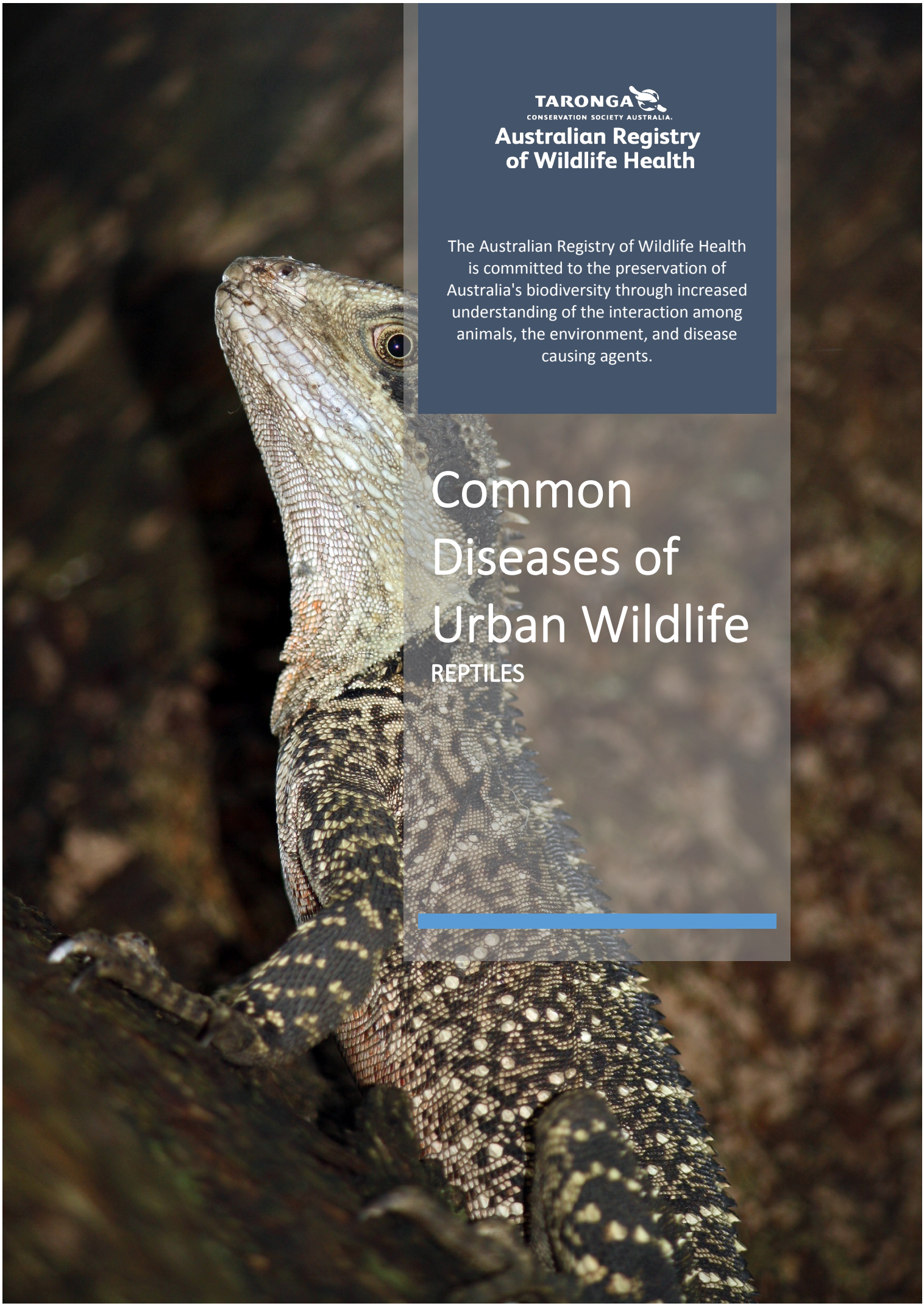

TARONGA
CONSERVATION SOCIETY AUSTRALIA.

Australian Registry of Wildlife Health

The Australian Registry of Wildlife Health is committed to the preservation of Australia's biodiversity through increased understanding of the interaction among animals, the environment, and disease causing agents.

Common Diseases of Urban Wildlife

REPTILES

Australian Government

Production of this document was made possible by: Wildlife Rescue and Rehabilitation – an Australian Government initiative

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Contents

1 List of Images

2 Introduction

A variety of diseases have been recognised within free ranging Australian reptiles. The purpose of this document is to review the diseases that occur often within particular species or taxonomic groups of reptiles. We hope that this information assists with the timely recognition of common parasites, microbes, intoxicants and injuries to accelerate the appropriate care and welfare of wild animals. Throughout the text we offer advice towards achieving a diagnosis. As best-practice wildlife treatments change rapidly over time, treatment of reptiles in a rehabilitation situation should be made in consultation with a veterinary professional.

A notifiable disease is one that must be reported to agricultural authorities. If you suspect or can confirm that an animal is showing symptoms of one of the diseases listed as reportable, you must report it to:

- your local vet or
- Wildlife Health Australia State Coordinator, www.wildlifehealthaustralia.com.au/AboutUs/ContactDetails.aspx
- your state or territory's department of primary industries or agriculture by phoning the Emergency Animal Disease Watch Hotline on 1800 675 888.

3 Parasitic Disease

3.1 Ectoparasites

Zoonotic: may be vectors for other pathogens
Species affected: All
Similar presentation to: viruses (pox virus), fungal dermatopathies

Reptiles may be infested with a wide variety of ectoparasites, primarily mites and ticks. Little is known about the relationship between haematophagous arthropods and their hosts, despite Australia having more reptile-specific ticks than any other region (Natusch, et al., 2018). Ectoparasites are capable of transmitting viruses, bacteria, protozoa 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 eliminated through a combination of environmental decontamination, and treatment of the reptile with suitable parasiticides.

The snake mite, *Ophionyssus natricis*, is an introduced species recently reported in a variety of captive and wild snakes and lizards in south-eastern and southern Australia (Norval, et al., 2020). This mite is of significance to both animal and human health, with the mite reported to cause abnormal shedding, anaemia, and death in snakes and lizards, and dermatitis in people (Norval, et al., 2020). The mites may easily be transferred to new locations via movement of their hosts such as via translocation, rehabilitation and release, or by introduction of infested animals to a naïve or mixed species environment in a captive setting. Mites often go undetected as they burrow under the scales, and accidental introduction could happen quite easily, therefore reptiles should be kept separate during rehabilitation, good hygiene practices should be maintained, animals should always be released at point of capture when possible, and on occasions where this is not possible animals should be treated for mites prior to release.

Figure 1 a) central netted dragon cloacal mites (Image: P Thompson), b) orange mites clustered around toenails of a wild Asian house gecko, and c) orange mites under scales along the ventral tail of a wild Asian house gecko

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.

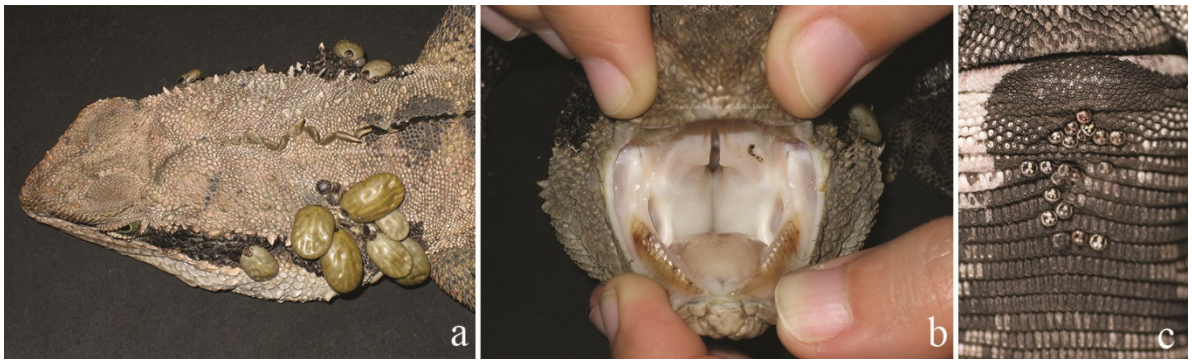

Figure 2 a) Engorged adults and nymphs, Amblyomma moreliae, causing b) anaemia in a wild eastern water dragon, and c) camouflaged ticks on the ventral surface of a lace monitor

Leeches may also be commonly found in the axilla and inguinal area of many turtles. The impact of these parasites and their capacity to act as a vector of disease are unknown, however, their presence is generally considered incidental with little impact on the host.

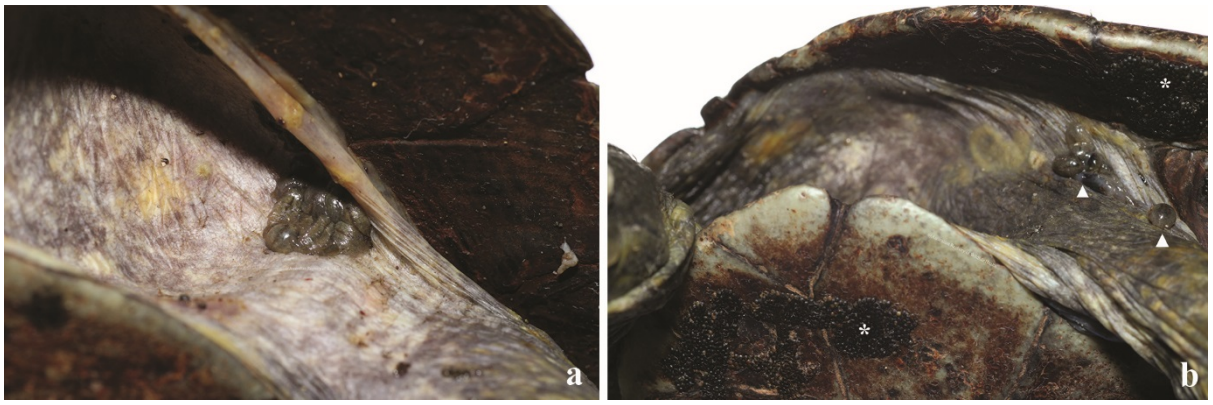

Figure 3 a, b) A ball of leeches in the axillary area of a Bellinger River Snapping Turtle (b, arrows), and b) invertebrate eggs (unknown species) glued over the surface of the plastron and underside of carapace (asterisk)

3.2 Endoparasites

3.2.1 Gastrointestinal parasites

Many endoparasites infect reptiles. The interpretation of faecal floatation's in reptiles must be undertaken with care, since parasite ova of prey are often found, including invertebrate parasites of insectivorous species.

