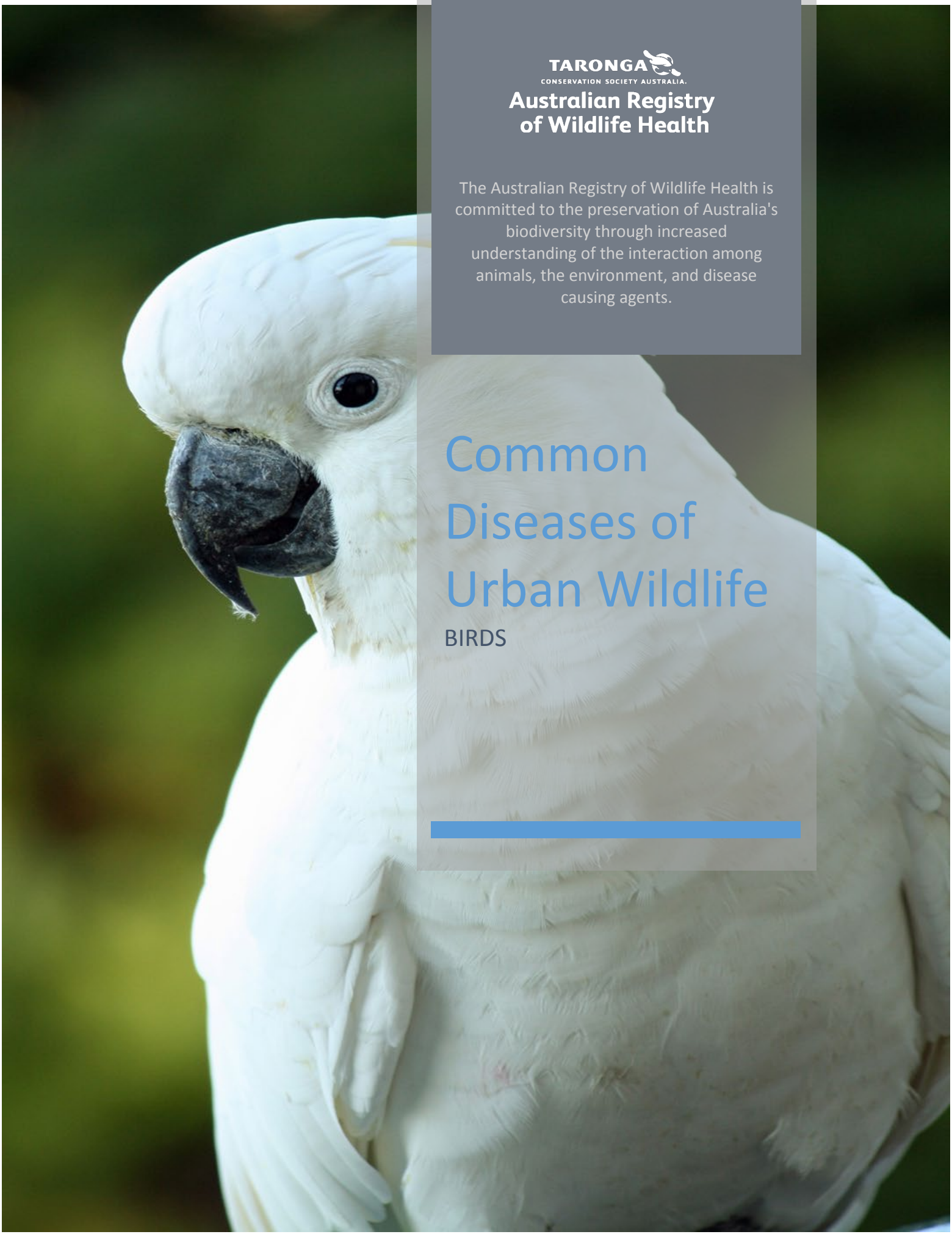

TARONGA
CONSERVATION SOCIETY AUSTRALIA.

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

BIRDS

Australian Government

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Contents

1 List of images

2 Introduction

A variety of diseases have been recognised within free ranging Australian birds. The purpose of this document is to review the diseases that occur often within particular species or taxonomic groups of birds. 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 birds 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's state coordinator, 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

For additional information see also: Atkinson C.T., Thomas N.J., Hunter D.B. (2009) Parasitic Diseases of Wild Birds. Wiley-Blackwell, Ames, Iowa.

3.1 Ectoparasites

Zoonotic: May be vectors for zoonotic pathogens
Species records: All
Similar presentation to: viruses (e.g. pox virus), fungal dermatopathies

Birds can be parasitised by ticks, lice, mites, fleas and hippoboscid flies. Heavy burdens of ectoparasites are often a reflection of a debilitated bird that is insufficiently grooming. Biting ectoparasites can transmit blood parasites, and can contribute to anaemia when present in large numbers.

Ixodes holocyclus, the paralysis tick, is found occasionally on birds. Anecdotal reports of tick paralysis in birds have been documented. Three species of the genus *Ornithodorus* are known in Australia, *O. macmillani* on wild birds, *O. capensis* on sea birds and *O. gurneyi* primarily on kangaroos (Barker & Walker, 2014).

Lice commonly infest wild birds, but rarely cause disease, such as anaemia. Heavy infestations may be treated with topical antiparasitic powders and by regularly changing substrate or enclosure materials such as towels for birds undergoing rehabilitation.

Knemidocoptes intermedius infestations are recorded as causing severe debility in forest ravens, pied currawongs, and superb lyrebirds (Holz, et al., 2005). Infestations are associated with significant epidermal proliferation, primarily involving the skin of the lower portions of the legs. Mites can be demonstrated by microscopic examination of scrapings of the thickened skin. Oral or subcutaneous administration of Ivermectin-like drugs will control *Knemidocoptes* sp. infestation, but more severe

infestations are often associated with foot deformation and general debility, which may not be amenable to treatment.

Figure 1 Progressively severe proliferative lower limb lesions from Knemidocoptes sp., pied currawong (a-c). Knemidocoptes sp. mites from a skin scrape stained with methylene blue (d).

Figure 2 Progressively severe proliferative lower limb lesions from Knemidocoptes sp., satin bowerbird. (Images courtesy of South Penrith Veterinary Clinic and Carol Probets).

A similar mite, presumed to be from the *Harpyrhynchus* genus has been described in Rainbow lorikeets, scaly-breasted lorikeets, red-collared lorikeets, musk lorikeets, and some finches. The presentation of this mite is very different from those above and appear as firm, raised, yellow subcutaneous skin nodules or cysts on the head, wings, and body. These lesions contain mites which are visible microscopically if material from a cyst is squashed between two glass slides, or in a formalin fixed biopsy processed routinely for histology. If you see affected birds, consider collecting a sample into ethanol to enable more precise identification of these parasites.

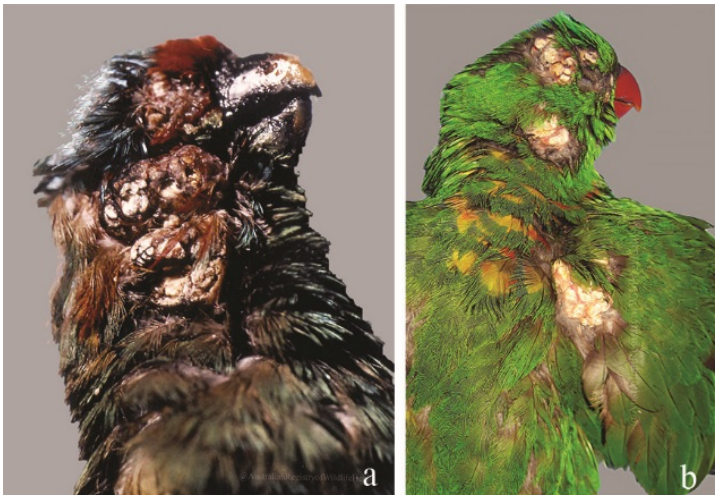

Figure 3 Harpyrhynchus rosellasinus cysts over the face, head and neck of a musk lorikeet (a), and similar lesions over the face, neck and wings of a scaly-breasted lorikeet (b), also presumably caused by Harpyrhynchus sp. mites.

We have a lot to learn about the identity and ecology of these organisms.

Hippobosca are a genus of fly that occurs commonly within the plumage of pigeons and birds of prey, especially tawny frogmouths, and owls. Hippoboscid flies can bite and are capable of acting as a vector in the transmission of disease. These flies will infest and bite humans, but do not seem to remain on human hosts for longer than 12-24 hours.

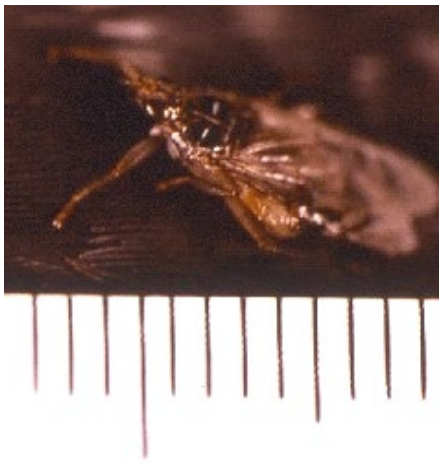

Figure 4 Hippoboscid fly

Sternostoma tracheacolum is the tracheal and air sac mite of Gouldian finches. Heavy burdens of these mites are capable of causing coughing, sneezing, and open mouth breathing. Disease associated with this parasite is most common in captive finches. A diagnosis can often be achieved by reflecting the neck feathers and shining a bright light across the trachea, or via microscopic examination of respiratory discharges. Ivermectin-like drugs can be used to treat affected birds.

3.2 Endoparasites

3.2.1 Throat worm: Cheilospirura (Xenocordon) gymnorhinis

Zoonotic: No
Species records: Australian Magpie
Similar presentation to: gapeworm, trichomoniasis, capillariasis

Cheilospirura (*Xenocordon*) *gymnorhinis*, also referred to as throat worm, has been described in juvenile magpies (De Chaneet & Robertson, 1983). The same, or similar, parasite also occurs in the oral cavity and pharynx of pied currawongs, pied butcherbirds, magpie larks, black-faced cuckoo shrikes, painted firetail finches, and rufous whistlers.

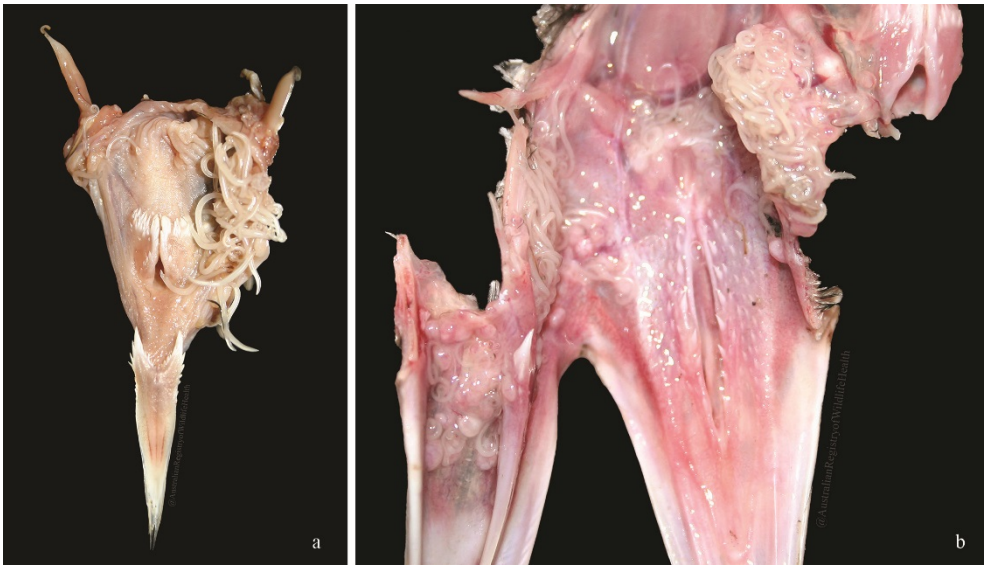

Figure 5 Cheilospirura (Xenocordon) gymnorhinis, oropharynx, a) Pied Butcherbird, b) Australian Magpie

C. gymnorhinis burrows its head into the mucosa of the oral cavity and pharynx. The host then responds by creating a fibrous nodule around the parasite. Although small numbers of parasites result

in self-limiting infections, large numbers can impair prehension of food, or partially obstruct the glottis causing malnutrition or respiratory symptoms respectively. Repeated manual removal of the parasites is recommended and this is assisted by application of moxidectin directly to the nematodes. Euthanasia may be a humane option for severely debilitated young birds with heavy burdens of *C. gymnorhinis*.

3.2.2 Gapeworm: Syngamus trachea

Zoonotic: No
Species records: domestic poultry, Australian magpie, metallic starling, Australian raven, pied currawong, variegated fairy-wren
Similar presentation to: throat worm, oral capillariasis, trichomoniasis

Syngamus trachea (gapeworm), should not be confused with *C. gymnorhinis* (throat worm; see above), although similar in presentation, the symptoms and treatment are different. *S. trachea* are small, red, worms that typically infect domestic poultry. *S. trachea* has been found in the trachea of a variety of native birds and occasionally magpies that die suddenly are found to have complete tracheal obstruction with masses of these parasites. The male parasites are very small, and they stay in copulation with the much larger females, creating a forked appearance.

