Common Diseases of Urban Wildlife

REPTILES

MISSION STATEMENT:
The Australian Registry of Wildlife Health is committed to contributing to the preservation of Australia’s biodiversity through increased understanding of the interactions among animals, the environment, and disease causing agents.
1 Common Diseases of Reptiles

1.1 Introduction

Lizards, snakes and turtles are commonly admitted to urban wildlife care centres. These reptiles are almost uniformly admitted due to traumatic injury. Infectious disease and clinically apparent parasitic diseases in urban reptiles are uncommon.

1.2 Parasitic Disease

Reptiles may be infested with a wide variety of ectoparasites, primarily mites and ticks. Although infestations are not often directly related to disease, haematophagous arthropods are capable of transmitting viruses, bacteria, haemoprotozoa and microfilaria.

Mites and ticks are usually evident upon close visual inspection between the scales in the region of the head and neck. Mites are most easily identified in the periocular and mandibular scales. Mites are eliminated through a combination of environmental decontamination, and treatment of the reptile with suitable parasiticides.

Ticks of the genera *Amblyomma* and *Aponomma* are most commonly found infesting reptiles (McCracken, 1994). Large tick burdens may result in anaemia. Treatment of tick infestation is usually accomplished by manual removal of the tick. Alternatively, antiparasitic agents may be used to treat tick infestations. Of other ticks reported to infest reptiles, the genus *Hyalomma* does not occur in Australia. The original record (Cleland 1910) was from northwest WA and thought to be introduced from India on camels. No species of this genus has since been seen in Australia (David Spratt, personal communication).

Many endoparasites also infect reptiles. *Strongylurus paronai* is a gastric roundworm of bluetongue lizards, water dragons and frilled lizards (Griffiths et al, 1998). In dead animals, this parasite often crawls into the pharynx and oral cavity. The interpretation of faecal floatation’s in reptiles must be undertaken with care, since parasite ova of the snake’s prey are often found.

Haemoprotozoa and microfilaria are common incidental findings in injured reptiles. Haemogregarina species, Trypanosomes, *Haemoproteus* spp. and *Plasmodium* spp. are frequently found during haematological examinations.
Haemogregarine parasites have been identified within the pulmonary parenchyma of a taipan, diamond python, carpet python, and several snakes and monitors seized by Australian customs service officials upon illegal entry into Australia. Mosquitoes and mites are the arthropod hosts most likely to transmit haemogregarines; however, leeches, ticks and other haematophagous arthropods may act as intermediate hosts. These intermediate hosts release sporozoites during a blood meal. Sporozoites enter erythrocytes and undergo schizogony in various tissues throughout the body. Merozoites and gametocytes are also found within the erythrocyte and are ingested by haematophagous insects to allow subsequent transmission of the parasite.

Microsporidia are protozoal parasites that have been detected within necrotising lesions in the muscle of a yellow-bellied sea snake, desert death adder, eastern water dragon, and central knob-tailed gecko. These organisms have also been identified within granulomatous lesions in the ovary of an eastern water dragon and Central knob-tailed gecko (Reece and Hartley, 1994, ARWP). Microsporidia appear as clusters of basophilic oval to round bodies when viewed in tissue sections stained with haematoxylin and eosin. Microsporidia are gram positive. Mature spores are acid fast, and contain a polar granule that stains positively with periodic acid-Schiff (PAS) staining protocols.

*Cryptosporidia* spp. are coccidian parasites that have been identified along the gastric brush border in captive red-bellied black snakes, a Stimson’s python, a taipan, and a tiger snake. Cryptosporidiosis is characterised by hypertrophic gastritis. Clinical signs associated with cryptosporidiosis include weight loss, regurgitation, diarrhoea and death. The parasite undergoes asexual and sexual reproduction within the host cell cytoplasm along the mucosal brush border. The oocysts sporulate *in situ*, resulting in continuous self-infection. Ante-mortem diagnosis can be achieved through demonstration of oocysts within modified acid fast stained faecal smears. The sensitivity of faecal staining tests is increased through serial testing and centrifugation techniques that concentrate the oocysts. There is no known effective treatment for cryptosporidiosis.

Epizootics of neurologic dysfunction and mortality in green turtles have occurred in subadult to adult was identified along the east coast of NSW and Queensland on several occasions. Affected turtles are often found circling in estuaries, rolling in the surf, or stranded on the beach moribund or with a head tilt. These epizootics have been attributed to disseminated coccidiosis, characterised by the presence of necrosis and non-suppurative inflammation in the intestinal tract, renal interstitium, thyroid gland
interstitium, and throughout the parenchyma of the brain, caused by *Caryospora cheloniae*. Each epizootic has been associated with El Nino conditions and very low inland and coastal rainfall (Rose et al, 2003).