In most cases, the presence of intestinal parasites is incidental and low levels of parasites are expected in all free-ranging wildlife. There are occasions however, where parasite burdens may become severe enough to cause clinical disease including anaemia, melena, thrombosis or blockage of the gastrointestinal tract. Treatments are best undertaken after parasite identification, and aimed at reducing rather than eliminating parasites. Retaining natural host-parasite relationships should be a consideration for animals in care and for health maintenance during translocation or other conservation action.

Strongylurus paronai is a common gastric roundworm of Eastern bluetongue skinks, Eastern water dragons and frilled lizards (Griffiths, et al., 1998). In dead animals, this parasite often crawls into the pharynx and oral cavity.

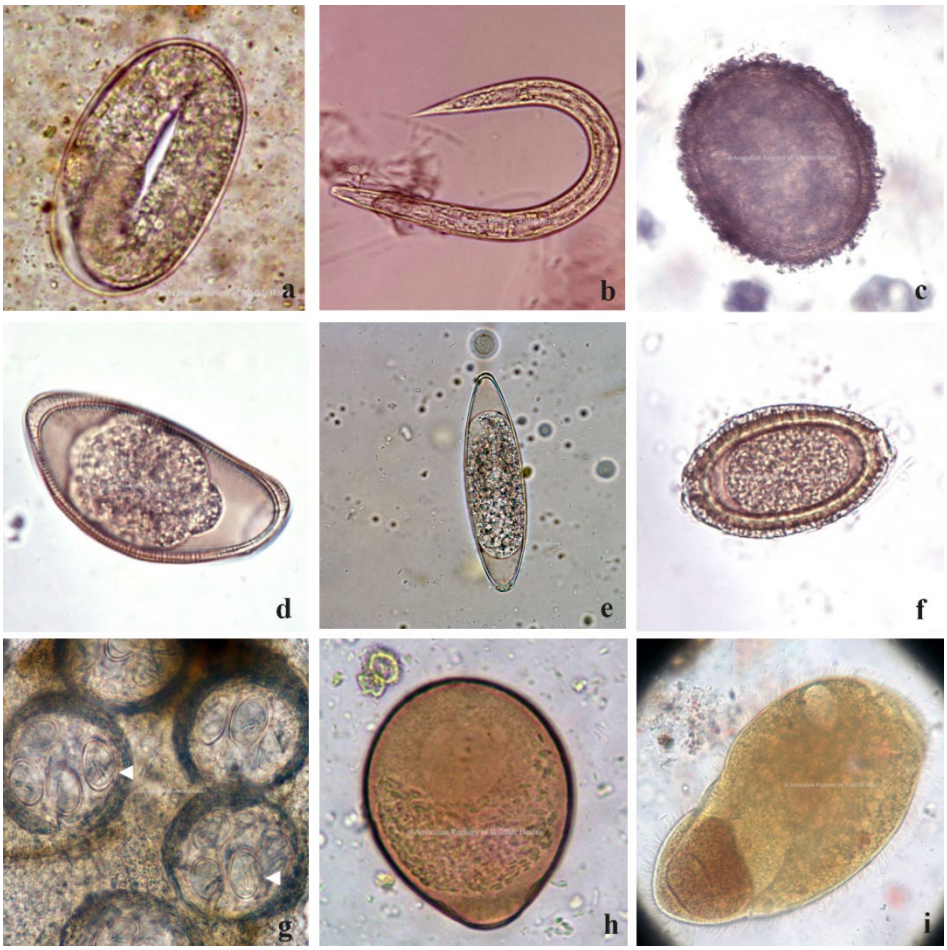

Figure 4 Various reptile faecal parasites a) strongyle-type ova, b) strongyle larvae, c) ascarid ovum, d) oxyurid ovum, e) oxyurid ovum, f) capillaria ovum, g) cestode ovum (note characteristic hooklets), h) encysted Nyctotherus sp. ciliate, i) Nyctotherus sp. ciliate. Images courtesy of P. Thompson.

Nematodes of the *Abbreviata* and *Spinicauda* genus' are common, species-specific parasites of the Eastern water dragon, and can be found within 100 km of the coast mainly, perhaps exclusively, in

NSW (Jones, 2007). These parasites are commonly found in tight coils in the stomach, but can migrate to the oesophagus and stomach after the host's death.

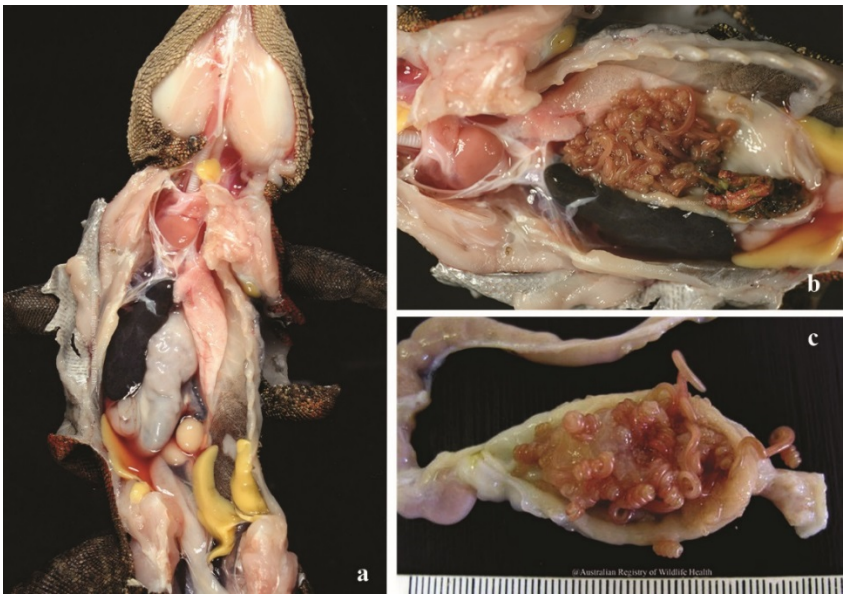

Figure 5 Eastern water dragon with a) profound anaemia caused by a severe co-infection of ticks and (b,c) Abbreviata physignathi nematodes in the stomach

3.2.1.1 Sparganosis

Reptiles are the secondary intermediate host for the plerocercoid cestode *Spirometra* sp. which needs a minimum of three intermediate hosts before infecting the definitive host; canids, felids and other mammals, including humans. Crustaceans and copepods are the primary intermediate host. In Australia, *Spirometra eranacei* is often reported from wild short-beaked echidnas which are, presumably, like humans, accidental hosts. While this parasite causes little disease for the intermediate host, people who are infected through ingestion of contaminated meat or water may experience prolonged inflammation and tissue damage as the parasite migrates through the body.

3.2.1.2 Spirorchid flukes (trematodes) in sea turtles

Zoonotic: No
Species affected: Loggerhead turtle, hawksbill turtle, green turtle, flatback turtle, leatherback turtle
Similar presentation to: bacterial infection, coccidiosis

Flukes of the *Spirorchiidae* family cause morbidity and mortality in marine turtles, especially green and loggerhead turtles (Marchiori, et al., 2017). In Australia, approximately 50% of green turtles are infected with spirorchid flukes (Gordon, et al., 1998). Flukes may be found in blood vessels, or within the alimentary tract of infected turtles depending on the species of fluke found. Small flukes are sometimes present in the lumen of the urinary bladder of marine turtles. Adult flukes may cause aortic blockages, aneurisms, ulceration of the intestinal mucosa or urinary bladder, while eggs may cause disseminated granulomas (inflammatory nodules) throughout various tissues, most notable along the serosal surface of the small intestine (Gordon, et al., 1998).