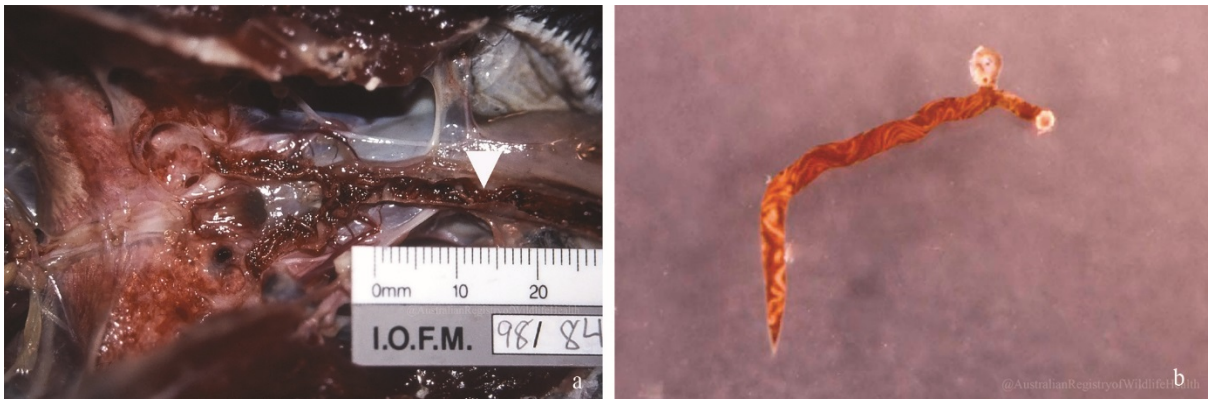

Figure 6 Gapeworm (Syngamus trachea) in the trachea of an Australian Magpie. These worms (arrow) have formed a thick raft along the entire length of the trachea in this bird. When removed, the worms are red and have the typical forked appearance (b).

3.2.3 Trematodes (flatworms)

Austrobilharzia spp. are schistosome parasites that have been reported from the nasal cavity of various duck species (Blair & Ottesen, 1979), and from blood vessels of silver gulls and various other waterbirds (Appleton, 1983). Most commonly reported in silver gulls, *A. terrigalensis* uses the whelk *Velacumantus australis* as its intermediate host, and can cause contact dermatitis or "swimmers itch" in people (Appleton, 1983). *Austrobilharzia* sp. infection is most often an incidental finding in birds.

Mawsonotrema eudyptulae and *Renicola* sp. are trematodes of little penguins that can be found in the liver and bile ducts (Obendorf & McColl, 1980; Harrigan, 1991). *M. eudyptulae* were found to be highly pathogenic and contributed to little penguin deaths in Victoria (Harrigan, 1991).

3.2.4 Nematodes (roundworms)

Gastrointestinal nematodiasis is usually an incidental finding in wild birds and parasite burdens are generally mild. Captive birds, however, may experience excessive parasite burdens that can contribute to debility.

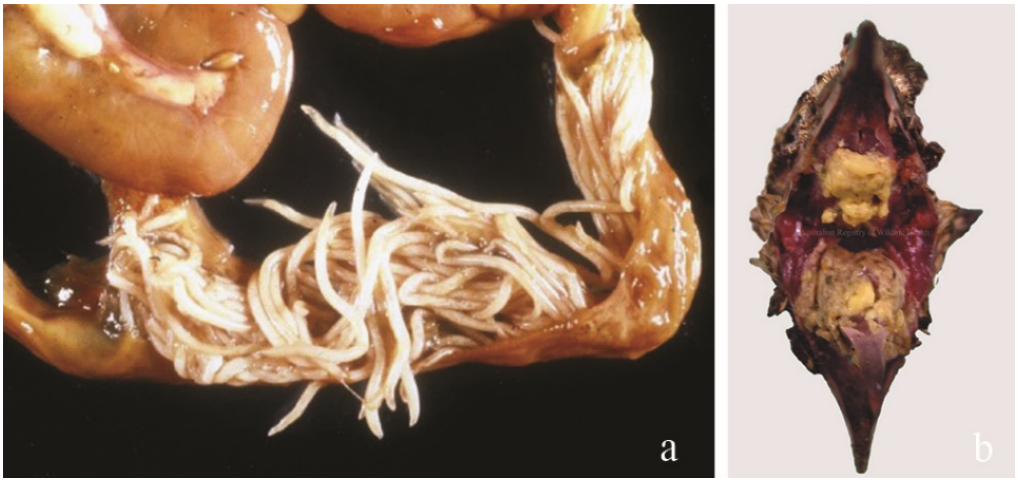

Figure 7 a) captive long-billed corella with a heavy burden of intestinal ascarids, and b) painted button quail with severe oral capillariasis presented as thick yellow oral plaques occluding the palate and throat.

Capillaria spp. are often evident within serpiginous tracts created as the nematode burrows through the mucosa and lamina propria of the oesophagus, proventriculus and liver. Eggs may also be present in faeces and appear as football shaped organisms with a thick shell and distinct bipolar plugs. Capillariasis occurs primarily in captive birds. Infected birds may suffer from extensive hyperplasia of the oesophageal mucosa and marked inflammation surrounding the parasites in the mucosa and lamina propria. Emaciation, dehydration, weight loss, diarrhoea, regurgitation, anaemia and oral necrotic plaques (Figure 9) can result from these infections. *Capillaria* sp. infections in wild birds are primarily an incidental finding during post mortem examination.

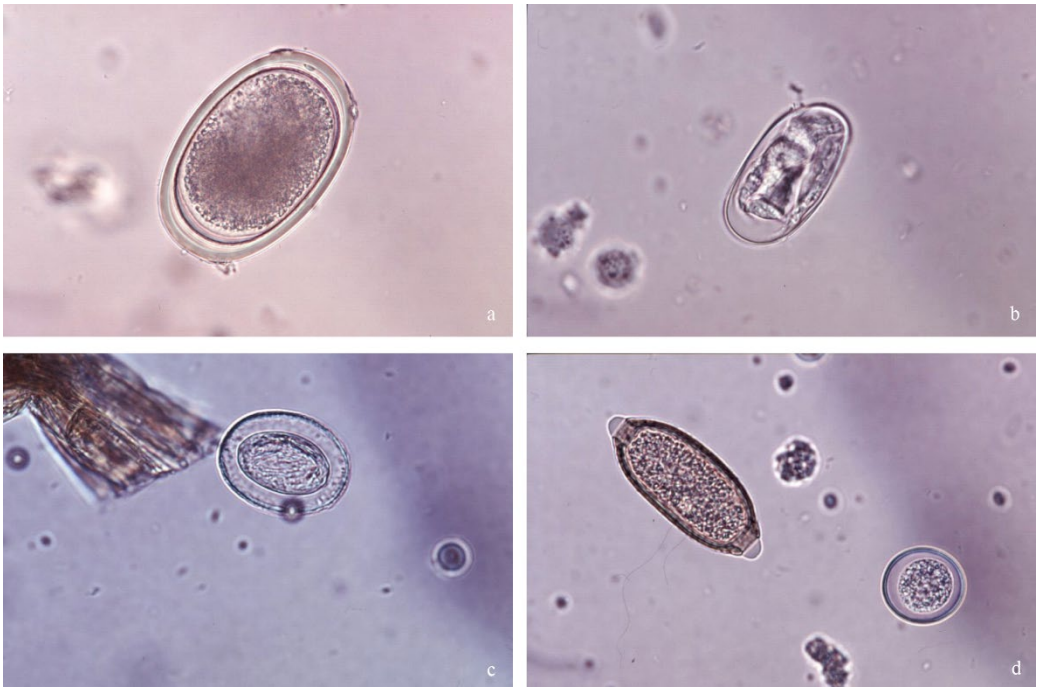

Figure 8 a) Ascarid ovum, b) strongyle-type ovum, c) cestode ovum, and d) capillaria ovum with coccidial oocyst in bird faeces

Contracaecum spp. are nematodes that parasitise the oesophagus and proventriculus of piscivorous birds. Small numbers of parasites pose no threat to the host. There are reports of large parasite burdens of *C. spiculigerum* being associated with proventricular ulceration, haemorrhage, emaciation and death in little penguins in Victoria (Harrigan, 1991).

Dispharynx species are nematode parasites that burrows into the mucosa of the crop, oesophagus or proventriculus in water birds, finches and birds of prey. Heavy infections can be associated with poor condition, diarrhoea, undigested seed in the faeces or death. Small numbers of organisms are easily tolerated, whereas larger numbers of organisms are often accompanied by inflammation, mucosal thickening and secondary infections.

Serratospiculum sp. reside within the airsacs of birds of prey, and infection is most common in falcons and Australian Hobbies. Square-tailed kites have also been identified as possible hosts. Two species are known in Australia: *S. guttatum* from *Falco longipennis* and *F. peregrinus* and *S. tendo* from *F. peregrinus.* Clinical respiratory disease has been described in Australian birds infected with *Serratospiculum* sp. that are subject to stress or concurrent disease. Diagnosis requires endoscopic examination of the air sacs. Ivermectin-like drugs can be used to treat affected birds.

Oxysprirura spp. are nematode parasites that can be found within the conjunctiva and nictitating membrane of a number of species. Infection with this parasite is usually asymptomatic, but may be associated with conjunctivitis in a small proportion of infected birds (Pass, 1993).

Angiostrongylus cantonensis, the rat lungworm, has been found to cause neurological dysfunction associated with eosinophilic or non-suppurative encephalomyelitis in yellow-tailed black cockatoos, and more commonly in tawny frogmouths (Montali, et al., 2004; Monks, et al., 2005; Ma, et al., 2013). Infection in tawny frogmouths is now a common occurrence and seems to have a seasonal prevalence. Birds become infected with the parasite by eating infected snails and slugs, the intermediate hosts. The parasites migrate through the intestinal wall and find their way to the spinal cord, where they ascend to the brain. Affected tawny frogmouths present unable to fly, with various levels of weakness/paresis and although they appear alert, are unable to right themselves if placed on their back. Spinal damage progresses to central nervous signs. Diagnosis of the infection can be very difficult, since birds do not usually develop eosinophilia in blood. Cerebrospinal fluid taps collected from infected animals are also often non-suppurative rather than eosinophilic, making it difficult to differentiate angiostrongylosis from viral or protozoal infection. NSW Department of Primary Industries now offer PCR to aid diagnosis via CSF ante mortem or central nervous tissue post mortem. Treatment of the infection in birds is also difficult. The parasite's cuticle retains many antigens and killing the worms can result in release of antigens with subsequent severe host immune response. Concurrent antiparasitic and anti-inflammatory treatment may halt parasite progression, but damage to the host's central nervous system is unlikely to regress.

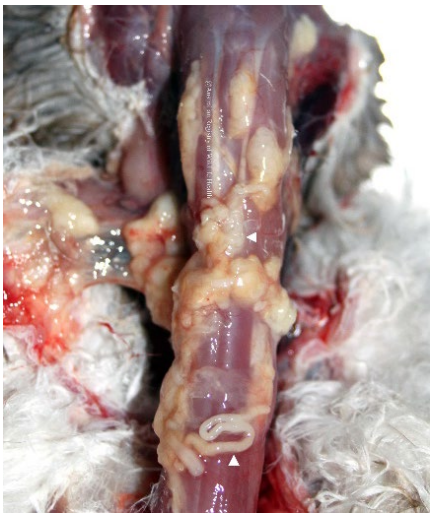

Figure 9 Cestode parasites, dorsal neck, spotted harrier

3.2.5 Cestodes (tapeworms)

Birds are parasitised by many species of cestode. None of these is considered to be highly pathogenic in free-living birds. It is possible, however, that large burdens of cestodes will add to the debility of a captive or compromised bird. If necessary, cestodiasis is treated with praziquantel. The treatment is usually repeated 10 days after the first dosage.

3.2.6 Protozoa (single-celled parasites)

A wide variety of protozoa has been reported within the gastrointestinal tract, cardiovascular system, musculature and renal tissues of free-flying birds. The following discussions regarding protozoa are limited to those protozoal infections known to be clinically significant.

Spironucleus-like (formerly Hexamita) organisms have been associated with numerous outbreaks and individual cases of emaciation, diarrhoea and fatal enteritis in Australian king parrots (Philbey, et al., 2002). Similar parasites have been identified in emaciated, wild sulphur crested cockatoos from western NSW (Registry unpublished). These birds become emaciated and have very thin walled intestinal tracts, often filled with fetid brown fluid. The intestinal tissues of affected birds seem to decompose very rapidly making it difficult to identify organisms on histologic examination. Microscopic examination of saline wet-mount preparations of intestinal scrapings can be used to demonstrate the organism during gross post mortem examination.

Gastrointestinal *Giardia* spp. infections have been documented in a variety of wild and aviary birds in Australia. *Giardia* spp. have been recovered from the intestinal lumen of straw-necked ibis in Western Australia, and a sulphur-crested cockatoo in Victoria (Forshaw, et al., 1992; Gallagher, et al., 1995). Giardiasis in captive young budgerigars can result in decreased growth rates, dehydration, and diarrhoea (Filippich, et al., 1998). Diagnosis of giardiasis is based upon direct microscopic examination of faeces or intestinal content. *Giardia* sp. trophozoites are pear shaped, binucleate, and have eight flagella. A cyst form, with four nuclei is occasionally shed in the faeces. Infection can be diagnosed with PCR, Wrights or modified acid-fast stained faecal samples or intestinal scrapings. Treatment of budgerigars with metronidazole decreased shedding of these protozoa in the faeces (Filippich, et al., 1998). Treatment for giardiasis is the same as for trichomoniasis. Careful attention to hygiene will prevent clinical infection in most captive birds.

Cryptosporidia spp. have been observed within the intestinal and proventricular brush border of wild Pacific black ducks, red-tailed black cockatoo, various captive finches and mannikins, Australian magpie, rock parrot, scaly-breasted lorikeet, southern boobook, and regent honeyeater (Registry). The significance of this parasite as an avian pathogen is poorly understood.