Coccidia harvested from the intestinal tract or faeces can be cultivated in filtered seawater, where they develop into the stellate sporulation pattern pathognomonic for *Caryospora cheloniae*. A diagnosis of systemic coccidiosis can also be made when sporozoites are identified within circulating monocytes within a blood film or buffy coat preparation.

*Caryospora* spp are coccidian parasites that can be found within the intestinal lamina propria and mucosa of carnivorous reptiles, but these are generally incidental findings. *Caryospora* spp. can be found infecting reptiles, birds and rodents and can have a single host, or two host (predator-prey) lifecycle. The intestinal forms of *Caryospora* spp. are characterised by a single sporocyst containing eight elliptical sporozoites.

Gastric ascarid infections with *Ophidascaris* spp. are commonly seen in free-ranging snakes. These nematodes are often found in clusters, with their heads burrowed deep into the intestinal wall. The parasites tend to drag bacteria into the deeper tissues, thus, septic granulomas within the gastric wall or other tissues where the parasites have migrated are not uncommon.

### 1.3 Nutritional Disease

#### 1.3.1 Nutritional Osteodystrophy

**Aetiology**

Nutritional osteodystrophy, or metabolic bone disease, is characterised by either failure to mineralise a growing skeleton, or demineralisation of a mature skeleton. This condition is seen in native reptiles that have been collected from the wild and held as pets. Nutritional osteodystrophy occurs primarily in reptiles that have been on a long-term diet deficient in calcium or containing excessively high concentrations of phosphorus. Osteodystrophy may also occur when reptiles have had insufficient dietary vitamin D3 and no exposure to the ultraviolet rays required to produce metabolically active vitamin D.

Plant derived vitamin D2 (ergocalciferol) is not considered to be metabolically active in reptiles. The active form of vitamin D in reptiles is vitamin D3 (cholecalciferol). Vitamin D3 must be supplied in the
food, or the animal allowed access to ultraviolet light to convert vitamin D$_2$ to vitamin D$_3$ in the skin (Boyer, 1996).

Ideally, reptile diets should contain a 2:1 ration of calcium to phosphorus. Lean beef meat contains a ratio of approximately 1:16 Ca:P, and beef heart contains approximately 1:38 Ca:P. Feeding the insects a calcium rich diet two to three days prior to feeding them to reptiles prevents nutritional osteodystrophy in insectivorous species. Additionally, insects may be dusted with a supplement containing calcium carbonate immediately prior to being fed to reptiles.

**Clinical signs**

Reptiles with nutritional osteodystrophy have soft, misshapen bones. Lizards with osteodystrophy have an abnormal posture, since they are unable to lift their trunk. The mandible is pliable, and as the lesion becomes chronic, muscle traction results in shortened and bowed mandibles. The long bones, particularly the femurs, are often very swollen due to periosteal thickening around a thin, weakened cortex. The animal may have kyphosis, lordosis, scoliosis, or vertebral compression fractures. If spinal cord injury accompanies vertebral fracture, the reptile will have rear limb paresis or paralysis. Radiographically, affected lizards have a diffusely reduced bone density. The cortical shadow may appear thin, or may be very thick, due to fibrous tissue proliferation. Folding fractures of the long bones or compression fractures of the spine may also be evident on radiographic examination.

Chelonians with nutritional osteodystrophy have a soft, misshapen shell with upturned marginal scutes. The lesions are most severe if they occur in a young reptile that has not yet mineralised its skeleton. The carapace may sag centrally, scutes become uneven, and the shell may be too small in comparison with the rest of the body. Radiographically, these chelonians have a reduced bone density and porous shell, and may have pathological long bone fractures.

**Diagnosis**

Nutritional osteodystrophy is diagnosed by visual inspection and palpation of the skeleton in conjunction with radiographic examination. Serum calcium and phosphorus concentrations are often normal.

**Treatment**

Due to extensive bony deformity and the extensive time in a captive environment, some reptiles with osteodystrophy will not be suitable for release. When the lesions are mild, reptiles are often treated with
parenteral calcium and possibly also vitamin D₃. The diet must be corrected to include a 2:1 ratio of calcium to phosphorus. Ultraviolet light should be provided through exposure to sun, or a broad-spectrum artificial light placed within 60 cm of the reptile, without any filtration through glass or plastic (Boyer, 1996). Response to therapy should be monitored through radiographic examination every four to six weeks.

Internal fixation of pathological fractures most often results in further splitting of the fracture site, especially in small reptiles. Cage rest and an external splint will often usually result in satisfactory resolution of fractures in reptiles with osteodystrophy.