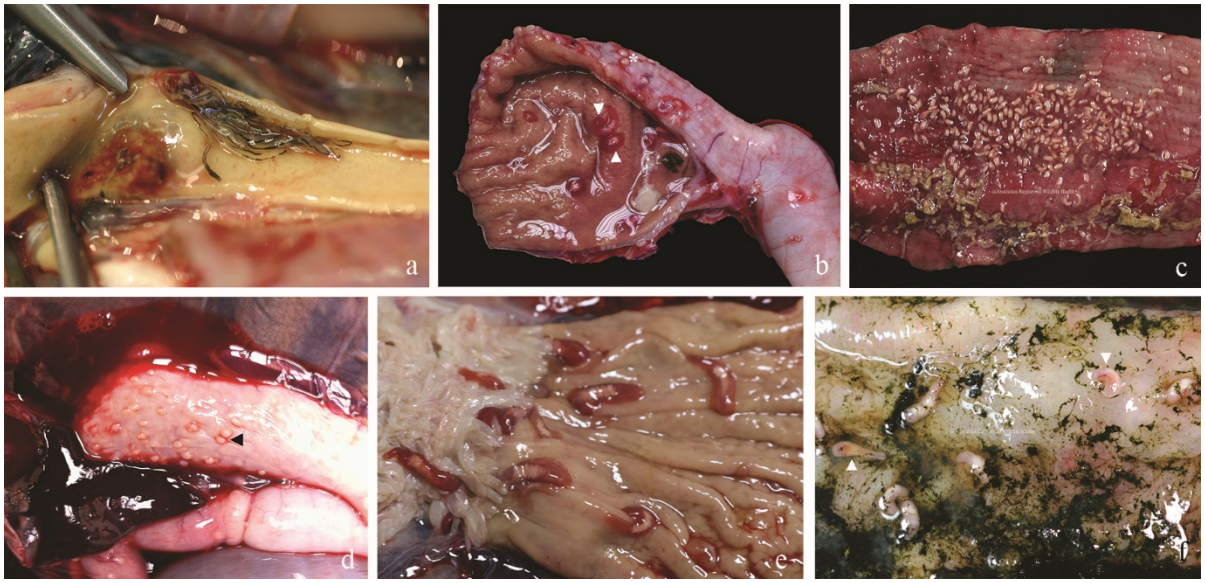

Figure 6 Marine turtles with a) aortic aggregate of adult trematodes, b) adult trematodes (arrow) and serosal granulomas (asterisk) in small intestine, c) adult trematodes along mucosal surface of small intestine, d) serosal granulomas, e) adult trematodes in stomach (black arrow), and f) adult trematodes in small intestine (arrows)

Gross and microscopic observation of adult trematodes or ova in blood vessels, tissue, and faeces are suitable diagnostic tools, however identification of species using morphological features is difficult and molecular techniques are more reliable and readily used (Marchiori, et al., 2017).

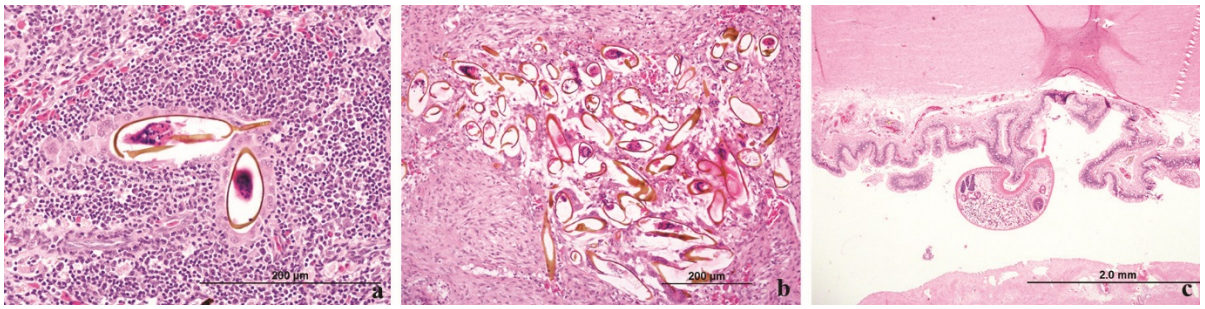

Figure 7 Marine turtle histopathology (H&E) a) trematode ova in spleen, b) trematode ova in aorta, and c) adult trematode in small intestinal lumen

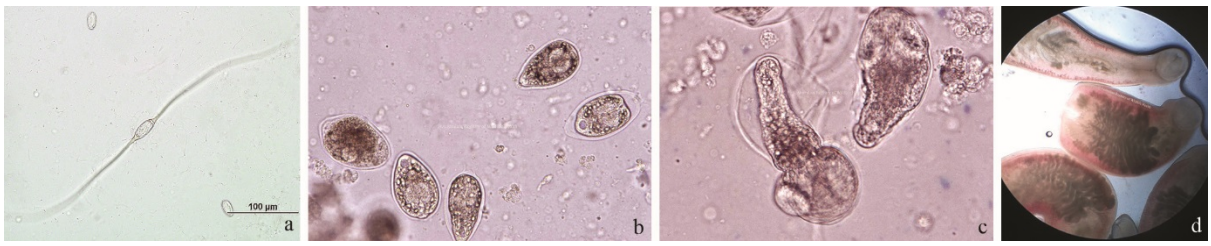

Figure 8 Trematodes seen a) in a faecal float, b) on a wet preparation of a serosal surface scrape of small intestine, c) excysting trematodes on serosal surface scrape of small intestine, and d) adult trematodes viewed under a dissecting microscope

3.2.2 Blood parasites

Haemoprotozoa and microfilaria are common incidental findings in reptiles. *Haemogregarina* species, *Trypanosoma* spp., *Haemoproteus* spp. and *Plasmodium* spp. are frequently found during haematological examinations.

Haemogregarine parasites have been identified within the pulmonary parenchyma and red blood cells of numerous snakes and monitors seized by Australian customs service officials upon illegal entry into Australia. Mosquitos 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.

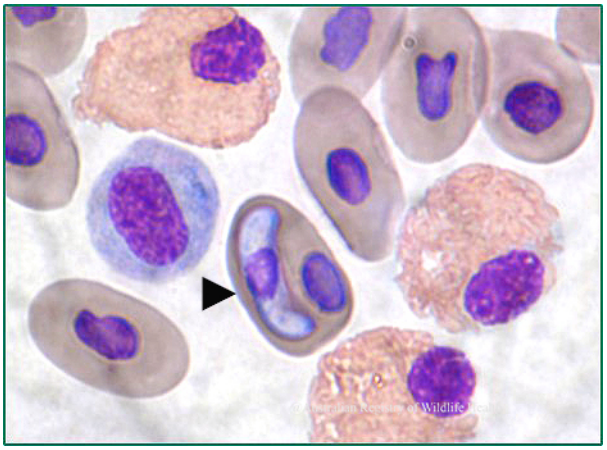

Figure 9 Haemogregarine parasite within a red cell identified on blood smear, Diamond Python. Image courtesy of P. Thompson.

3.2.3 Protozoa

Zoonotic: Yes
Species affected: yellow-bellied sea snake, desert death adder, eastern water dragon, central knob-tailed gecko, inland bearded dragon, red-bellied black snakes, Stimson's python, inland taipan, tiger snake, frilled lizard, freshwater crocodile
Similar presentation to: bacterial infection, other parasitic infections, viral infections

Microsporidia are protozoal parasites that have been detected within various reptiles and may or may not represent clinical pathology (Sokolova, et al., 2016). Infection has been associated with necrotising lesions of the musculature, bone pathology in sea snakes (Gillett, et al., 2016), granulomatous lesions in the ovary of an eastern water dragon and central knob-tailed gecko (Reece & Hartley, 1994), and granulomatous multi-organ inflammation in inland bearded dragon (Sokolova, et al., 2016). Overseas, the microsporidian *Encephalitozoon pogonae* has been reported to cause weight loss and death in numerous captive inland bearded dragons in Austria, the USA, and Japan (Shibasaki, et al., 2017). 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 mass/body that stains positively with periodic acid-Schiff (PAS) staining protocols. Traditionally, morphology has been used to identify microsporidia however this is challenging, and molecular techniques are required to definitively identify the parasite. Immunocompromised humans are also susceptible to infection, and microsporidia have been reported to infect the eyes of otherwise healthy people, therefore care must be taken when handling infected reptiles (Gillett, et al., 2016).

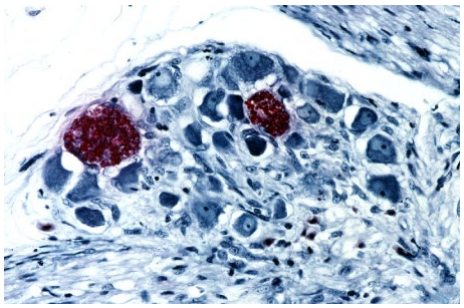

Figure 10 Microsporidia, central knob-tail gecko

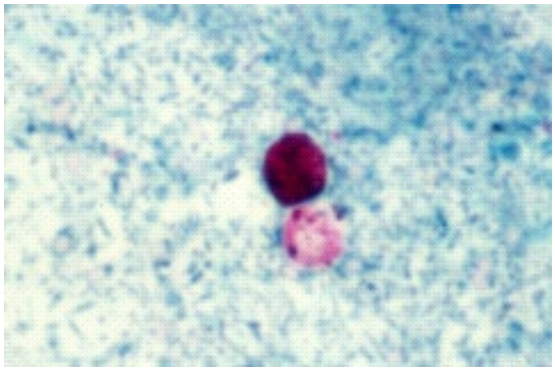

Figure 11 Cryptosporidia spp. Faecal sample, Inland Taipan. Modified Zeihl-Neelsen stain

Cryptosporidia spp. are coccidian parasites that can cause hypertrophic gastritis (inflamed stomach lining) in snakes and lizards. In Australia, cryptosporidiosis has been identified in captive red-bellied black snakes, Stimson's python, inland taipan, tiger snake, inland bearded dragon, and a wild frilled lizard (Oros, et al., 1998; Koehler, et al., 2020). Clinical signs associated with cryptosporidiosis include weight loss, regurgitation, diarrhoea and death. Infection occurs via the faecal-oral route and is more prominent in animals with concurrent disease (Wildlife Health Australia, 2018). 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, however oocysts are shed in faeces or regurgitate intermittently and repeat testing may be required to confirm infection (Wildlife Health Australia, 2018). 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. DNA sequencing, PCR, histopathology, and direct fluorescence antibody testing are also used in the laboratory setting. Molecular detection and identification are preferred. There is no known effective treatment for cryptosporidiosis.