3.2.6.1 Toxoplasma gondii

Zoonotic: Yes
Species records: pied currawong, tawny frogmouth, satin bowerbird, little penguin, regent bowerbird, red-whiskered bulbul, crimson rosella
Similar presentation to: septicaemia, *Angiostrongylus cantonensis* infection

Toxoplasmosis is a potentially fatal disease in native and aviary birds, caused by the single celled parasite *Toxoplasma gondii* (Wendte, et al., 2011; Dubey, 2002). Cats are the definitive hots of this parasite and excrete oocytes in their faeces. Birds become infected when they ingest these oocytes, or when they ingest cysts in the tissues of small birds or mammals. Birds with toxoplasmosis are depressed, fluffed, debilitated or are found dead. Gross post mortem findings consist of pulmonary oedema, pulmonary congestion, and pale foci within the liver, spleen and intestinal mucosa. Histologic examination of fixed tissues reveals pulmonary oedema and congestion, fibrin within the distal airways, and non-suppurative inflammation or necrosis within the liver, spleen, brain, skeletal muscle, ventriculus, adrenal gland, or intestine. Numerous protozoa, morphologically consistent with *T. gondii* may be observed in avian tissues. Definitive diagnosis of *T. gondii* infection can be established in native birds using PCR or immunohistochemistry (Hartley & Dubey, 1990; Dubey, 2002).

Protozoal cysts resembling those of *T. gondii* are observed in the absence of inflammation during routine histologic examination of nervous tissue of a variety of native birds, especially the tawny frogmouth. These cysts are consistent morphologically with *T. gondii* and they appear to be a common incidental finding. Clinical disease occurs when these cyst walls break down and liberate the internal zoites, which incite the host's immune response causing tissue damage.

3.2.6.2 Trichomoniasis

Zoonotic: No
Species records: Peaceful dove, little penguin, rock (feral) pigeon, southern boobook, painted button-quail, brown goshawk, budgerigar, peregrine falcon, bar-shouldered dove, powerful owl, Australian bustard, barking owl, superb lyrebird, rainbow lorikeet, crested pigeons, spotted dove, Australian hobby falcon, black-shouldered kite, little eagle, pied currawong, Australian raven, Australian magpie, channel-billed cuckoo, tawny frogmouth (Park, 2011).
Similar presentation to: oral *Capillaria* species, throat worm

Oral trichomoniasis has been observed in debilitated free ranging birds, but is most common in captive wildlife that are undergoing treatment for various injuries. Trichomonads are common commensal agents within the avian alimentary tract and can be present in faeces, oral mucous or crop secretions. Trichomonads are ovoid single celled parasites (protozoa) that have four anterior flagella and an undulating membrane. These organisms are spread through either direct or indirect contact, including; contaminated food or water sources, consumption of infected prey, or during feeding of young. The factors that predispose a bird to develop trichomoniasis are unknown.

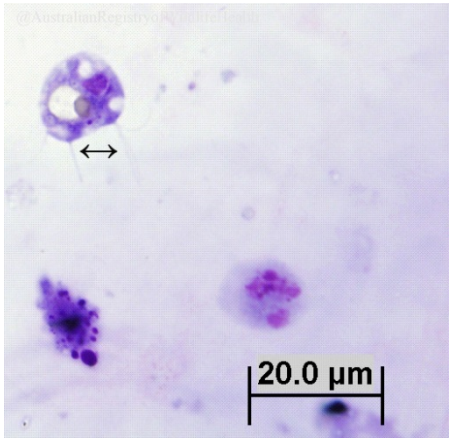

Figure 10 Trichomonads in an oral smear and stained with Diff Quick. You can see the anterior flagella (arrow)

Caseous oral plaques are created when the organisms cause tissue necrosis. Lesions are often subject to secondary bacterial infection. Weight loss, depression, anorexia, vomiting and difficulty swallowing are common clinical signs, and difficulty breathing and asphyxiation may occur in severe cases where plaques block the airway. A diagnosis of trichomoniasis is best made by examining a wet mount preparation of the caseous debris (Figure 3). Flagellates can be seen moving within the wet preparations under light microscopy. The organisms are much more difficult (often impossible) to see within cytologic and histologic preparations of affected tissues.

Treatment of trichomoniasis includes debridement of the caseous plaques, supportive care and administration of antiprotozoal agents.

3.2.6.3 Coccidia

Eimerian and isosporan coccidial oocysts are commonly identified within the faeces of healthy captive and free-flying birds. Disease associated with coccidial infection in free ranging birds is rare. Coccidia may cause necrotising enteritis in young captive birds of a variety of species. Microscopic examination of faecal wet preparations and flotations is recommended to monitor coccidia burdens in birds maintained in aviaries. When large numbers of faecal oocysts are detected or if oocysts accompany diarrhoea, treatment is advisable.

Renal coccidiosis is a common incidental finding within little penguins, Australasian gannets, and short-tailed shearwater (mutton bird). Limey disease is the term used to describe clinically apparent renal coccidiosis in nestling short-tailed shearwater. Chicks with limey disease are thin and have urate and faecal soiling of the pericloacal feathers (Munday, et al., 1971). Renal enlargement and multiple pale foci throughout the kidney are evident on gross post mortem examination. The ureter and cloaca may also be distended with urates. Microscopic examination of the affected renal tissue reveals inflammation within the interstitium surrounding large collecting ducts. Coccidial oocysts are often

evident within multiloculated granulomas within the collecting duct mucosa and in the surrounding interstitium.

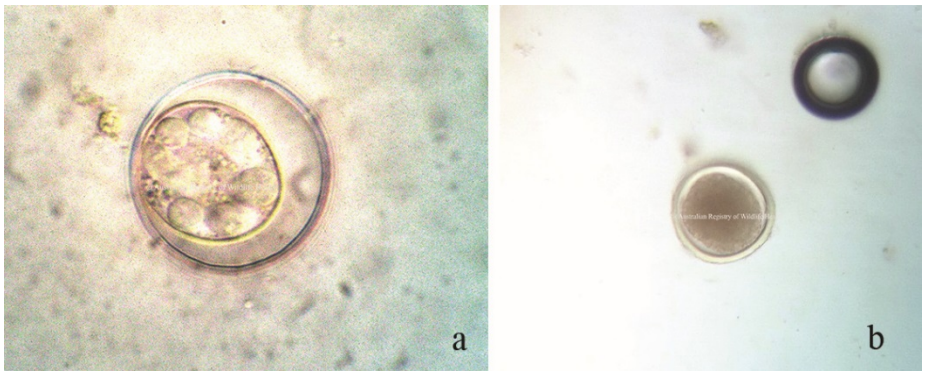

Figure 11 Coccidia oocysts in faeces from a) tawny frogmouth, and b) little penguin.

Caryospora spp. are coccidian parasites that can be found within the intestinal lamina propria and mucosa of carnivorous birds, 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.

Systemic coccidiosis associated with *Atoxoplasma* (formerly *Lankestrella*) spp. and *Isospora* spp. have been identified within circulating monocytes of various birds within Australia. Some of these include the regent honeyeater, red wattlebird, brown treecreeper, house sparrow, silvereye, and various aviary birds (Mackerras & Mackerras, 1960; Yang, et al., 2015). These systemic coccidian parasites undergo sexual reproduction (gametogony) within the mucosa of the gastrointestinal tract, and asexual reproduction (schizogony) within extra-intestinal tissues such as the liver and spleen. Sporozoites of this organism are transported among these sites within mononuclear cells. *S*porozoites are round basophilic organisms that have a small basophilic nucleus. These sporozoites are visible, usually as individual organisms, within the cytoplasm of mononuclear cells, which have an indented nucleus.

3.2.7 Protozoan blood parasites

Haemoproteus, *Leucocytozoon*, *Plasmodium*, *Atoxoplasma*, and a *Babesia*-like organism are genera of the family *Plasmodiidae* that are commonly found within the peripheral blood of wild Australian birds. Each of these organisms is arthropod borne. A bird may be infected with two or more of these organisms concurrently without any clinical signs. Young or debilitated birds may develop anaemia, anorexia, and depression as a result of large parasite burdens.

Although most often a common, incidental infection, on occasion, Leucocytozoon megaloschizonts. can cause clinically significant myopathy in pied currawongs and Australian magpies within the Sydney region. Infection occurs in juvenile, sub-adult and adult birds of both sexes, at any time of year. Heavy parasite burdens result in lethargy, weakness and debility. If the breast feathers are parted, pale oval foci may be evident throughout the pectoral musculature of affected birds. There is no known treatment for this protozoal infection and birds often die shortly after initial examination. The lifecycle of this protozoal agent is unknown.

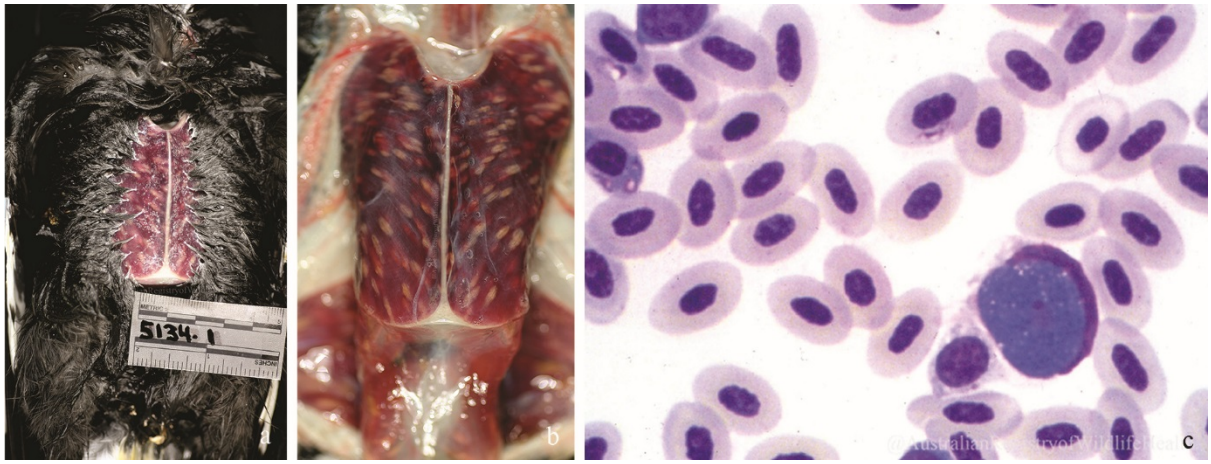

Figure 12 Lesions due to Leucocytozoon sp. in the pectoral muscle of a pied currawong before (a) and after (b) the skin is reflected, and Leucocytozoon sp. schizont in a routine blood smear of a pied currawong (c).

Upon post mortem examination of affected birds, discrete, pale oval foci measuring up to 1.5 cm long and 0.5 cm wide are scattered throughout the skeletal muscles, tongue, myocardium and ventricular muscularis externa. Histopathologic examination demonstrates that pale foci consist of central megaloshizonts, surrounded by necrotic muscle and an intense inflammatory response. Haemorrhage, necrosis, and inflammation are most severe around ruptured megaloschizonts. Pigmented oval *Leucocytozoon* gamonts may or may not be evident within blood of affected birds, and are generally most concentrated in renal tissue.

Haemoproteus columbae were first described from the rock (feral) pigeon in 1890 and *Haemoproteus* sp. have since been described from a variety of bird species. The taxonomy of *Haemoproteus* sp. has been subject to many restructures as DNA sequencing technology evolves. The vectors for these parasites are believed to be hippoboscid flies and infection is generally mild and incidental. In severe infections, birds may develop anaemia, become reluctant to move, anorexic, appear fluffed-up, become lame, have difficulty breathing, or simply be found dead. Post mortem findings may include enlarged spleen, liver, kidneys, or gizzard, chocolate-brown coloured organs, and large skeletal muscle cyst-like lesions similar to those described above for *Leucocytozoon* infections. Blood films and tissue impression smears may contain infected blood and skin cells respectively.

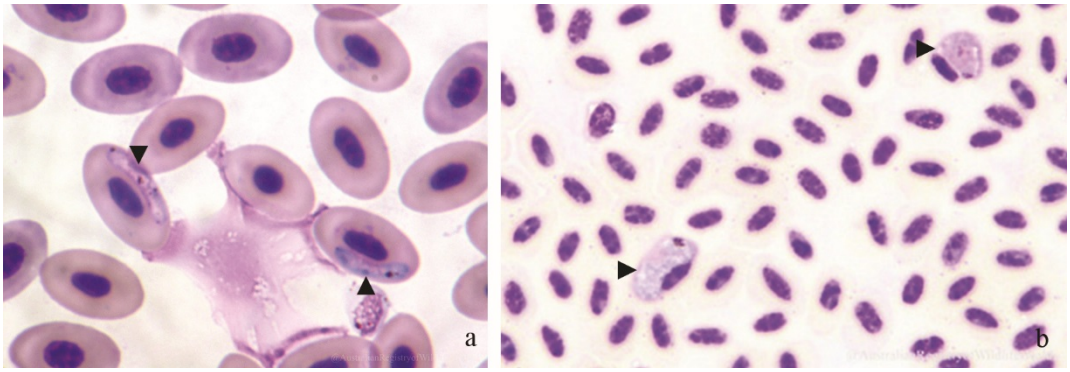

Figure 13 Haemoproteus sp. in erythrocytes on blood smears from a little penguin (a) and an Australian magpie (b)

The family *Plasmodiidae* includes the genus *Plasmodium* which is responsible for malaria infections. The vectors for this organism are typically mosquitoes and infection may be incidental, severe or fatal. Naïve populations are most at risk of severe disease. Birds may present weak, thin, or dead. Gross findings may include an enlarged, dark brown spleen or liver. Blood films and tissue impression smears are important for diagnosing avian malaria. Organisms can be difficult to locate and identify within histological sections.