1.4 Fungal diseases

1.4.1 Mycotic and other dermatoses

Mycotic infection of the skin is common in reptiles, especially lizards. Organisms include *Basidiobolus* spp, *Geotrichium* spp., *Paecilomyces* spp., *Trichphyton* spp., and *Aspergillus* spp. (Mc Cracken, 1994). Typically the fungi invade opportunistically, when the skin is damaged, or macerated; therefore infection is more common in animals from habitats or enclosures that are damp and have limited sunlit areas. Although this is most common in captive reptiles, the condition can occur in free-ranging animals in unsuitable habitats or exposed to concurrent disease or other stressors. Often, infection of bluetongue skinks by dermatophytes such as *Trichphyton terrestre* is associated with dysecdysis, and this can lead to strangulation and sloughing of the digits or feet.

*Dermatophilus congolensis* is another common invader of the skin of reptiles, especially bearded dragons. Hyperkeratotic lesions may be superficial, but these sometimes obscure subcutaneous abscesses. As with fungal infection, these are typically associated with animals kept in damp or humid conditions, or under cool conditions where there is insufficient access to heat sources. Dermatophilisis can be identified within gram stained skin scrapings or biopsies. The organisms can appear as cocci or as beaded, branching double chains of cocci (as pictured above). *D. congolensis* required extended periods (up to 14 days) to grow in anaerobic culture. The infection is often treated with topical iodine preparations and parenteral long-acting, broad spectrum antibiotics, along with improvement of habitat and reduction in environmental stressors.
1.5 Traumatic Injury

1.5.1 Soft Tissue Injury
Soft tissue injury in reptiles is often inflicted by predators, lawnmowers, or vehicles. Careful examination is required to assess the degree of damage sustained. Healed wounds should be monitored to ensure that the scar tissue does not impede ecdysis.

Cloacal or penile prolapse
Chelonians and lizards that experience severe blunt trauma to the lumbosacral region may develop cloacal, colonic, urinary bladder, oviductal or penile prolapse. Prolapse of the hemipenes is also seen in snakes that receive crushing injuries to the caudal body. Prolapse of these organs in captive reptiles usually occurs secondary to enteritis, urinary calculi formation, or inflammation within the reproductive tract.

Bite wounds
Bite wounds are heavily contaminated with bacteria. Many reptiles will become septicaemic.

1.5.2 Shell Injury
Shell abrasions and erosions are a common finding in debilitated or injured chelonians. These wounds may be the result of traumatic injury, such as predation by canids, or infection. Shell erosions are often covered by a tan or green exudate. Swabs from the wound should be collected for direct microscopic examination, cytologic examination, and microbial culture. When the shell is damaged, any necrotic scutes and underlying necrotic bone should be debrided (Barten, 1996). Old shell injuries may have an exposed core of necrotic bone. Although the bone may not appear infected, the deeper tissues lining the sequestrum often are infected.

1.5.3 Shell Fracture
Shell fractures in chelonians are most often inflicted by vehicles, lawn mowers and canids. Injury sufficient to fracture the shell is usually accompanied by other traumatic injuries. The chelonian may be suffering concurrent long bone fractures, shock, internal haemorrhage and pulmonary contusion. Radiographic examination of the reptile should be undertaken to evaluate the full extent of the injuries.
Anterior-posterior, lateral and dorso-ventral views will provide more detailed information regarding the location and extent of injuries.

1.5.4 Tail injuries

The tail muscles of many skinks are arranged in compartments so that the tail can easily break off and heal if the animal is attacked by predators. These tail wounds often heal well if just kept clean. The tail will usually regrow, but not necessarily to the original length or shape.

2 Animals mentioned in text

2.1 Reptilia

Coastal Taipan (*Oxyuranus scutellatus*)
Inland Taipan (*Oxyuranus microlepidota*)
Diamond python (*Morelia spilota spilota*)
Carpet python (*Morelia spilota variegata*)
Stimson’s python (*Liiasis stimsoni*)
Tiger snake (*Notechis scutatus*)
Red-bellied black snake (*Pseudechis porphyriacus*)
Desert death adder (*Acanthophis pyrrhus*)
Yellow-bellied Sea-snake (*Pelamis platurus*)
Eastern water dragon (*Physignathus lesueurii*)
Central knob-tailed gecko (*Nephrurus levis*)
Green turtles (*Cheloniae mydas*)
Eastern Bluetongue Skink (*Tiliqua scincoides scincoid*)
Frilled Lizard (*Chlamydosaurus kingii*)
Central Netted Dragon (*Ctenophorus nuchalis*)
Hosmer’s Skink (*Egernia hosmeri*)
Mitchell’s Water Monitor (*Varanus mitchelli*)
Olive Python (*Liiasis olivaceus*)
Corn Snake (*Elaphæ guttata*)
3 References