There is a risk of zoonotic infection from reptiles that have consumed prey infected with non-reptilian *Cryptosporidia* sp. such as *C. parvum* and *C. muris* (Wildlife Health Australia, 2018).

3.2.3.1 Coccidiosis in green turtles

Zoonotic: No
Species affected: Green turtles
Similar presentation to: trauma, trematodiasis, septicaemia

While coccidia can potentially be found in all reptiles without causing clinical disease, there are instances where coccidia can cause morbidity and mortality. Systemic coccidiosis in green turtles is one such example.

Repeated epizootics of neurologic dysfunction and mortality in subadult and adult green turtles have been 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. Grossly visible inflammatory changes in the intestinal tract, kidney, thyroid gland, or brain may or may not be evident (Rose, et al., 2003).

Mortalities were thought to be caused by a coccidian parasite *Caryospora cheloniae*, however, Ban de Gouvea Pedroso, et al. (2020) used molecular characterisation to identify the presence of protozoa 98.8% similar to *Schellackia orientalis*. They found that mortality outbreaks were more likely to occur in larger animals (curved carapace length >68cm), in warmer months particularly October, during or one month prior to El Nino like events and that disease occurrence can be expected to occur anywhere between the southern end of Fraser Island, Qld to Sydney, NSW (Ban de Gouvea Pedroso, et al., 2020). Coccidiosis was not detected north of Fraser Island.

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

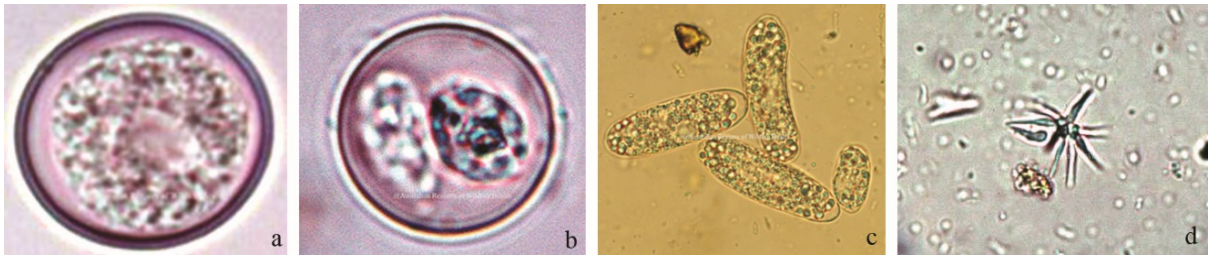

Figure 12 a) unsporulated and b) sporulated coccidia (Isospora) in faeces, and c) unsporulated Schellackia-like coccidian and d) sporulated Schelackia-like coccidian in green turtle faeces

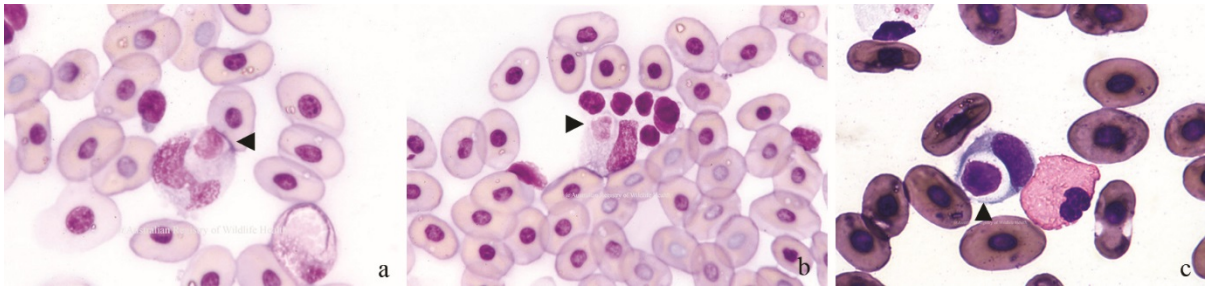

Figure 13 Monocytes containing Schellackia-like coccidian zoites (black arrows), blood film of a green turtle

4 Bacterial diseases

Ideally treatment of bacterial infection is based upon isolation of the organism within lesions, and antimicrobial sensitivity testing. Treatment without consultation and confirmation of the infectious agent may lead to ineffective treatment, and antimicrobial resistance. Some of these organisms are potentially zoonotic. Sound hygiene protocols for reptiles in rehabilitation will protect both reptiles and their rehabilitators.

4.1 Salmonellosis

Zoonotic: Yes
Species affected: All
Similar presentation to: may be no signs of disease, other bacterial infections, trauma, parasites

Salmonella spp. can be found readily in wild and captive reptiles, however, infection in reptiles is not often associated with clinical disease. *Salmonella* does have the potential to become a severe, possibly fatal, zoonotic pathogen, therefore good hygiene practices are recommended to anyone handling reptiles, especially young children and immunocompromised people. Various studies have found that snakes are more likely to carry *Salmonella* spp. than other reptiles, but lizards and turtles can also be carriers, and while captive reptiles are significantly more likely to carry *Salmonella*, those in the wild can also be a reservoir (Wildlife Health Australia, 2018; McWhorter, et al., 2021).

Clinical disease is often the result of an insult that disrupts the barrier between the gastrointestinal system and the body cavity such as trauma, parasites, or other disease, or from an increase in general stress such as handling, overcrowding, or suboptimal environmental conditions (Wildlife Health Australia, 2018). Salmonella may also be transmitted thorough ingestion of contaminated food such as feeder mice (in a captive setting), native rodents, and insects (McWhorter, et al., 2021).

While many animals may be asymptomatic, clinical disease may present as anorexia, regurgitation, depression, respiratory disease, dehydration, or diarrhoea, and in turtles lesions of the plastron or carapace (Wildlife Health Australia, 2018).

Microbiological culture or PCR are diagnostic however repeat testing may be required due to intermittent shedding in faeces. Cloacal swabs are preferable over faecal samples for testing. Treatment of asymptomatic reptiles that are otherwise healthy is discouraged, while supportive treatment with possible antibiotic treatment of sick reptiles is preferred. However, some isolates of *Salmonella* spp. are antibiotic resistant and treatment without susceptibility studies should be avoided (McWhorter, et al., 2021). Recovery from clinical infection may be slow. Good hygiene practices and minimising stress is the best method for limiting clinical disease, while good hygiene practices for any people handling either wild or captive reptiles are recommended.

4.2 Mycobacteriosis

Zoonotic: Yes
Species affected: All
Similar presentation to: other bacterial infections, fungal infections

Reptiles, like any animal, have the potential to suffer significant disease through infection with *Mycobacterium* spp. bacteria. A wide variety of *Mycobacterium* spp. have been isolated from all classes of reptiles, including painted dragons, knob-tailed geckos, carpet pythons (centralian and north-western), desert death adders, Arafura file snakes, freshwater crocodiles, Jardine River turtles, eastern long-necked turtles, hawksbill turtles, loggerhead turtles, flatback turtles and green turtles.

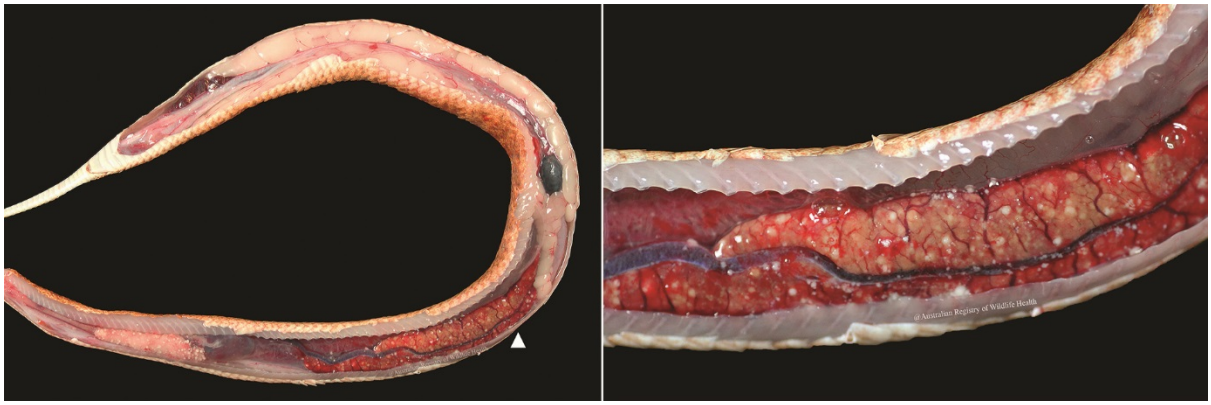

Figure 14 Desert death adder with lesions throughout the liver caused my Mycobacterial infection

Mycobacteria are able to survive in soil for many years, but appear to be most common in aquatic environments and are pathogens of concern in the rehabilitation setting. Clinical signs may vary and are often non-specific, dependant on the site of infection. On gross examination, infection is often associated with granulomatous lesions of liver, spleen, lungs (especially in marine turtles), gut, bone and skin. Granulomatous centres are often necrotic therefore sample collection at the margin of the lesion is recommended.

Diagnosis requires culture of the organism; however acid-fast bacteria can often be seen on Zeihl-Neelsen stain of impression smears of the lesion. Treatment is generally not recommended due to zoonotic potential and generally poor response to treatment. Mycobacteria also appear to have a high resistance to chemical disinfectants and UV (Wildlife Health Australia, 2013). For this reason, it is recommended that turtles undergoing rehabilitation should be kept in isolated/individual water bodies for the duration of their treatment. Water filtration systems should contain a sterilisation element to prevent mycobacteria from proliferating in water and in biological filtration systems.