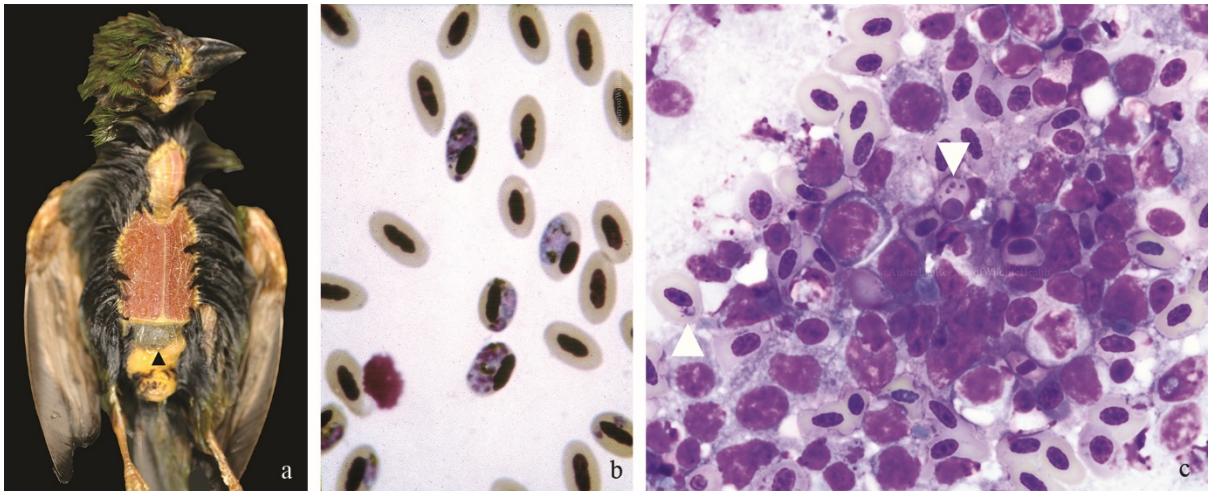

Figure 14 (a) Enlarged liver extending beyond the keel (arrow) of a blue-faced parrot finch with severe Plasmodium sp. infection. (b) Blood smears from a blue-faced parrot finch showing Plasmodium sp. in erythrocytes. (c) Intraerythrocytic Plasmodium sp. (arrows) seen on spleen impression of a little penguin

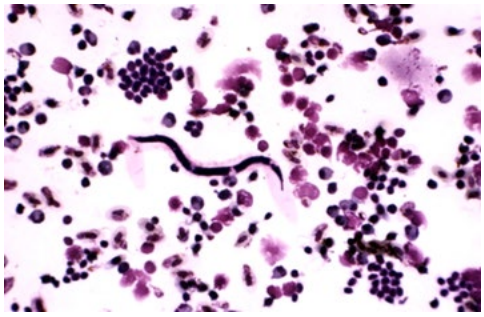

Figure 15 Microfilaria, peripheral blood smear, barking owl

Microfilariae are occasionally found during examination of peripheral blood smears and histological sections of wild birds. Adult filarial nematodes may reside within the air sacs, coelomic cavity, subcutaneous tissues, heart, greater vessels, or lungs, but they are very fine and can be difficult to spot. Infection is diagnosed during microscopic examination of peripheral blood smears or buffy coat smears. Microfilariae are transmitted by haematophagous arthropods. Microfilarial infections are incidental to the host.

Trypanosomes are occasionally found within the peripheral blood of native birds (Zidkova, et al., 2012). These single celled parasites are extracellular flagellates that are transmitted by biting flies or midges. Trypanosomes are reported most commonly in little penguins, and they do not appear to be pathogenic (Jones & Woehler, 1989).

4 Bacterial Disease

Sporadic outbreaks of mortality in native birds have been attributed to localised or systemic infection with *E. coli, Salmonella* spp., *Pasteurella* spp., *Mycobacterium* spp., *Erysipelothrix rhusiopathiae*, *Listeria monocytogenes*, *Streptococcus* spp., *Staphylococcus aureus*, *Haemophilus* spp., *Mycoplasma* spp., and *Clostridium* spp. (Registry). 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 birds in rehabilitation will protect both birds and their rehabilitators.

4.1 Escherichia spp.

Zoonotic: Yes
Species records: various
Similar presentation to: other bacterial pathogens, toxoplasmosis

While *Escherichia coli* can be considered an opportunistic bacterium and a pathogen of concern in its own right, there are several specific strains of *E. coli* that can cause considerable disease in both wildlife, and people. Enteropathogenic *E. coli* (EPEC) causes diarrhoea and mortality in both birds and humans globally. While most reports are from captive birds such as parrots, owls, doves and pigeons, this disease has been recovered from wild rainbow lorikeets during a localised unusual mortality event in Australia (Registry, unpublished) which highlights the importance of wild birds as a reservoir for human disease (Sanches, et al., 2017). Transmission is via ingestion of contaminated food or water, or contact with other infected animals. EPEC adheres to the intestinal mucosa causing severe ulceration and watery or haemorrhagic diarrhoea. In order to identify the specific pathotype, diagnosis is via traditional culture methods followed by molecular typing or via PCR. Treatment is complicated by multi-drug resistance making positive identification with sensitivity testing integral for treatment of infected birds.

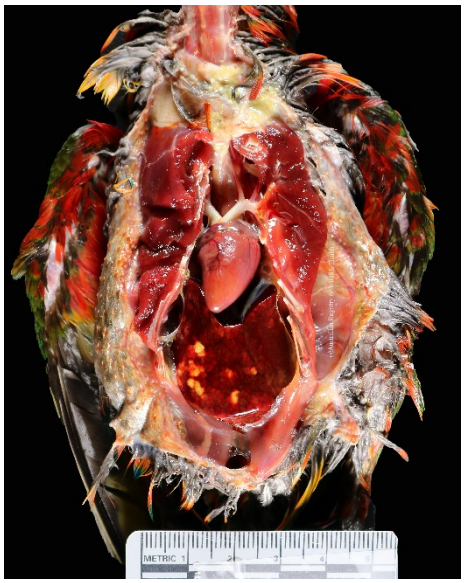

Figure 16 Rainbow lorikeet with multifocal yellow caseous lesions caused by enteropathogenic Escherichia coli (EPEC)

Escherichia albertii is very difficult to distinguish from *E. coli*, is also zoonotic, found in wild and captive birds, and causes diarrhoeal disease and sudden death in various bird species. Some birds may act as carriers (Wildlife Health Australia, 2013). Dead birds are often in good body condition but gross lesions may vary depending on species infected. Infection has been recorded in spotless crake, chickens, Australian magpie, little corella, galah, rainbow lorikeet, brown-headed honeyeater, grey fantail and superb fairy-wren from the east coast of Australia, but not in Western Australia or Tasmania (Wildlife Health Australia, 2013). Transmission is by ingestion of food and water contaminated by infected faeces. *E. albertii* is capable of surviving in the environment for some time under optimal conditions, therefore hygiene of bird feeding and bathing stations is essential. Diagnosis can be made from culture of faeces or cloacal swabs in live birds, or liver and spleen in dead birds, on MacConkey agar. PCR is also required to definitively identify *E. albertii*.

4.2 Yersiniosis

Yersinia pseudotuberculosis infections can result in either acute enteritis and septicaemia (systemic bacterial infection, most often spread in the circulatory system), or multisystemic abscesses. Some birds show no symptoms and are simply found dead while others may show chronic diarrhoea, weakness, ruffled feathers, lameness and progressive weight loss.

4.3 Necrotic Enteritis

Wild rainbow lorikeets, scaly-breasted lorikeets, and king parrots in coastal eastern Australia are seasonally affected with necrotising enteritis. A variety of organisms, primarily coliforms, have been isolated within the necrotic intestinal tissue. *Clostridium perfringens*, *Cl. tertium*, and *Escherichia coli* are most commonly isolated within the intestine and other tissues of birds with necrotic enteritis. It can be difficult to interpret these findings, as these organisms can be normal residents of the intestinal tract.

An investigation into the occurrence of necrotic enteritis identified 58 dead rainbow lorikeets, red-collared lorikeets, and scaly-breasted lorikeets originating from 18 different flocks in eastern Australia over a ten-year period (McOrist & Reece, 1992). *Cl. perfringens* was isolated from the intestinal tissues

of many birds, and beta toxin was demonstrated within the bacterial colonies and within intestinal content using gas liquid chromatography (McOrist & Reece, 1992).

Carbohydrate overload has been suggested as a means of causing intestinal overgrowth with Clostridium sp., and subsequent necrotic enteritis (Pass, 1993; McOrist & Reece, 1992; Ferrell & Tell, 2001). Artificial feeding stations established for lorikeets in urban areas which provide only sugar water, and have questionable hygiene for cleaning surfaces between feeds, are potential sources of infection. The presence of an underlying viral infection in birds suffering from necrotic enteritis, has not been thoroughly investigated.

Necrotic enteritis occurs in male and female birds, juvenile animals and adults. Free-ranging birds are most commonly diagnosed with necrotic enteritis; however, the disease has been observed in captive lorikeets. Necrotic enteritis is most often observed in July and August.

Necrotic enteritis is identified based upon the clinical signs and microbial culture of faeces. Many birds with necrotic enteritis are found dead. Post mortem examination and microbial culture of segments of intestine, liver and other filtering organs are used to establish a diagnosis.

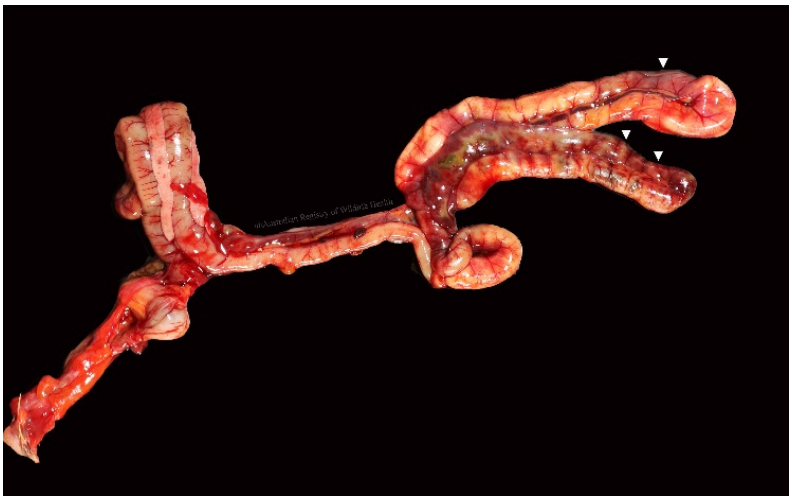

Figure 17 Necrotic segments in the small intestine (arrows) of a Rainbow Lorikeet

Live birds with necrotic enteritis exhibit a variety of clinical signs. Most of these birds are in good body condition, but are weak, depressed, dehydrated, regurgitate clear fluid, and have soiled vent feathers as a result of watery diarrhoea. The bird's abdomen may be palpably distended. Species affected by necrotic enteritis normally have wet faeces, and the detection of diarrhoea may be challenging.

During the gross post mortem examination of these birds, the intestinal tract is distended by gas or reddish-brown fluid. A tan inflammatory (diphtheritic) membrane coats the mucosa, or the mucosa is found to be friable and haemorrhagic. Microscopic examination of intestinal scrapings illustrates large numbers of bacterial colonies, epithelial and inflammatory cells. Histological examination of formalin fixed segments of intestine reveals the following: mucosal to transmural necrosis, intense mononuclear cell infiltration, oedema and congestion throughout the lamina propria and submucosa, and colonies of bacteria scattered throughout a superficial layer of necrotic debris and fibrinous exudate.

Although antimicrobial sensitivity testing of bacteria isolated within the intestine of birds with necrotic enteritis guides therapy, treatment of these birds is rarely successful. Presumably, the birds are suffering from either enterotoxaemia or septicaemia by the time they demonstrate clinical signs and reach care.

4.4 Chlamydiosis

Zoonotic: Yes
Species records: sulphur-crested cockatoos, little corellas, crimson rosellas, king parrots, feral (rock) doves
Similar presentation to: septicaemia, toxoplasmosis, intoxication, malnutrition

Chlamydophila psittaci is a bacterium of the family *Chlamydiaceae*. This bacterium is capable of causing severe disease in free-living birds, aviary birds and humans. When this zoonotic bacterium infects humans, it is referred to as psittacosis, and may cause serious pneumonia. *C. psittaci* is endemic throughout Australia. It is a notifiable disease. Avian infections with this organism are termed ornithosis, or chlamydiosis. Psittacine and columbiform birds are most susceptible to *C. psittaci* infection. Chlamydial disease is most common in rosellas, and to lesser extent lorikeets, cockatoos, budgerigars, and king parrots, but any bird can be affected and a potential source for human infection.

C. psittaci is transmitted through the faecal-oral route, respiratory secretions or the inhalation of organisms in aerosolised feather dander or faeces. *C. psittaci* is highly infectious and a single organism can cause severe disease in birds and humans. The bacterium can form compact elementary bodies that can remain infective within the bird, dried faeces or dander for several months. Chlamydiosis should be considered among the differential diagnoses in any emaciated wild bird, and barrier methods should be employed to prevent potential spread of infection to other wildlife or humans.

Birds with active chlamydiosis may exhibit a broad range of symptoms associated with either acute or chronic disease. Many birds will function as asymptomatic carriers of the organism, while others may suffer severe or fatal infection. Chlamydiosis is most often manifested as respiratory or gastrointestinal illness. Neurological signs are rare, but a recognised outcome of avian chlamydiosis. Clinical signs associated with *C. psittaci* infection include: weight loss, depression, lethargy, anorexia, diarrhoea, bile stained faeces, ocular or nasal discharge, and dyspnoea (difficult or laboured breathing). Monocytosis is a common haematological finding in affected birds and on occasion, elementary bodies may be visible within monocytes, particularly if concentrated in a buffy coat preparation.

Post mortem findings can be highly variable in birds suffering from chlamydiosis. Some birds may die acutely with very few morphologic lesions, while some may exhibit splenomegaly and hepatomegaly, and others may have fibrinous air sacullitis, pericarditis and enteritis.

