elderly, cataracts, and increased mortality rates (Telisman et al. 2007; reviewed in Toscano and Guilarte 2005). It has also been shown to be carcinogenic in laboratory animals, to negatively impact the immune response and increase the risk of renal disease in humans. High lead exposure has been associated with hypertension, diabetes, gastrointestinal problems and neurological effects in humans (reviewed in Toscano and Guilarte 2005).

Conclusion

Lead contamination of the Australian environment occurs at lead mining and processing sites, in urban environments, from urban runoff into marine and freshwater ecosystems, and as the result of hunting and fishing activities. Concerted efforts have been made to reduce the amount of lead that enters the environment, but release of lead still continues and, because lead is not biodegradable, legacy lead will continue to be a source of wildlife and human exposure indefinitely. Impacts of acute lead poisoning on birds have been documented in Australia in ducks and geese and in nectarivorous birds. Whether acute lead poisoning is occurring more often in birds who consume hunter-killed carcasses is not known. There is also evidence that chronic lead poisoning is occurring in urban rainbow lorikeets and could be occurring in other
species. What impact this has for these species and what it indicates about the extent of lead contamination of the Australian environment merits further investigation.

Acknowledgements

This fact sheet was produced by Nerida Sweet and David Phalen of Sydney University.

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Updated: 25 Mar 2014.

To provide feedback on this fact sheet

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

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