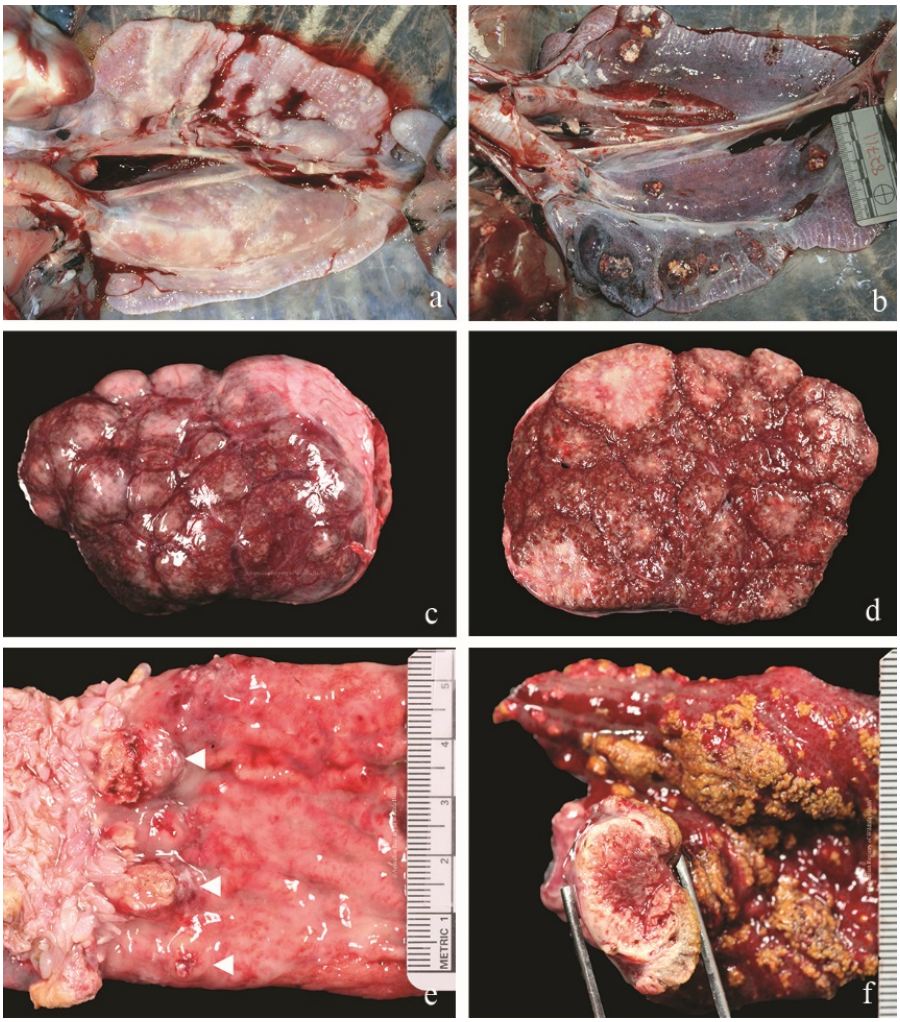

Figure 15 a,b) Mycobacterium chelonae in green turtle lungs, c,d) Mycobacterium marinum granulomas in enlarged spleen of a hawksbill turtle (whole, and cut surface), and d,e) gastrointestinal granulomas caused by Mycobacterium marinum in a hawksbill turtle

4.3 Enterococcus lacertideformus in skinks and geckos

Zoonotic: No
Reportable: please report to Australian Registry of Wildlife Health
Species affected: Christmas Island blue-tailed skink, Christmas Island Lister's gecko, Asian house gecko, common four-clawed gecko
Similar presentation to: neoplasia, trauma, sparganosis, other bacterial infections

In 2014 on the remote Australian Territory of Christmas Island some of the critically endangered, conservation dependent Lister's Geckos developed unusual facial swellings and those animals became emaciated and died. At the same time, similar lesions were noted in some of the feral geckos found on the island. These lesions were found to be caused by a novel bacterium, which has now been identified as *Enterococcus lacertideformus* (Agius, et al., 2021). *E. lacertideformus* has been identified to cause fatal infections in 3 species of gecko and one skink species on Christmas Island, with probable reports also recorded in reptiles in the USA, Poland and Malaysia. The biosecurity risk to Australian native fauna is significant, making examination of reptiles with ulcerative or proliferative lesions extremely important.

Infection is thought to be spread by biting or via the oral cavity and animals present most commonly with gingival swelling leading to gelatinous subcutaneous nodules of the head and face, and finally multisystemic expansile nodules throughout multiple tissues whereby this biofilm coated bacteria begin to replace bone and soft tissues (Rose, et al., 2017; Agius, et al., 2021). There is little inflammatory response from the host and animals go on to die, often from emaciation. Clinical presentation in geckos is much more dramatic as the soft tissues of the head and face are contorted with swelling and nodules, however in skinks presentation may be much more subtle, and ante-mortem signs may not be easily appreciated. In skinks, infection is often associated with open, non-healing wounds.

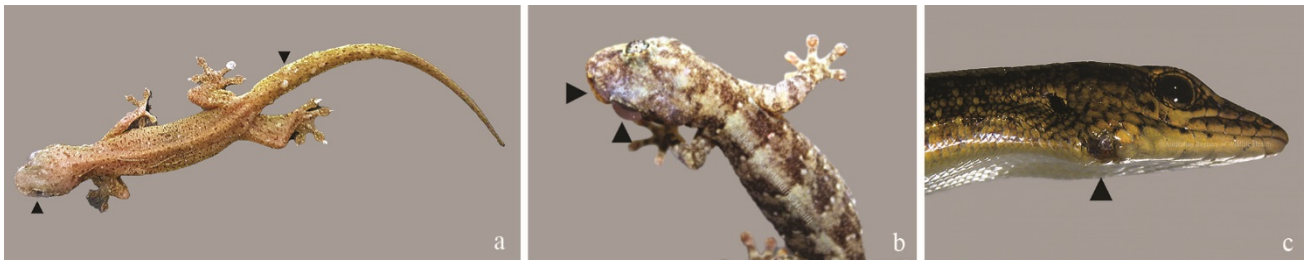

Figure 16 a) emaciated Asian house gecko with facial and tail lesions, Enterococcus lacertideformus, b) Lister's Gecko with expanded spectacle and facial lesions, E. lacertideformus, c) Blue-tailed skink with open wound, E. lacertideformus

4.4 Dermatophilosis

Dermatophilosis, caused by *Dermatophilus congolensis*, is a common invader of the skin of reptiles and an important zoonotic disease that can cause serious skin lesions in humans. *D. congolensis* has mistakenly been referred to as a fungal pathogen in the past, however it is a filamentous bacterium that causes crusty scabs in a variety of animals, and 'brown spot disease' in farmed crocodiles (Shilton, 2019). Proliferative crusty skin lesions may be superficial, but these sometimes obscure deeper abscesses. As with fungal infection, this disease is typically associated with animals kept in damp or humid conditions, or under cool conditions where there is insufficient access to heat sources.

Dermatophilosis can be identified within gram stained skin scrapings or biopsies. The organisms can appear as cocci or as beaded, branching double chains of cocci. *D. congolensis* requires 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 husbandry and reduction in environmental stressors.

5 Viral diseases

5.1 Nidoviruses

5.1.1 Bellinger River Virus

Zoonotic: No
Reportable: please report to Australian Registry of Wildlife Health
Species affected: Bellinger River snapping turtle
Similar presentation to: trauma, ranavirus

A large scale mortality event in 2015, which resulted in the loss of approximately 90% of the population of wild Bellinger River turtles, was likely caused by a novel nidovirus commonly called Bellinger River virus (Zhang, et al., 2018). The virus affected predominantly adult and subadult animals which either presented dead, or with bilateral swelling of the eyelids which became

ulcerated in severely affected animals, and in some cases tan coloured lesions on the skin of the ventral thighs.

Gross and microscopic examination showed necrosis of the kidneys, spleen and lacrimal glands of the eyes. Real-time PCR is available for molecular confirmation of infection with Bellinger River virus. Infection with Bellinger River Virus was uniformly fatal in animals found exhibiting lesions; however clinically health animals may return recurrent positive PCR ocular swabs.

Figure 17 Bellinger River snapping turtles with progressing eye lesions from a) unilateral mild (left eye) swelling, b) bilateral swelling and c) severe bilateral swelling and ulceration

Despite extensive work in the field it is not known where this virus originated or if it is present in other waterways, highlighting the need for vigilance when observing any freshwater turtles coming into, or being released from, rehabilitation care.

5.1.2 Bobtail flu (Shingleback nidovirus-1)

A syndrome commonly referred to as 'bobtail flu' has been identified in both wild and captive shingleback lizards from Western Australia. This syndrome is most likely due to infection of the upper respiratory tract with a novel nidovirus (Shingleback nidovirus-1), however a causal association between the syndrome and viral infection has not been proven (O'Dea, et al., 2016).

Clinically, shinglebacks may show a loss of condition, lethargy, depression, pale mucous membranes, increased mucous in the naso-oral cavities that may bubble from the eyes or nostrils, sneezing, or watery or swollen eyes (O'Dea, et al., 2016). Diagnosis is based on PCR from oral/tracheal and/or eye swabs and infection appears to be highly contagious highlighting the need for good hygiene during rehabilitation of injured or ill shinglebacks. Early reports suggest that the same virus may be present in centralian, eastern and northern blue-tongued skinks across multiple states (Shilton, et al., 2019). This highlights the importance of biosecurity control around the movement of reptiles for conservation or for the global pet trade.