Definitive diagnosis of chlamydiosis relies upon detection of the organism using PCR. Marked leucocytosis, monocytosis, and an elevated AST may be suggestive of *C. psittaci* infection, however, there is significant variability in the haemogram of birds with chlamydiosis. Antigen can be detected within conjunctival, nasal, or faecal swabs using antigen capture ELISA tests or direct immunofluorescence testing. Diagnostic tests based upon antigen capture are highly sensitive, but may not be highly specific. Some gram negative bacteria will cross react with the antibody used in the ELISA test, thus, conjunctival and choanal swabs will provide far fewer false positive reactions compared with faecal swabs. ELISA based antigen capture test kits are commercially available for in-house identification of *Chlamydia* sp. antigen. These kits are marketed for the detection of human *C. trachomatis* within urine samples, but they are effective in the identification of *C. psittaci*.

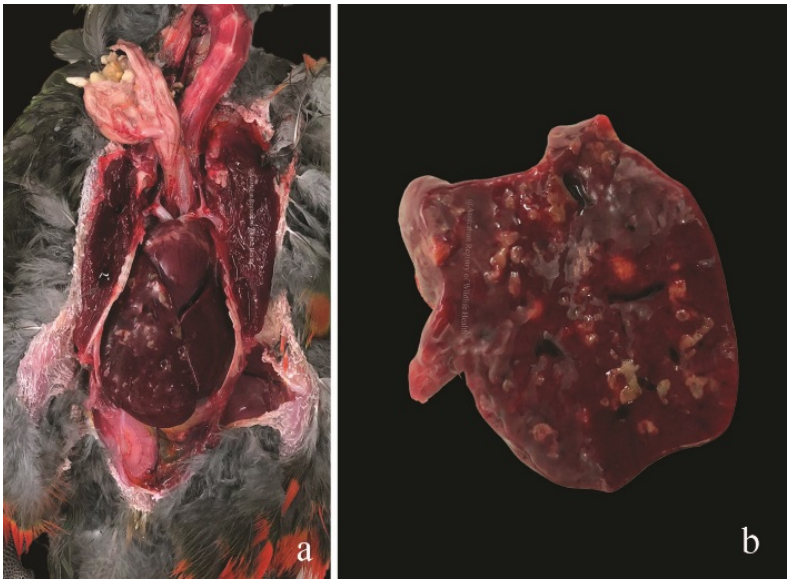

Figure 18 King parrot liver with scattered, randomly distributed, coalescing areas of cavitation and yellow-brown discolouration (necrosis) positive for Chlamydia psittaci by PCR.

Post mortem diagnosis of chlamydiosis is usually based on finding multisystemic histiocytic inflammation on histologic examination and identification of the organism within lesions. Methanol fixed impression smears of spleen, lung, and liver can be stained using modified Machiavello's staining protocols. This protocol can also be used to identify the organism within paraffin embedded tissue, but organisms are easier to identify within tissue impression smears. Fresh tissues, such as liver, spleen and lung may be submitted fresh, or after freezing at – 80°C, to a microbiology laboratory for culture, or swabs from fresh tissues can be tested with antigen capture ELISA tests or PCR. Immunohistochemical demonstration of the organism is possible in fixed tissues. Always communicate your concern regarding the possibility of chlamydiosis when submitting samples to a laboratory, museum, taxidermist or researcher.

5 Viral Disease

5.1 Circovirus: Psittacine Beak & Feather Disease (PBFD)

Zoonotic: No
Species records: sulphur-crested cockatoos, rainbow lorikeets, many others
Similar presentation to: feather mites, fungal infections, feather trauma, nutritional insults

Psittacine circovirus, the causative agent of Psittacine Beak and Feather Disease (PBFD), was listed as a key threatening process for the survival of 5 endangered species, including the orange-bellied parrot, in 2004 by the Commonwealth Government.

PBFD is a common disease in wild and aviary psittacines throughout Australia. PBFD has also been identified in rainbow bee-eater, Gouldian finch, zebra finch, powerful owl, boobook, brown goshawk, laughing kookaburra, Australian raven, tawny frogmouth, Australian white ibis, and wedge-tailed eagle (Phalen, 2019). The disease is manifested by lesions in the feathers, beak and occasionally the claws. Disease transmission may occur via the faecal-oral route, feather dust, regurgitated crop contents or possibly through the egg. Raptors may become infected though the consumption of infected parrots (Phalen, 2019).

Clinical signs of infection with psittacine circovirus are highly variable depending on the age and species of the bird, and the quantity of virus in the infective exposure. The progression of disease is

also highly variable, ranging from acute to chronic. Young birds most often exhibit the acute form of infection. Clinical signs of acute psittacine circovirus infection include diarrhoea, weight loss, anorexia, depression and either death or residual feather damage. The chronic form of psittacine circovirus infection in cockatoos begins with loss of the powder keratin in the plumage, and the production of abnormal down feathers over the hips. Powder down feathers become short and lose the plumaceous barbs. The loss of powder down feathers results in a dull and dirty look of contour and flight feathers, and imparts a glossy black appearance to the beak.

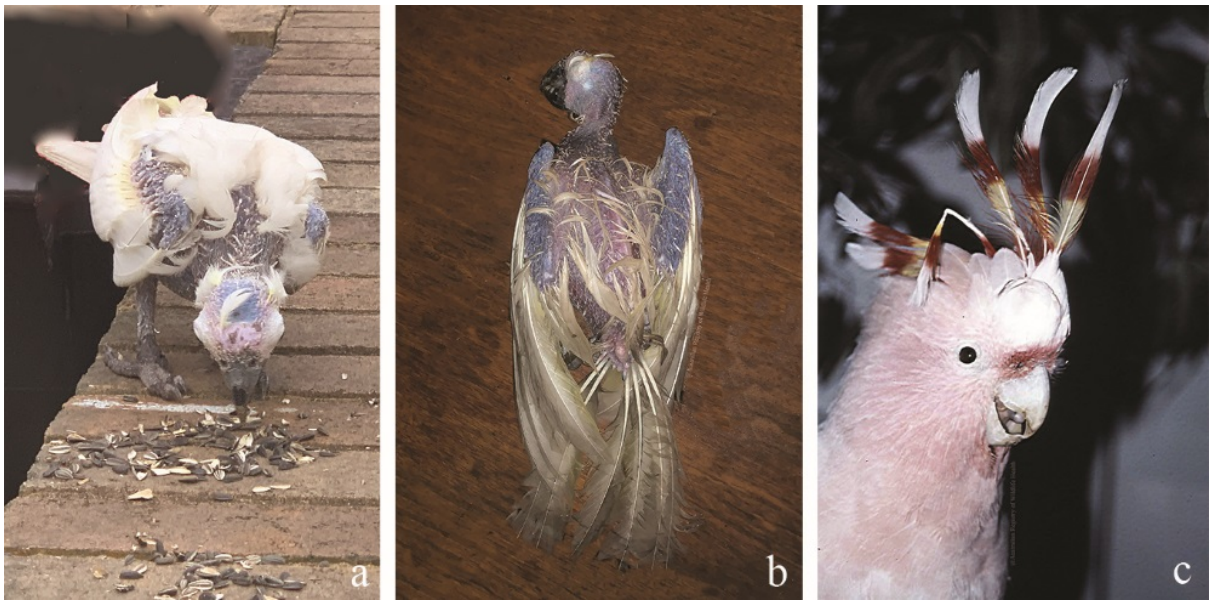

Figure 19 Sulphur-crested cockatoo with moderate (a) and severe (b) clinical signs of Psittacine beak and feather disease (Images courtesy of Dr John Martin), and a Major Mitchell's cockatoo with mild crest feather lesions caused by infection with PBFD.

When damaged by psittacine circovirus, the beak may become elongated, softened, broken, cracked, or it may have uneven wear. These changes are most commonly seen in cockatoos in the late stages of infection. If the germinal epithelium of the beak is exposed by fractures or cracks in the keratin, the bird will often stop eating due to pain.

Young lorikeets of the genus *Trichoglossus* that are infected with psittacine circovirus will often present with the last two to four primary feathers of the wings broken or missing. If pulled from their follicles, the calamus of the remaining tail feathers and flight feathers will often exhibit characteristic morphologic lesions. These lorikeets are called “runners” since they are unable to fly, yet healthy enough to forage and run on the ground. Young lorikeets are identified by their dark brown beaks. Occasionally, these young lorikeets will have a blotchy yellow pattern on the tail feathers that are usually green. Beak lesions rarely occur in lorikeets infected with psittacine circovirus.

Feathers damaged by psittacine circovirus are curled, clubbed, easily broken, or they have retained feather sheaths, haemorrhages within the calamus (shaft), or annular constrictions of the calamus. Replacement feathers grow slowly, or fail to regrow.

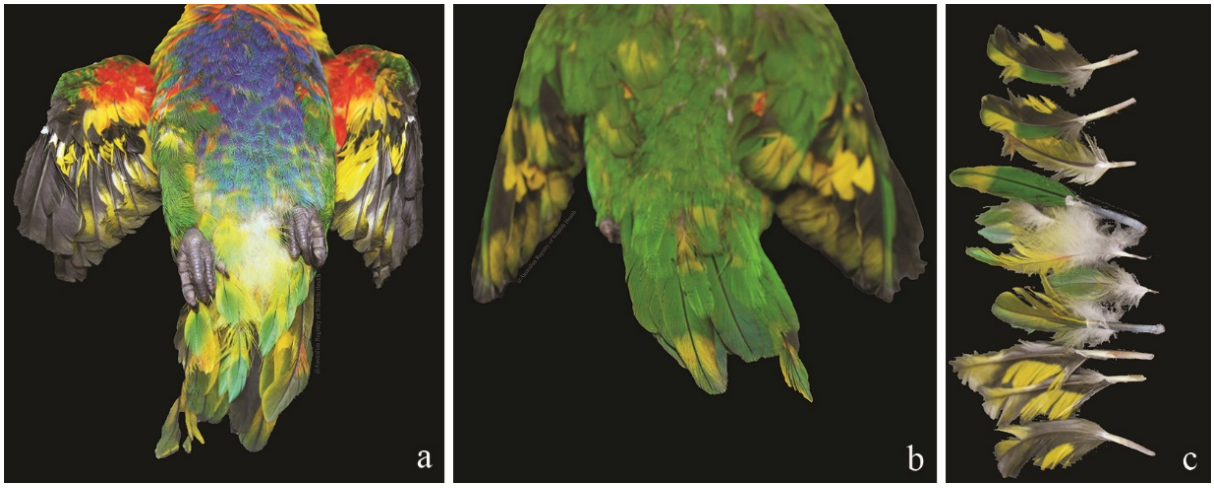

Figure 20 Rainbow lorikeet with Psittacine Beak & Feather Disease (Circovirus) showing stunted tail and wing feathers including missing primary wing feathers (a and b), and deformed calamus of pulled feathers (c) (Images courtesy of Dr Lydia Tong).

Histologic lesions associated with psittacine circovirus occur primarily in the growing feather, but may also be evident within the follicle. Necrosis occurs in the germinal layer of the follicular and feather epithelium, and basophilic cytoplasmic inclusion bodies may be evident within the epithelium, and in reticuloendothelial cells in the dermis, feather pulp, and bursa of Fabricius.

Psittacine circovirus infects the thymus and bursa of Fabricius and is associated with lymphoid necrosis and premature atrophy of these tissues, resulting in immunosuppression. Birds with psittacine beak and feather disease often succumb to secondary viral, bacterial or fungal infections.

The presumptive diagnosis of psittacine beak and feather disease is based upon gross and microscopic lesions in the feather and feather follicle.

Figure 21 Juvenile White-bellied sea eagle with feather damage to both wing and tail feathers and prominent lesions of the feather calamus (insert). Images courtesy of Dr L Tong.

Psittacine circovirus is very difficult to isolate in culture. Definitive diagnosis of infection with this virus can be established through serological testing, which is available through commercial and academic laboratories. Haemagglutination inhibition (HI) testing detects antibodies to psittacine circovirus in blood, serum and yolk, while haemagglutination (HA) testing detects the virus in faecal samples or feathers. Birds that suffer from severe psittacine beak and feather disease may not mount an effective immune response to the virus and their titres measured via HI tests may not be elevated. Elevated HI titres merely indicate that antibodies have been formed in response to exposure to psittacine circovirus. Birds with either the acute or chronic form of circovirus infection often have low titres. Budgerigars, lorikeets, and king parrots, however, usually have high psittacine circovirus titres and continue to shed the virus.

Immunohistochemistry, PCR, and DNA *in-situ* hybridisation tests for psittacine circovirus are available overseas, while PCR, HI and HA are primarily used in Australia. In a live bird, a blood spot on filter paper and affected feathers can be tested (Phalen, 2012).

Some species of psittacine can spontaneously recover from the acute form of beak and feather disease. Rainbow lorikeets, budgerigars, eclectus parrots and king parrots may recover from this infection with only mild residual feather changes. Birds with the chronic form of psittacine circovirus infection rarely recover, but they can live for several years. The cause of death most often relates to secondary infection with other viral, bacterial or fungal agents as a result of immunosuppression.

There is no known cure for PBFD. Nursing care to keep the bird warm and eating will prolong the life of cockatoos. Lorikeets may spontaneously recover from psittacine beak and feather disease, but can shed the virus for a prolonged period.

Since there is no effective treatment for birds suffering from PBFD, controlling the spread of the virus relies of strict hygiene and euthanasia of affected birds. If euthanasia is not under consideration, affected birds should be maintained under strict quarantine. Viracidal disinfectants used to kill parvovirus should inactivate psittacine circovirus.

5.2 Poxvirus

Australian magpies, native pigeons and raptors are occasionally clinically affected by poxvirus infection. Poxvirus is a member of the genus Avipox, which has a worldwide distribution. Poxvirus is shed in saliva, nasal secretions, faeces and wound exudates or scabs. The virus is stable for several months in the environment under favourable conditions. Poxvirus is transmitted primarily by haematophagous arthropods, such as mosquitos; however, other vectors and fighting can also result in transmission. Infection results in viraemia and then localisation within the skin or mucosa.

Clinical signs of poxvirus infection vary from blistering and small nodules in the skin to large dermal nodules with markedly hyperplastic epithelium, which may have foci of ulceration. These lesions primarily occur on the skin of the feet, legs and head, and around the eyes, mouth and cloaca. Secondary bacterial infection is a common finding in poxvirus lesions. Some birds will recover spontaneously, while others will become debilitated due to difficulty walking or obtaining food.

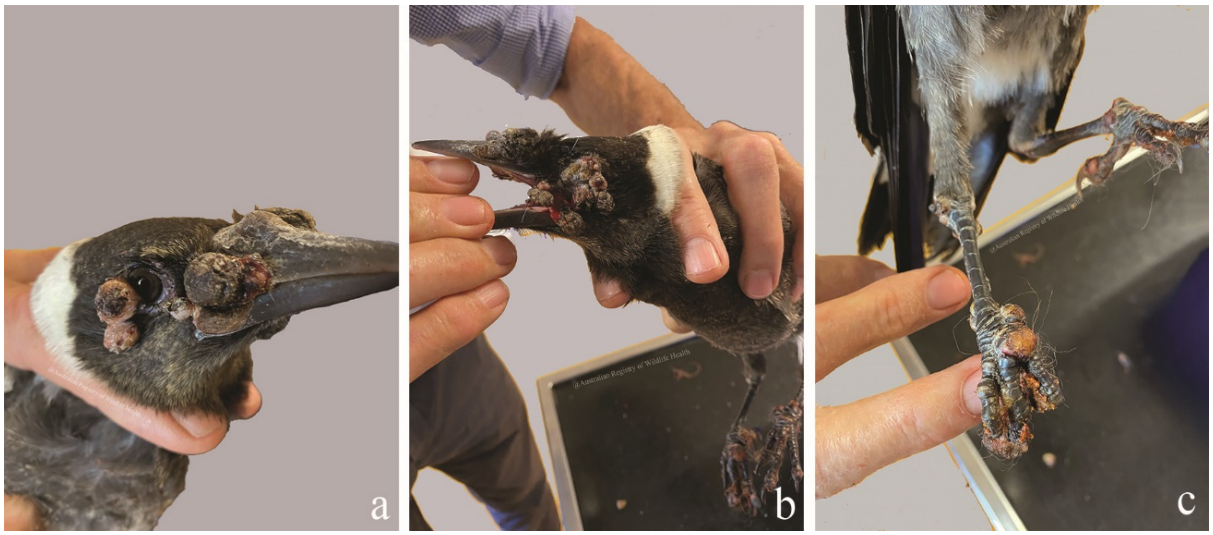

Figure 22 Pox lesions on the face, commissures of the beak, and feet of an adult Australian Magpie (Image courtesy of Dr Bryn Lynar, Pittwater Animal Hospital)

The microscopic lesions associated with poxvirus infection include marked epidermal thickening. Hyperplastic epithelial cells may contain cytoplasmic vacuoles that house large, eosinophilic inclusion bodies. These inclusion bodies are called Bollinger bodies.

A diagnosis of poxvirus infection is based on finding the characteristic intracytoplasmic eosinophilic inclusion bodies within epithelial cells upon microscopic examination of formalin fixed biopsy or tissue sections. Some diagnosticians can use Diff Quik® stained scrapings of the proliferative lesions to identify intracellular inclusions, but this is very difficult. Alternatively, biopsies of the proliferative wounds can be submitted for electron microscopy to look for viral particles.

Many birds will respond to cage rest and nursing care. Surgical debulking of large lesions may provide relief for some birds where lesions are preventing breathing or food prehension. Nutritional support and prevention of secondary infections will aid in the recovery of many birds suffering from poxvirus infection.

5.3 Adenovirus

Evidence of Adenoviruses by PCR have been found in a wide variety of psittacines, passerines and birds of prey including, but not limited to, rainbow lorikeets, galahs, sulphur-crested cockatoos, silver gulls, pigeons, orange-bellied parrots, red-bellied parrots, and purple-crowned lorikeets (Phalen, 2019; Vaz, et al., 2020). Acute lethargy and death, haemorrhagic droppings, mottled, friable, enlarged and rounded liver, and dilated loops of intestine have been described (Phalen, 2019). Lesions associated with adenovirus infection can be seen in the conjunctiva, kidneys, bursa, liver, spleen, or intestines and large basophilic to eosinophilic intranuclear inclusion bodies are characteristic on microscopic examination of tissues (Phalen, 2019). Birds may be asymptomatic, subclinical, succumb to other more debilitating co-infections, or harbour latent disease (Phalen, 2019). Diagnosis of adenovirus infection in live birds is based on Adenovirus specific PCR of faeces. Whereas post mortem diagnosis is based on observation of inclusion bodies upon microscopic examination of tissues, PCR of liver and other organs, and possibly electron microscopy targeting inclusion bodies.

5.4 Avian Herpesvirus

Zoonotic: No
Reportable: Yes (Duck herpesvirus-1)
Species records: pigeons, powerful owls, Australian hobby, Gouldian finch, eclectus parrot, Bourke's parrot
Similar presentation to: poxvirus, trichomoniasis, yeast, *Pseudomonas* spp., salmonellosis, adenovirus

Psittacid herpesvirus 1 (PsHV-1) is a reportable disease of birds in Australia (Phalen, 2012). Infections can be subclinical and activation of the virus may become pronounced in an immunocompromised bird therefore disease patterns associated with avian herpesviruses infection can vary. Acute infections can result in necrosis of the gastrointestinal or respiratory tract, while chronic infection may manifest as papillomatous lesions of the skin, gastrointestinal tract and bile duct (Phalen, 2012).

Diseases such as Duck Virus Enteritis (duck plague), columbid herpesvirus, Pacheco's disease (mucosal papilloma), pscittacid herpesvirus 2 and 3, passerid herpesvirus, Loon herpesvirus, frigatebird herpesvirus, and vulturine herpesvirus have all been described (Phalen, 2012). Columbid herpesvirus has been found in high prevalence in pigeons worldwide and has caused several loft mortalities in Australia in recent years. In Australia, as well as in North America, acute deaths of wild and captive owls and falcons that have fed on infected pigeon carcases have been recorded.

Herpes infected birds may be found dead, or exhibit clinical signs including anorexia, nasal or ocular discharge, sneezing and difficulty breathing, cere changes, oral, oropharyngeal or nasal passage ulcers, or green or sulphur coloured urates (Phalen, 2012). Inclusion bodies are usually present in liver and other tissues microscopically.

6 Fungal Disease

6.1 Aspergillosis

A variety of fungi are capable of causing respiratory infections in birds, however, *Aspergillus fumigatus* is the organism most commonly isolated within mycotic lesions. *A. fumigatus* is considered to be an opportunistic pathogen that causes disease in birds that are otherwise debilitated by bacterial, viral, nutritional, traumatic, or toxic disease.

Aspergillosis is primarily a disease of captive birds, however, wild birds may also contract this infection. Spores of *A. fumigatus* are common within the environment. Captive birds may be exposed to spores that occur within food, bedding or nesting material.

Wild rainbow lorikeets that are infected with psittacine circovirus are commonly presented to wildlife care centres with extensive mycotic pneumonia. Birds of prey, aquatic birds, and some passerine birds seem to be particularly susceptible to infection with *A. fumigatus*.

Birds with mycotic lesions in their respiratory tracts may exhibit a variety of clinical signs. Initial changes may reflect a subtle change in the voice, weakness, weight loss, or oculonasal discharge. Infection may become dormant, yet will most often progress to depression, emaciation, coughing or sneezing, and dyspnoea.

Birds with clinical aspergillosis will have a marked leucocytosis. Heterophilia occurs in the early stages of disease, and monocytosis reflects more chronic infection. Active fungal infection may result in toxic change within heterophils, characterised by irregularly shaped granules and cytoplasmic vacuoles.

A. fumigatus may cause focal infections of the upper respiratory tract, in the sinuses or trachea. More often aspergillosis is associated with lesions within the pulmonary parenchyma and air sacs. Regardless of the location of the lesion, mycotic plaques are formed that have a white to yellow capsule, and may have a green centre or surface. This green coloration represents the presence of conidiophores, or fruiting bodies.

The histological appearance of aspergillosis is quite variable. Early lesions most often reflect focal granulomatous inflammation. Fungi often have an affinity towards blood vessels. *Aspergillus* sp. may spread from the primary site of infection into blood vessels resulting in haemorrhage and necrosis of adjacent tissues.

Endoscopic examinations are used to visualise fungal plaques within the air sac, lung or trachea of live birds. Cytologic examination of oculonasal discharges, or infraorbital lavage fluid may be useful in the diagnosis of mycotic sinusitis. Clinical signs and the presence of leucocytosis may also be suggestive of aspergillosis. Serology for the detection of aspergillosis is available at commercial veterinary laboratories. Radiographic examination may reveal multifocal densities within the lungs, trachea, or air sacs that reflect the presence of fungal plaques. Impression smear and Diff-Quick staining of plaques can be examined microscopically to form a presumptive post mortem diagnosis of aspergillosis. Definitive diagnosis is dependent upon microbial culture and isolation of the organism within lesions.

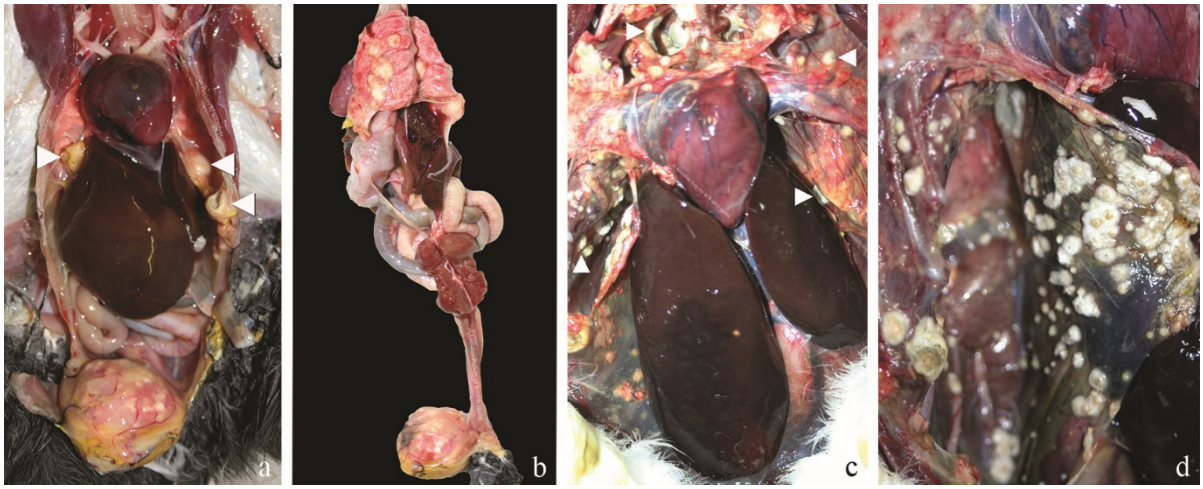

Figure 23 a,b) Pied currawong, multiple white-yellow granulomas, lungs (arrows), and massively expanding bursa, c,d) Australasian gannet with characteristic fluffy white-green lesions, air sacs and lungs caused by Aspergillus fumigatus.

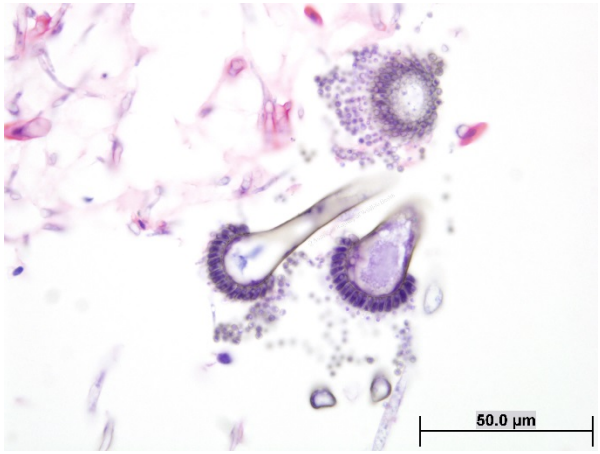

Figure 24 Conidiophores with associated spores of Aspergillus fumigatus.

The prevention of aspergillosis in captive birds is based upon strict standards of hygiene. Bedding materials used for the care of birds should be clean and dry. Straw, hay, and other agricultural products should not be used as bedding or nesting material for birds. Bird food should be properly stored and regularly examined for the presence of fungi. Any husbandry technique that minimises the stress experienced by captive birds is likely to reduce the risk of aspergillosis. Some species are known to be particularly susceptible to aspergillosis. These birds are often treated with antifungal agents on a prophylactic basis.

6.2 Candidiasis

Candida spp. are yeasts that are commensal within the upper gastrointestinal tract of a variety of birds. Disease associated with an overabundance of this organism occurs most commonly in young, hand-raised birds. Candidiasis in young birds is associated with poor hygiene, and inappropriate hand rearing formulas. Adult birds that are administered broad-spectrum antibiotics orally or those birds that have concurrent systemic illness are also susceptible to candidiasis. Occasional feather loss and hyperkeratosis in captive native birds have been associated with external candidiasis, but most of these birds also have yeast overgrowth within their gastrointestinal tracts (Registry).

Overgrowth of *Candida* sp. within the oral cavity, oesophagus, proventriculus and ventriculus results in weight loss, depression, anorexia, crops stasis, regurgitation, and diarrhoea. Oral infections may result in visible white plaques along the mucosa.