5.1.3 Snake nidovirus

In 2017 an experimental infection trial confirmed Nidoviruses were responsible for a disease seen previously in boids and pythons in Australia, USA and Europe (Shilton, et al., 2019). In these species, clinical symptoms were primarily respiratory and included anorexia, reddened mucosa in the mouth, oral ulceration, excessive mucous in the mouth and/or lungs, or sudden death (Shilton, et al., 2019). Immunohistochemistry and in situ hybridisation testing of lung, intestine, oral swabs, and faeces accurately confirm nidovirus infection. The mode of transmission for this virus is unknown.

As this is an emerging disease, little is known about infection in Australian snakes, however initial work has detected nidovirus in a number of species across most states (Shilton, et al., 2019) and as such, increased awareness about the symptoms of this disease is necessary for early detection and control especially where animals may be kept in rehabilitation without strict quarantine and either mixed with other species or released into the wild.

5.2 Adenoviruses

Adenoviruses have been identified in numerous species of lizards, snakes, chelonians and crocodiles globally including bearded dragons, central netted dragon, and eastern blue-tongue skinks (Hyndman & Shilton, 2011; Hyndman, et al., 2019). Adenoviruses are generally species specific. Infection can result in anorexia and weight loss, diarrhoea, weakness, neurological signs, or sudden death, especially in young animals, however infection in adult animals may be sub-clinical and these individuals may become carriers of the virus. Concurrent parasitic infections are reported (Hyndman & Shilton, 2011; Hyndman, et al., 2019). Transmission is believed to be via the faecal-oral route, and there may be vertical transmission from mother to offspring however this is unconfirmed. Microscopic examination of liver in affected animals may reveal hepatocellular degeneration to necrosis, and large eosinophilic to basophilic intranuclear inclusion bodies. Diagnosis can be confirmed by PCR testing of oral and cloacal swabs.

5.3 Paramyxovirus

5.3.1 Sunshine virus

Sunshine virus is an emerging disease of captive Australian pythons associated with outbreaks of neurological and respiratory disease (Shilton, et al., 2019). The effect of this virus on wild pythons is unknown. Clinical signs may include lethargy, anorexia, abnormal skin sloughing, and various neurological signs, however in red-tailed boas infected with sunshine virus in Thailand, neurological signs have not been reported (Shilton, et al., 2019).

Transmission is via direct contact, but may also be vertical from infected parent to egg/offspring. PCR of oral/cloacal swabs is diagnostic in live pythons however animals may be asymptomatic for several years before exhibiting clinical signs, therefore serial sampling is recommended (Shilton, et al., 2019). In deceased pythons, brain is preferred for PCR however lung, kidney and liver may also be tested.

6 Fungal diseases

6.1 Mycotic and other dermatoses

Zoonotic: Yes
Species affected: All
Similar presentation to: bacterial infection (particularly *Dermatophilus* spp.), viral infection, trauma, nutritional deficiency (vitamin A, Zinc)

Mycotic infection of the skin is common in reptiles, especially lizards. Organisms are considered opportunistic as they are often present in soil or substrate and come into contact with damaged skin or immunocompromised animals. Infections may be more common in animals from habitats or enclosures that are damp and have limited sunlight. Typical infections include *Basidiobolus* spp. *Geotrichium* spp., *Paecilomyces* spp., *Trichophyton* spp., *Aspergillus* spp., *Fusarium* spp., *Acremonium* spp., *Chrysosporium* spp., and *Nannizziopsis* spp. (McCracken, 1994; Shilton, 2019; Peterson, et al., 2020). Although most common in captive reptiles, free-ranging animals in unsuitable habitats or exposed to concurrent disease or other stressors may suffer infection with opportunistic fungi. *Nannizziopsis* and similar fungal genera are considered primary fungal pathogens that are capable of infecting otherwise healthy reptiles.

Fungal infections may be more common in animals with dysecdysis, or abnormal skin shedding. This can be easily demonstrated in eastern blue-tongue skinks where dysecdysis can lead to strangulation and sloughing of the digits or feet with secondary infection by the fungus *Trichophyton terrestre*.

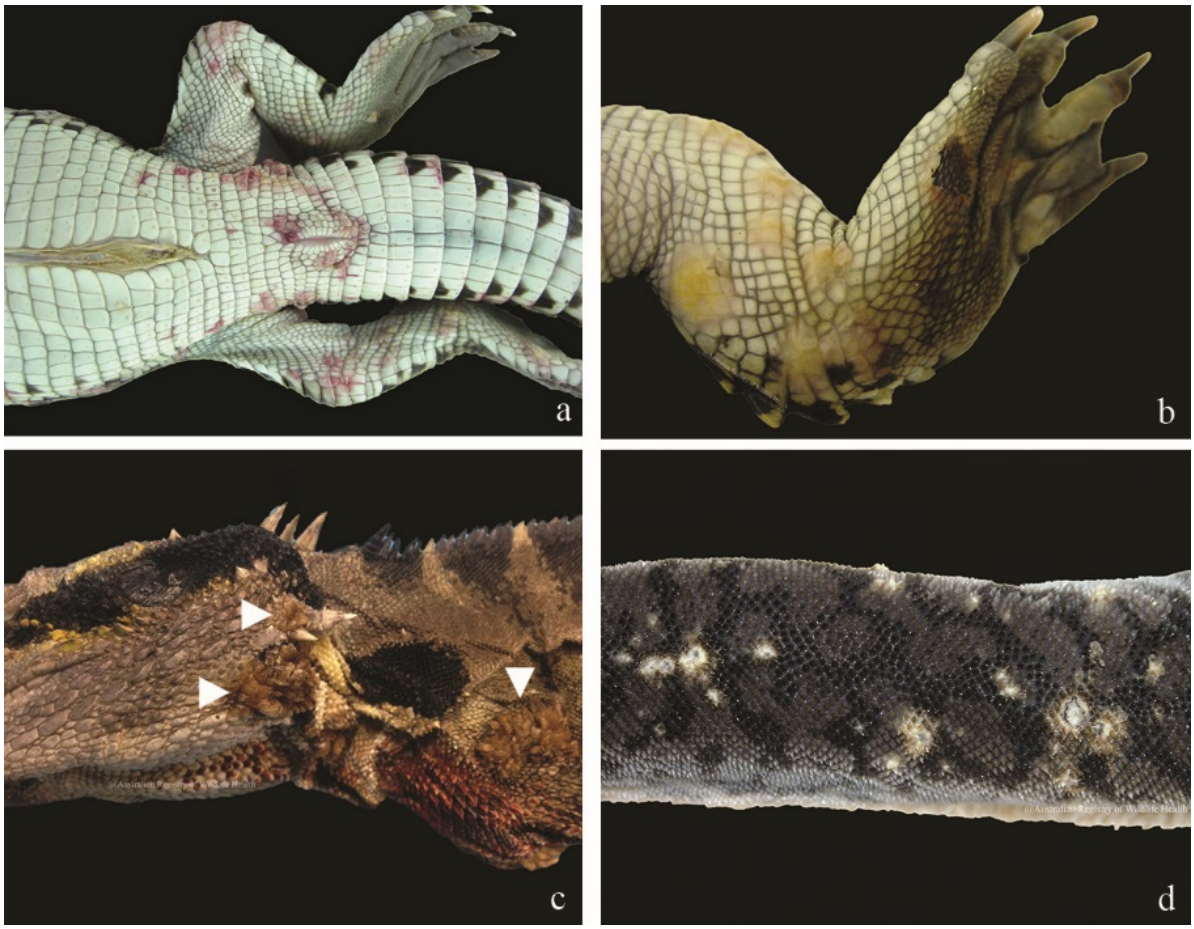

Figure 18 Mycotic dermatosis a) juvenile saltwater crocodile, Rhizomucor variabilis, b) juvenile saltwater crocodile, Fusarium solani, c) eastern water dragon, Paecilomyces lilacinus, d) Arafura file snake, Trichophyton verrucosum

6.1.1 Nannizziopsis

Zoonotic: No
Reportable: please report to Australian Registry of Wildlife Health
Species affected: crocodilians, eastern water dragon, eastern blue-tongue skink, centralian blue-tongue skink, tommy roundhead dragon, shingleback lizard
Similar presentation to: other fungal infections, pox virus, trauma, Dermatophilus spp. Infection, nutritional deficiency (vitamin A, Zinc)

Skin or systemic infection with the fungus *Nannizziopsis spp.*, is an emerging disease which has been described as causing fatal disease in a number of both captive and wild reptile species. Previous reports of *Chrysosporium* spp. in farmed crocodilians may actually be attributed to *Nannizziopsis crocodili*; however this is unconfirmed (Shilton, 2019).

Nannizziopsis barbatae has been ascribed as the cause of a significant event in wild eastern water dragons in Qld and has also been recorded in wild eastern bluetongue skink, centralian blue-tongue skink, tommy roundhead dragon, and shingleback lizard (Peterson, et al., 2020).

Skin lesions can differ depending on species but generally, infection is characterised by thickening of the skin, proliferative yellow/tan crusting lesions, inflammation, necrosis, and ulceration of skin, and

severe emaciation (Peterson, et al., 2020). Infection is contagious and capable of causing significant disease which inevitably leads to death. There is no known effective treatment. Infection is confirmed using histopathology, culture, PCR and sequence analysis.