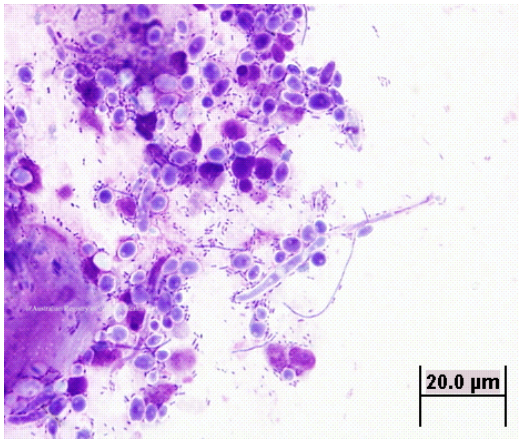

Figure 25 Budding yeasts and hyphae of Candida albicans stained with Diff Quik

Diagnosis of candidiasis relies upon cytologic examination of smears made from oral lesions. Wet preparations of faeces may be examined microscopically for the presence of yeast. Gram stains and Diff Quik® stains are useful to illustrate the presence of yeast within dry smears. *Candida* spp. are commensal within the gastrointestinal tract, and scattered yeast cells within tissue smears of faeces are not unusual. The presence of large numbers of budding yeast, and pseudohyphae reflect active infection with *Candida* sp. *Candida* sp. can also be identified through standard fungal culture techniques.

6.3 Macrorhabdus ornithogaster

Formally known as megabacteria, *Macrorhabdus ornithogaster* is a yeast that grows exclusively on the mucosal surface of the junction between the avian proventriculus and ventriculus (Phalen, 2008). *M. ornithogaster* has been described in both captive and wild birds worldwide. Most infections do not result in clinical disease however in cases where disease is present, birds appear thin and may exhibit thickening of the proventricular mucosa, increased mucous content in the proventriculus, and ulceration of the proventriculus or ventriculus which may lead to acute haemorrhage (Phalen, 2008).

A wet mount preparation of mucosal scrapings can be a useful diagnostic tool for confirming infection with *M. ornithogaster*. Microscopically, *M. ornithogaster* is Gram and PAS positive, appearing as tightly packed, dense rafts of long yeast cells that resemble filamentous bacteria. The organism may be present on the mucosal surface, distending proventricular glands or penetrating the koilin layer.

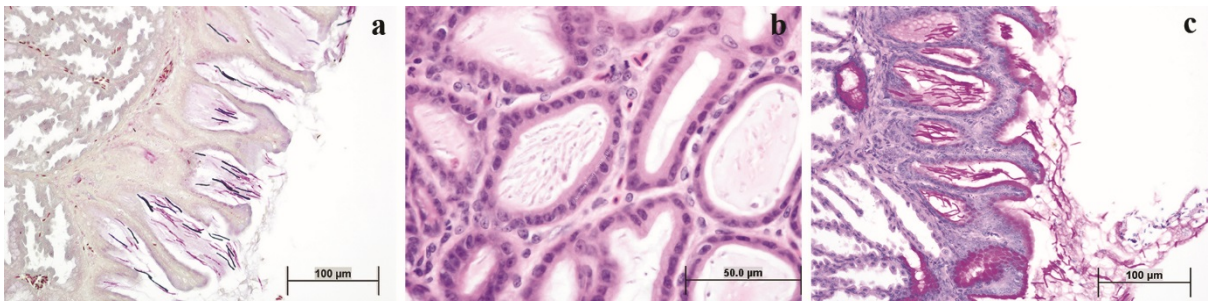

Figure 26 Macrorhabdus ornithogaster in the proventriculus of a Zebra Finch stained using a) Brown & Brenn Gram stain, b) H&E stain, and c) PAS stain

6.4 Mycotoxins

Mycotoxins are toxic products that can be produced by a wide variety of fungal species. Exposure to mycotoxins occurs when birds ingest mouldy grain, grasses or other foodstuffs. The clinical signs of mycotoxicosis will depend upon the type of mycotoxin ingested. Clinical signs may include oral ulcers, and aberration of the gastrointestinal tract, liver, kidney or central nervous system. Diagnosis of mycotoxin exposure is difficult since few laboratories will isolate mycotoxic fungi and quantify the toxins.

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 or other infection could result in altered nutrient absorption.

7.1 Nutritional Osteodystrophy

Figure 27 a) Australian white ibis, soft beak, malnutrition b) sulphur-crested cockatoo, skeletal deformities including feet and keel - sunflower seed diet, c) semi captive guineafowl, malnutrition and keel deformity (arrow), crop parasites (asterisk)

The clinical syndrome associated with nutritional osteodystrophy is called rickets in growing birds, and osteodystrophy in older birds. Nutritional osteodystrophy occurs as a result of prolonged feeding of diets either deficient in calcium or vitamin D_3, or diets that have a high concentration of phosphorus. Nutritional osteodystrophy occurs most commonly in young birds that are hand raised with inappropriate diets, or free ranging birds such as magpies and kookaburra which are provided with supplemental feeds consisting of lean meat. Lean meat contains scant calcium and abundant phosphorus, which can trigger nutritional osteodystrophy. The primary lesion in animals suffering from rickets is failure to properly mineralise cartilaginous bone models. This lesion result in stunting, curved long bones, folding fractures of the long bones, a soft beak. Poor feather growth often accompanies these bone changes. Gross post mortem examination may also reveal enlarged parathyroid glands as a result of hyperphosphataemia. Treatment of advanced osteodystrophy bears a poor prognosis. If birds are presented with early lesions, such as a soft beak, and do not have any fractures, treatment through calcium supplementation and an appropriate diet may be successful. Animals with osteodystrophy are often in severe pain and this should be factored into treatment and prognostication.

7.2 Thiamine Deficiency of Red Wattlebirds

Thiamine deficiency in red wattlebirds was thought to occur as a result of altered migration patterns. These birds ceased migrating north for winter due to the presence of flowering ornamental shrubs planted in suburban gardens. Red wattlebirds are both nectivorous and insectivorous; however, they obtain most of their thiamine from insects. Nectar contains only 0.01 to 0.02 *u*g thiamine per kilojoule of energy (Pass, 1993). Insects normally supply both amino acids and thiamine (4 *u*g thiamine per gram

of insect). Research has demonstrated that red wattlebirds require 200 kilojoules of energy per day and 20 *u*g of thiamine per day (Paton, et al., 1983). Thus, a red wattlebird can obtain its daily thiamine requirements by eating as few as five large insects. Large insects are not available in Melbourne in winter, and a red wattlebird would have to ingest up to 500 small insects to obtain their daily thiamine requirement. Since only 9% of the red wattlebird's foraging time is invested in searching for insects, it is very difficult for these birds to ensure adequate thiamine intake in winter (Paton, et al., 1983). Rehabilitation centres in Victoria report that they have not seen this condition in many years.

Piscivorous birds maintained in captivity can also develop neurological signs of thiamine deficiency due to the presence of thiaminase in fish. Captive seabirds are usually provided with oral thiamine supplementation. Diagnosis of thiamine deficiency is challenging and response to thiamine treatment is a practical and useful diagnostic tool.

8 Toxicity

8.1 Botulism

Botulism is a paralytic disease of birds resulting from ingestion of toxins created by the bacterium *Clostridium botulinum*. This gram positive, spore-forming bacterium that is capable of forming seven types of botulinum toxin.

Sporadic outbreaks of botulism occur in both urban and rural environments. Significant risk factors associated with botulism include:

- an abundance of bacterial spores in the environment due to previous botulism outbreaks,
- birds that forage at the bottom or margins of freshwater ponds,
- decomposing vegetation, especially at the water margin,
- intermittent flushing of fertiliser run off or sewage into the waterway, which results in surges in plant growth, subsequent plant death and low oxygen concentrations,
- elevated water temperature due to increased environmental temperature and shallow water,
- stagnant water, and
- an abundance of flies.

These conditions can also promote the growth of blue green algae, which can produce toxins. Blue green algae toxicosis should be considered among the differential diagnoses for botulism.

Maggots are relatively resistant to the toxic effects of botulinum toxic. Thus, maggots are an important source of toxicosis as they may accumulate large concentrations of botulinum. Ingesting as few as two maggots that contain botulinum toxin can kill a bird.

Birds suffering from botulism will have paresis or paralysis of the legs, wings, and neck. The first sign of intoxication is usually a drooping head. Impaired vision, difficulty in opening eyelids, difficulty swallowing and paralysis of the third eyelid rapidly follow. Once the legs have become paralysed, the bird may attempt to locomote by weakly flapping its wings. Birds with botulism may succumb to drowning, predation, or they asphyxiate due to paralysis of the respiratory musculature.

Type C toxin is most commonly associated with avian botulism. Botulinum toxin inhibits the release of acetylcholine at the motor end plates resulting in peripheral neuropathy and then loss of muscle control.

Figure 28 A chestnut teal (a) with mild neck and leg paresis, and a mixed breed mallard (b) with severe paresis from an outbreak of botilism in the same lagoon.

Clostridial spores persist in the environment and are very resistant to heat and desiccation. The optimum microenvironment for the growth of *C. botulinum* includes an anaerobic environment, pH ranging between 5.7 and 6.2, high temperatures, and a protein source. Rotting vegetation and carcases of birds provide ideal conditions for the growth *of C. botulinum* and production of botulinum.

Gross and histological examination of birds that have died of botulism are unrewarding. Paralysis, mass mortality and the finding of maggots in the oesophagus or proventriculus are suggestive of botulism.

A presumptive diagnosis of botulism is based upon the clinical signs exhibited and lack of significant lesions on post mortem examination. Definitive diagnosis of botulism requires identifying the toxin within serum or gastrointestinal contents. Mouse inoculation was used to illustrate the presence of botulinum toxin, however, the test is no longer considered ethical. ELISA tests are available for types C and D toxins, but false negative tests are common. Some labs will enrich samples, which means that they will culture the gastrointestinal content and then test for botulinum toxin via ELISA. This enrichment method can easily result in false positive results if rare bacterial spores were passing through the gastrointestinal tract. Recently the West Australian agriculture department has developed a PCR test to identify genetic elements attached to the toxin molecule and conserved across botulinum toxins. Although still in validation phase, this appears to be a highly specific and sensitive diagnostic test for botulism.

Symptomatic treatment and supportive care are the primary means of treating botulism. Cathartic agents may aid in flushing the source of toxin from the gastrointestinal tract. Botulinum antitoxin has been recommended in the literature; however, it is not commercially available in Australia.

The prevention of botulism in urban environments depends upon management of ponds. Ornamental ponds should be designed to incorporate aeration, water circulation, steep sides and sufficient depth to keep water temperature stable. The layout should prevent water that is rich in organic material from flowing into the pond. Decomposing vegetation and other organic matter should be regularly removed from any pond. Animal carcases should also be regularly removed to prevent the build-up of flies and maggots in the environment.

8.2 Oil Toxicity

Sea birds and shore birds are commonly affected by oil spilled into waterways since they live at the water's surface where oil accumulates. The toxic effects of oil are as diverse as the products spilled. The external effects of oil exposure may include irritation of mucous membranes and displacement of air from the porous structure of the feather. This alteration in feather structure leads to altered function, such as loss of insulating properties, buoyancy, and flight. Internal effects of oil exposure

include aspiration pneumonia, inflammation of the gastrointestinal tract, altered activity of hepatic microsomal enzyme systems and haemolysis. Exposure to oil can also result in altered reproductive behaviour and physiology.

Wildbase Oil Response at Massey University have a wonderful collection of education, training and preparedness tools for responding to oiled wildlife which can be found online. We recommend you consult these resources for up to date advice and recent publications in this field.

8.3 Lead Toxicity

Wild birds are exposed to lead in the form of fishing sinkers and ammunition. Waterfowl ingest the lead pellets that accumulate on the soft mud bottom of waterways. Lead that is lodged within muscle is not a source of lead poisoning. Birds that ingest lead shot embedded in the tissues of their prey, however, may suffer lead poisoning. Captive birds that lick recently galvanised wire mesh may be exposed to toxic concentrations of lead or zinc. Wild birds have also become exposed to lead impregnated dust where this mineral is mined and transported.

Clinical signs of lead poisoning include a depression, weakness, regurgitation, vomiting, diarrhoea, droopy wings, tremors, pallor and convulsion (Locke & Thomas, 1996). Lead poisoning is diagnosed when blood lead concentrations are elevated. Anaemia may occur in subacute to chronic lead poisoning. Radiographic examination of the bird may reveal the presence of radio-dense particles within the gastrointestinal tract.

Treatment of lead poisoning includes removing lead from the gastrointestinal tract using cathartic agents. Chelating agents, such as calcium disodium edetate, bind to lead within the blood stream and aid in its elimination (Locke & Thomas, 1996).

8.4 Organophosphate Toxicity

Organophosphate and carbamate compounds are contained within insecticides, herbicides, and fungicides. A broad range of species are susceptible to the toxic effects of these compounds. Birds and bats can be exposed to organophosphates when they eat contaminated insects or vegetation, and when they fly through aerosolised chemical fogs during application. Unfortunately, access to these organophosphate compounds also occurs through malicious poisoning.

Birds that are exposed to organophosphate compounds may be found salivating, dyspnoeic, ataxic, with tremors, convulsing, paralysed, regurgitating, and with diarrhoea. It has been reported that a delayed-onset neuropathy, defined as a hindlimb paralysis syndrome, in Carnaby's cockatoos may be due to exposure to agricultural organophosphates (Le Souef, et al., 2020). Many animals subject to organophosphate or carbamate toxicity are found dead. Death most often occurs as a result of paralysis of the respiratory muscles and ischaemia.

The effect of exposure to lower concentrations of organophosphates and carbamates is not certain. Reproductive success may be altered due to changes in physiology and behaviour subsequent to exposure to these compounds. Birds experimentally exposed to organophosphates in their food lay fewer eggs, abandon their nests, and have altered feeding and activity patterns (Fairbrother, 1996).

Clinical signs of organophosphate and carbamate toxicity occur as a result of over stimulation of the parasympathetic nervous system, skeletal muscles, and, to a lesser degree, the central nervous system. The gross and histologic examination of birds suffering from organophosphate and carbamate toxicity is usually unremarkable. Occasionally a bird will have visible evidence of diarrhoea, salivation or increased respiratory secretions.