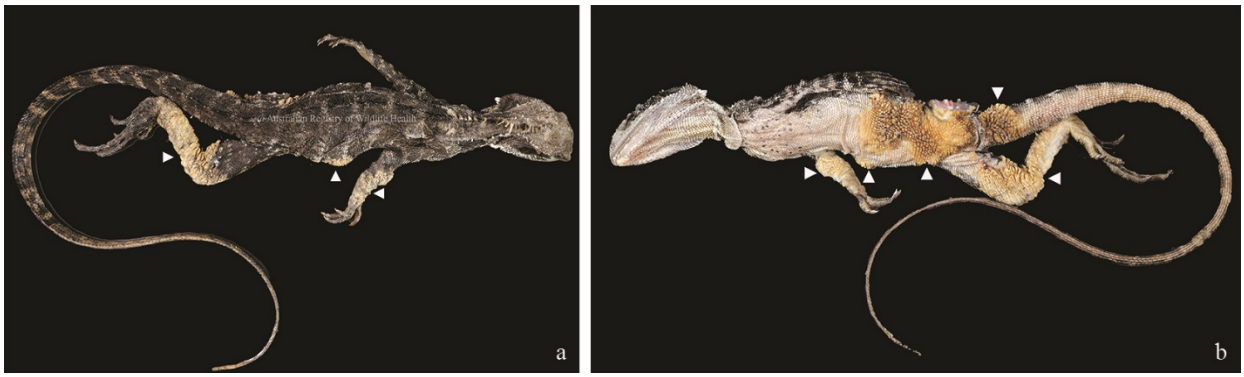

Figure 19 A severely emaciated eastern water dragon with proliferative yellow skin lesions over the legs, ventral abdomen and tail caused by Nannizziopsis barbatae

7 Nutritional Disease

Malnutrition, other than emaciation, is rare in free ranging wildlife. When malnutrition does occur in free ranging wildlife, it is primarily the result of inappropriate supplemental feeding by humans. Intestinal parasites, other infections, and inappropriate housing of captive reptiles can result in altered nutrient absorption.

7.1 Nutritional Osteodystrophy (metabolic bone disease)

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 or in prolonged rehabilitation. The disease is rarely seen in free-ranging reptiles, other than those that are suspected to be released 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 D_3 and no exposure to the ultraviolet rays required to produce metabolically active vitamin D.

Plant derived vitamin D_2 (ergocalciferol) is not considered to be metabolically active in reptiles. The active form of vitamin D in reptiles is vitamin D_3 (cholecalciferol). Vitamin D_3 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. The inappropriate nature of some common reptile dietary items are illustrated by lean beef meat that contains a ratio of approximately 1:16 Ca:P, and beef heart that contains approximately 1:38 Ca:P. Feeding 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.

Reptiles with nutritional osteodystrophy have soft, misshapen bones and shells. The lower jaw may be shortened when muscle traction draws in the soft bones. Lizards with osteodystrophy have an abnormal posture, since they are unable to support their body effectively. The animal may have a hunched spine, a swayed back, or a sideways curve of the spine, or vertebral compression fractures. If spinal cord injury accompanies vertebral fracture, the reptile will have rear limb paresis or paralysis.

The long bones, particularly the femurs, are often very swollen due to periosteal thickening around a thin, weakened cortex. 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.

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.

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 D_3. 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. Treatment of severely affected reptiles should be undertaken with care as the prognosis for return to function may be guarded and the advanced condition is extremely painful.

7.1.1 Severe emaciation in chelonians

In chelonians, severe nutritional imbalance and emaciation can lead to complete breakdown of the connective tissues of the carapace and plastron causing bone to collapse. This syndrome has been recorded in both wild marine and freshwater turtles. The cause of initial debility is generally due to a chronic disease process leading to profound emaciation and nutritional loss. In many cases both disease and shell instability are too severe to respond to treatment. In freshwater turtles, this may occur when the animal is already significantly debilitated upon entry into brumation and is accelerated once activity is resumed.

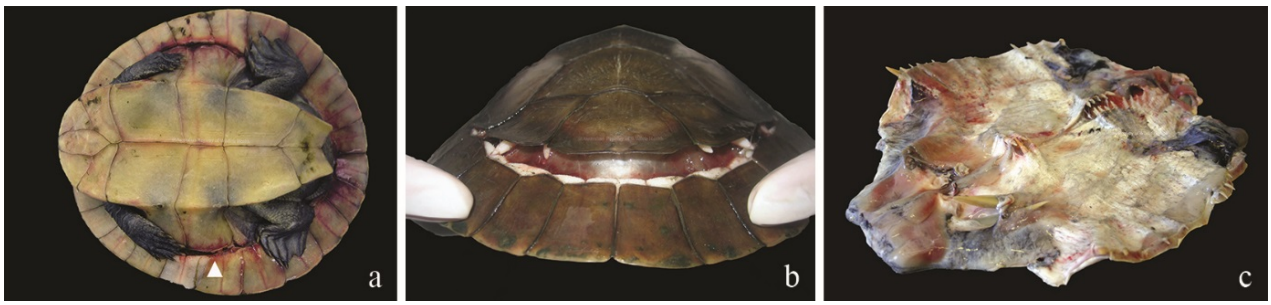

Figure 20 Emydura spp. kept in unfavourable housing for unknown period with a) plastron dismemberment from carapace and b) costal bone separation from marginal scutes and bones of the carapace, c) severely emaciated wild green turtle with dissolution of connective tissues of plastron resulting in bone instability and exposure

8 Traumatic Injury

Lizards, snakes and turtles are commonly admitted to urban wildlife care centres. These reptiles are almost uniformly admitted due to traumatic injury. Underlying infectious disease and clinically apparent parasitic diseases in urban reptiles presented with trauma are likely to be under-reported.

8.1 Shock

Many animals that have suffered a serious injury or are debilitated by disease are found in a state of shock. Shock is defined as acute circulatory failure that results in multisystemic decrease in blood flow and therefore low oxygenation of tissues. Clinical signs of shock are often related to low blood pressure. The mucous membranes of an animal in shock may be pale or muddy and the peripheral blood vessels are collapsed or provide a weak pulse. The heart rate may be weak and rapid. Animals in a state of shock are often weak, depressed, have rapid breathing and reduced urate output. Animals suffering from endotoxic shock, may have bright red mucosa.

Dehydration often contributes to the lack of peripheral perfusion and oxygenation. An animal is severely dehydrated when the eyes are sunken, the capillary refill time is very slow, the mucous membranes are dry and tacky, and the skin has lost its elasticity.

The neuroendocrine cascade that is initiated during shock is initially protective, but over time energy reserves are depleted and peripheral vasoconstriction contributes to hypoperfusion of tissues. The heart, lungs, liver, gastrointestinal tract, pancreas, and central nervous system are most susceptible to damage induced by hypoxia.

Pulmonary effects of shock can include consolidation of tissue, and increased risk of bacterial infection. The effects of shock on the lung can be highly species specific. Some species experience “Acute Respiratory Distress Syndrome”, also known as shock lung, which is manifested as pulmonary oedema and decreased activity of alveolar macrophages.

Cells exposed to hypoxia initially undergo degenerative change, but once cell death has taken place, the changes induced may be irreversible. Acute necrosis of the proximal renal tubules and periacinar (centrolobular) regions of the liver occur under conditions of low oxygenation. Mucosal ulceration and decreased mobility occur when the gastrointestinal tract is deprived of oxygen. These gastrointestinal lesions can allow bacteria or bacterial toxins to enter the blood stream. Animals that are treated in the early phase of shock may respond to initial fluid therapy, but succumb to acute renal tubular necrosis (urate nephrosis and visceral gout), gastrointestinal ulceration or sepsis three to five days later. If reduced blood flow continues, pancreatic damage can result in the release of vasoactive substances and myocardial depressant factor. Ultimately, reduced blood and oxygen flow to the brain causes nerve cell death.

8.2 Soft Tissue Injury including bite wounds

Soft tissue injury in reptiles is often inflicted by predators, lawnmowers, or vehicles. Careful examination is required to assess the degree of damage sustained and identify secondary infection of wounds. Bite wounds are heavily contaminated with bacteria and many victims will quickly become septicaemic. Healed wounds should be monitored to ensure that the scar tissue does not impede normal skin shedding.

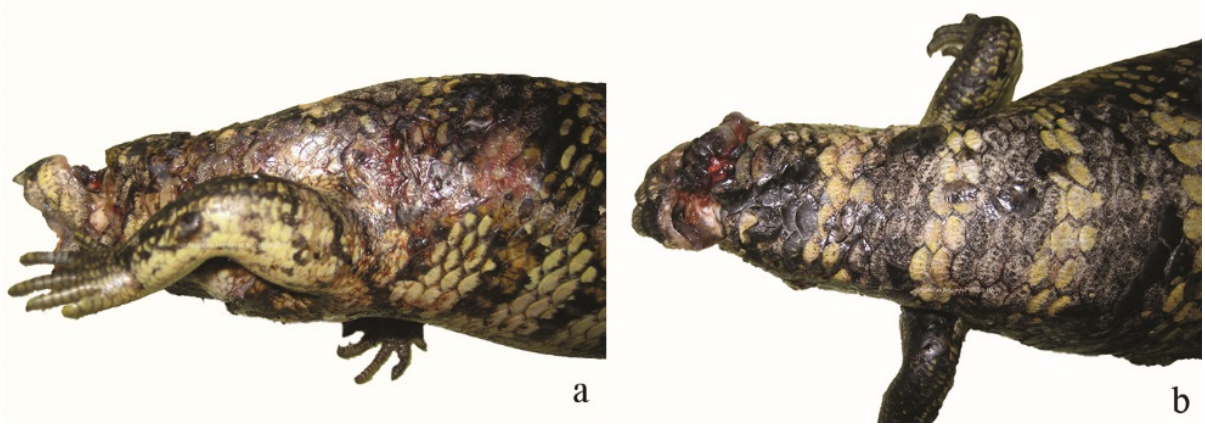

Figure 21 Eastern blue-tongue skink with traumatic bite wounds to lower lateral abdomen (a) and tail (b) leading to sepsis.

Predation is an everyday occurrence in wildlife. Bite wounds inflicted by feral or domestic pets account for a large proportion of the animals admitted to wildlife care centres. Bite wounds caused by canine and feline predators are most often centred over the neck, shoulders, and dorsal thoracic region. Puncture wounds caused by feline predators are often very fine. These wounds can be difficult to see, particularly in reptiles, where the skin is tight and often multi-coloured. Canid-inflicted bite wounds do not necessarily break the skin. The mild outward appearance of predator-induced lesions often masks very serious internal injuries. Feline bite wounds can puncture deep into the tissues, and felids have the potential to break bones or reduce the underlying muscle to pulp. Canine bite wounds are most often associated with circular subcutaneous contusions and crushing injury to the chest. Canine bite wounds often cause extensive pulmonary contusion and fractured ribs. Measuring the distance between paired puncture wounds can be used to estimate the inter-canine tooth distance, which can help to differentiate wounds inflicted by cats or foxes (18-22 mm inter-canine distance) from those inflicted by large dogs (>25 mm inter-canine distance).

Feline bite wounds are often heavily contaminated with *Pasteurella multocida*, and other bacteria, and sepsis is a very common sequela. Canine bite wounds may be contaminated with a wide variety of gram negative and anaerobic bacteria. The prognosis for any animal receiving predator bite wounds, however, is most often guarded to poor.

8.3 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.

8.4 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 appear normal, the deeper tissues lining the necrotic bone are often infected.

Shell fractures in chelonians are most often inflicted by vehicles, lawn mowers and canids. In marine turtles, boat strike and propeller injuries can cause significant damage. 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. Shell fractures may result in tears that enter the lungs of coelomic cavity, providing an opportunity for infection. Radiographic examination of the reptile should be undertaken to evaluate the full extent of the injuries. Anterio-posterior, lateral and dorso-ventral views will provide more detailed information regarding the location and extent of injuries.

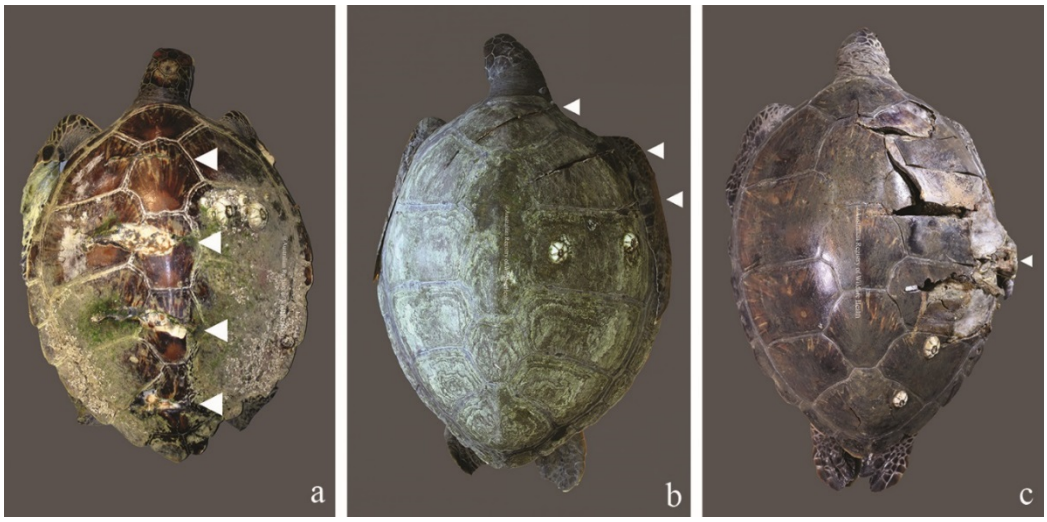

Figure 22 Propeller injuries of green turtles a) four centrally aligned linear shell fractures, b) three linear shell fractures to the upper right quadrant, c) multiple right side shell fractures and evisceration of internal organs (arrow)

8.5 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. If the wound progresses, surgical amputation may be warranted. The tail will usually regrow, but not necessarily to the original length or shape.

9 Diseases of Unknown Aetiology

9.1 Neoplasia

A wide variety of neoplastic processes have been described in reptiles however they are of low incidence, occur sporadically, and most often reported in captive animals. Neoplasms can involve one or multiple tissues, and may be found incidentally or causing clinical symptoms or sudden death. Clinical symptoms can vary widely from lethargy, anorexia, abnormal swelling, neurological signs, chronic regurgitation, or sudden death depending on the site and type of neoplasm. Treatment for neoplasia in reptiles is limited, however surgical removal may improve outcomes in some cases. Cases where renal tumours have been naturally excised and excreted by snakes have been recorded.

A recent publication outlined plastron associated squamous cell carcinoma in wild Bell's turtles (Hall, et al., 2020). Field reports suggest that more individuals within this population have been affected, however further research is needed to determine if this is a neoplasia of significance for this population.

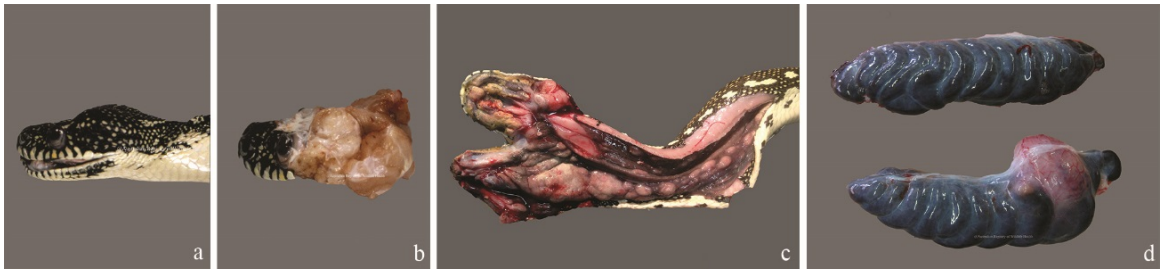

Figure 23 a, b) diamond python, retrolobular carcinoma, c) diamond python, granulocytic round cell tumour, d) north-western carpet python, kidney sarcoma

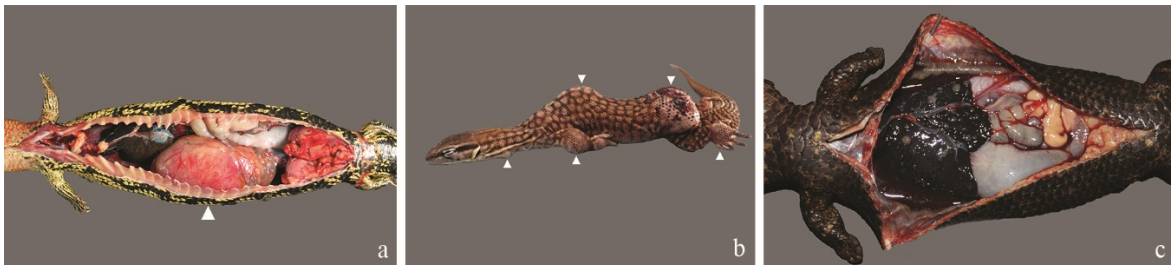

Figure 24 a) blotched blue-tongue lizard, oviductal sarcoma, b) pygmy mulga monitor, metastatic chondroblastic osteosarcoma, c) shingleback, multisystemic lymphosarcoma with multifocal liver lesions

10 Species mentioned in text

Arafura filesnake (*Acrochordus arafurae*)

Asian house gecko (*Hemidactylus frenatus*)

Bellinger River snapping turtle (*Wollumbinia georgesi*)

Blotched blue-tongue lizard (*Tiliqua nigrolutea*)

Centralian blue-tongue skink (*Tiliqua multifasciata*)

Centralian carpet python (*Morelia bredli*)

Central knob-tailed gecko (*Nephrurus levis*)

Central netted dragon (*Ctenophorus nuchalis*)

Christmas Island blue-tailed skink (*Cryptoblepharus egeriae*)

Christmas Island Lister's geckos (*Lepidodactylus listeri*)

Desert death adder (*Acanthophis pyrrhus*)

Diamond python (*Morelia spilota spilota*)

Eastern bluetongue skink (*Tiliqua scincoides*)

Eastern long-necked turtle (*Chelodina longicollis*)

Eastern water dragon (*Intellagama lesueurii*)

Flatback turtle (*Natator depresses*)

Freshwater crocodile (*Crocodylus johnstoni*)

Frilled lizard (*Chlamydosaurus kingii*)

Green turtles (*Cheloniae mydas*)

Hawksbill Turtle (*Eretmochelys imbricata*)

Hosmer's skink (*Egernia hosmeri*)

Inland bearded dragon (*Pogona vitticeps*)

Inland taipan (*Oxyuranus microlepidota*)

Jardine River Turtle (*Emydura subglobosa*)

Knob-tailed gecko (*Nephrurus amyae*)

Lace monitor (*Varanus varius*)

Loggerhead turtle (*Caretta caretta*)

Mitchell's water monitor (*Varanus mitchelli*)

Northern blue-tongued skink (*Tiliqua scincoides intermedia*)

Northwestern carpet python (*Morelia spilota variegata*)

Painted dragon (Ctenophorus pictus)

Pygmy mulga monitor (*Varanus gilleni*)

Rankin's dragon (*Pogona henrylawsoni*)

Red-bellied black snake (*Pseudechis porphyriacus*)

Saltwater crocodile (*Crocodylus porosus*)

Shingleback lizard, aka Bobtail lizard (*Tiliqua rugosa*)

Stimson's python (*Liasis stimsoni*)

Tiger snake (*Notechis scutatus*)

Western bearded dragon (*Pogona minor minor*)

Yellow-bellied sea-snake (*Pelamis platurus*)

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