When birds are found dead in good body condition and significant lesions are not evident on gross post mortem examination, toxicity should be suspected. The degree of suspicion should be increased in the face of a focal, mixed species, mass mortality event where bird crops are filled with bait-like material such as bread or commercial seed mixtures. A diagnosis of organophosphate or carbamate toxicity is established through measurement of cholinesterase activity in the blood or brain, or analysis of ingesta for organophosphate and carbamate compounds and metabolites using high pressure liquid chromatography. Samples of the brain are wrapped in aluminium foil and may be frozen prior to submission to a laboratory. Samples of brain and ingesta may be stored for up to six months at -80^{o}C prior to submission to a laboratory.

Atropine is administered to treat intoxication with anticholinesterase compounds. If the bird is not cyanotic, 2-PAM can be administered. 2-PAM will not reverse the effects of carbamate toxicity. Prevention of organophosphate toxicity relies upon judicious use and storage of the chemical agents.

9 Traumatic Injury

9.1 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 urine output. Animals suffering from endotoxic shock, may have bright red mucosa.

Dehydration often contributes to the lack of peripheral perfusion. 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 poor blood flow and poor oxygenation.

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.

Acute necrosis of the proximal renal tubules and periacinar (centrolobular) regions of the liver occurs under conditions of prolonged hypoperfusion. Mucosal ulceration and decreased mobility occur with gastrointestinal ischaema. These gastrointestinal lesions can allow bacteria or bacterial toxins to enter the blood stream. Cells exposed to hypoxia initially undergo degenerative change, but once cell death has taken place, the changes induced may be irreversible. Animals that are treated in this phase of shock may respond to initial fluid therapy, but succumb to acute renal tubular necrosis (urate nephrosis and visceral gout in birds), 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 flow to the brain, and hyperuricaemia, cause nerve cell death.

9.2 Skeletal Injury

Fractures of long bones are commonly encountered in injured birds. The prognosis of return to full function should be carefully considered prior to attempting fracture management. A thorough physical and radiographic examination will assist in the identification of other injuries, such as joint damage, and soft tissue injuries that may have an impact on the bird's overall prognosis. Evaluation of the blood and nervous supply distal to the fracture, and evaluation for potential underlying causes such as bone infection or metabolic bone disease, are imperative prior to mounting any attempts at fracture repair.

Luxations and subluxations are difficult to manage in birds. Bandaging techniques to stabilise the joint also contribute to joint stiffness. Support and physiotherapy are important elements for repair.

Blunt trauma to the chest can result in a transverse fracture of the keel. The irregular fragments of the fractured keel must be forced into the coelomic cavity at the time of trauma, and many of these birds sustain extensive myocardial contusions or hepatic rupture and haemorrhage.

Fractures that occur along the pectoral girdle can be very difficult to palpate, and the bird may only have a droopy wing. Coracoid fractures occasionally have sharp fracture fragments that lacerate the brachiocephalic trunk or the cardiac musculature, resulting in death due to massive haemorrhage. Coracoid fractures should be stabilised as quickly as possible. Radiographic examination of these birds is indicated to assess the full extent of tissue damage. A figure eight bandage and cage rest may result in satisfactory repair of non-displaced fractures. Birds of prey, however, may require surgical correction of fractured coracoid bones through intra-medullary pinning to regain sufficient flight to be releasable.

Radial and ulnar fractures frequently occur in birds subject to trauma. If one bone is intact, and the fracture is non-displaced, conservative management with a figure-eight bandage is often sufficient for bone repair and return to flight. If both the radius and ulna are fractured, the ulna should be treated with either intramedullary pinning and a figure of eight bandage, or external fixation.

Fractures of the carpus and phalanges are associated with extensive soft tissue injury. These fractures often result from high-energy trauma, which produces highly fragmented fractures. The prognosis for return to flight is poor when fractures occur within or distal to the carpus.

Femoral fractures are most often repaired with intramedullary pinning procedures; however, cage rest may be sufficient for return of function when the fracture is non-displaced.

Splints or other external fixation techniques are primarily used to stabilise tibiotarsal and tarsometatarsal fractures. These bones rely heavily upon their medullary blood supply. Intramedullary pinning or KE pins that interfere with the blood supply of these bones may result in ischaemic necrosis.

Fractured toes may be amputated if the wounds are severe. Alternatively, the foot may be bandaged. The contralateral foot must be monitored closely for bumblefoot, due to increased weight bearing.

Spinal fractures and luxations may result in paresis, paralysis and an inability to void the cloaca. Intensive nursing care is required to support these birds, and the prognosis for recovery is guarded when birds have significant neurological deficits. Spinal luxations and fractures most often occur at the junction of the thoracic and lumbar vertebrae. It has been proposed that because the thorax vertebrae are fused and very rigid, thoracic trauma results in damage at the first flexible vertebral junction.

Beak injuries occur occasionally in wild birds. The prognosis for these birds depends upon the degree of damage. If only the tip of the beak is injured, bleeding may be stopped with cautery and the rough edges trimmed. Trimming the opposing beak may aid the bird in prehension of food. Surgical glue, bone cement, fibreglass, or cerclage wire may be used to construct beak prosthetics. Prosthetic devices often require routine monitoring and intermittent replacement and should not be placed on birds to be released. When extensive beak damage occurs in a wild bird, the bird is unlikely to return to a releasable state.

9.3 Soft Tissue Injury

Uncomplicated soft tissue injuries in birds heal relatively rapidly due to effective contraction and epithelialisation. Soft tissue wounds may be left open and a sliding grafting procedure undertaken once there is a healthy granulation bed.

Scalping injuries occur in wild and captive female birds as a result of intraspecific aggression. These injuries can expose a large portion of the cranium. Successful management of scalping injuries most commonly relies upon initial debridement, and wound lavage, followed by a period of open wound management.

Exertional myopathy is occasionally reported in birds subjected to exertion through chase, fear or isometric forces during restraint. Lameness, shifting leg lameness, weakness, and recumbency are clinical signs associated with avian exertional myopathy. Elevated serum concentrations of AST, CK and LDH may occur in affected birds. Recommended treatment regimens for exertional myopathy consist of fluid therapy, glucocorticoid administrations to stabilise membranes, and administration of vitamin E and selenium. Diazepam may aid in relaxation and increase perfusion of injured tissue. Prevention of exertional myopathy is dependent upon minimising stressful stimuli and proper care when capturing and restraining birds.

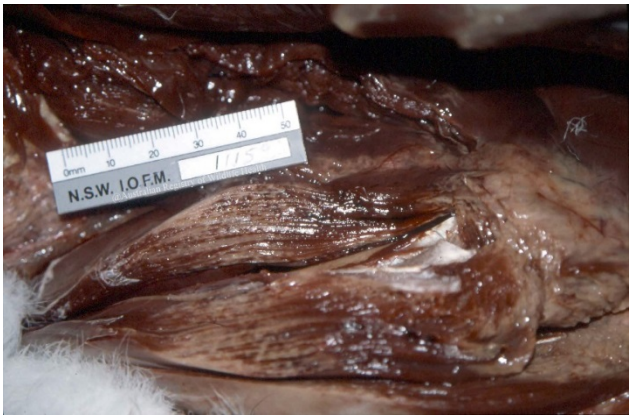

Figure 29 Wandering albatross with marked degeneration of the leg musculature

Pododermatitis (bumblefoot) is a common injury of birds of prey, waterfowl, and pelagic birds in captivity or rehabilitation care. This injury is often attributed to poor hygiene or inappropriate substrates and perches within the bird's enclosure. Calluses, or nodules of hyperplastic epidermis, along the plantar surfaces of the feet are the earliest clinical signs of pododermatitis. These lesions often go unnoticed and progress to ulceration and infection. Chronic ulceration and secondary bacterial invasion may then lead to infectious tenosynovitis.

Treatment of pododermatitis focuses on improved hygiene and perching surfaces. Soft tissue wound management, including wound cleansing, flushing and bandaging, and sometimes surgical debridement are used to accelerate healing of the lesions. Prevention, and early detection and treatment are the best strategy for positive outcomes for birds in care.

Bite wounds 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. Birds may also be predated by other birds, using talons or beaks. Puncture wounds

caused by feline predators are often very fine. These wounds can be difficult to see, and often the only outward sign of attack is moist or matted feathers over the shoulders. Canid-inflicted bite wounds do not necessarily break the skin. Bird predation may be by talon or beak puncture wounds, with beak punctures most commonly found over the cranial region of the skull. 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 over the dorsal thorax, 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). Talon wounds are often associated with feline like deep tissue punctured, generally over the thoracic muscles, while beak wounds often singular and puncture through the skull and into the brain.

Feline bite wounds are often heavily contaminated with *Pasteurella multocida*, or 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.

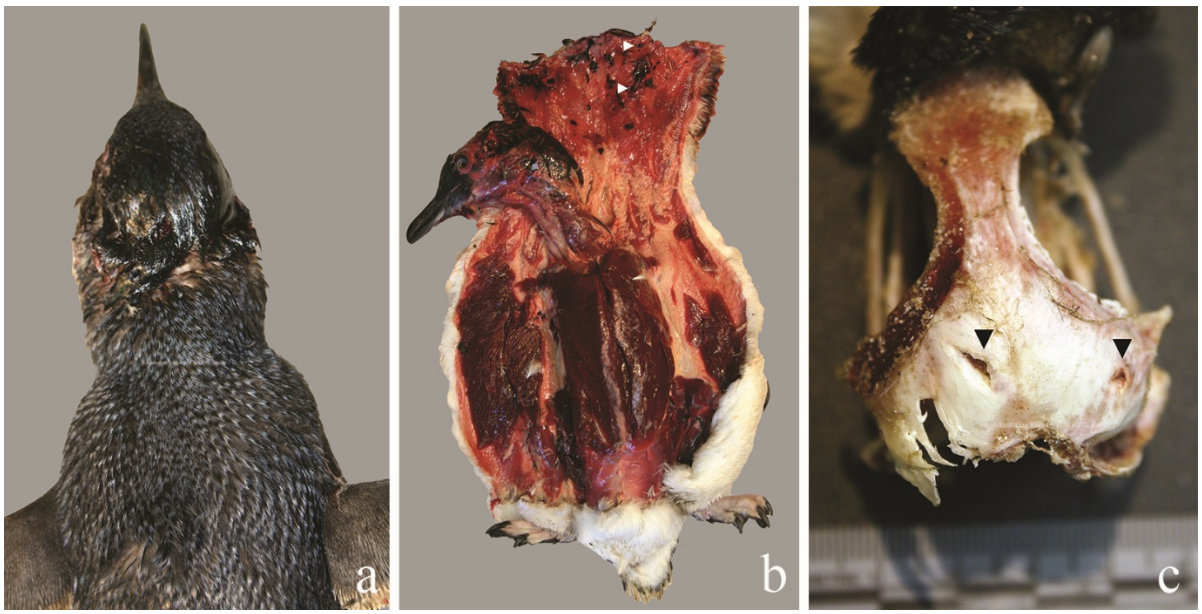

Figure 30 Little penguins showing a) minimal external wounds from bite marks, b) puncture wounds and severe cranial haemorrhage and fracture, c) penetrating bite wounds over skull damaging brain and introducing feathers as a foreign body

9.4 Central Nervous System Injury

Zoonotic: No
Species records: all
Similar presentation to: *Angiostrongylus cantonensis*, intoxication, lorikeet paralysis syndrome, botulism, black and white bird disease, influenza, Newcastle disease, West Nile Virus, toxoplasmosis, etc.

Cranial trauma commonly occurs when birds fly into stationary objects. Blood tinged mucous within the oral cavity, periocular contusions and hyphaema, are often associated with cranial injury. Anisocoria, nystagmus, ataxia, head tilt, tremor, and paresis may also indicate central nervous system trauma. A thorough neurological examination should be conducted, and the presence of deep pain perception evaluated to formulate a prognosis for each bird.

10 Diseases of Unconfirmed Aetiology

10.1 Clenched Claw Syndrome of Rainbow Lorikeets

This neurologic syndrome in rainbow lorikeets has been reported sporadically since the early 1980s primarily along coastal NSW and south east Qld, and to a lesser extent Victoria. The syndrome has not been documented since the early 2000s.

Clenched Claw Syndrome is defined by the clinical presentation of rainbow lorikeets with recumbency, poor withdrawal reflexes, and clenched feet, in combination with non-suppurative encephalomyelitis and ganglioneuritis on microscopic examination (Chang, et al., 2020). Additional clinical signs associated with this syndrome in rainbow lorikeets include progressive paralysis of the legs and body. Affected birds are bright and alert, but may have a head tilt, unusually worn beak, or intention tremors. The bird's body condition may be good or the bird may be emaciated and dehydrated. Young lorikeets, with dark brown beaks, are most often diagnosed with this syndrome. There is no known treatment.

We have suspected a viral aetiology for this syndrome for many years but have been unable to identify any known virus in affected bird samples. However, using meta-transcriptomic approaches in combination with clinical and microscopic finings, we have been able to recover an avulavirus in the brain of affected birds along with a lorikeet chapparvovirus, and psittacine circovirus (Chang, et al., 2020). More work is needed to equivocally link one of these viruses to the clinical syndrome in lorikeets, however it appears that there is a high probability that a virus, likely the avulavirus, is the primary driver for this disease.

10.2 Lorikeet Paralysis Syndrome

Similar to clenched-claw syndrome (CCS), lorikeet-paralysis syndrome (LPS) is an emerging disease in rainbow lorikeets that we are just beginning to understand. Lorikeets with LPS present in high numbers (hundreds to thousands) to veterinary clinics in southeast QLD and northern NSW during spring, summer and Autumn. Brisbane and the Sunshine Coast are emerging as hot spots for this syndrome (D Phalen, pers. com).

Birds with LPS present with voice changes, difficulty swallowing and blinking and ascending paralysis. Mild to moderately affected birds respond well to long-term supportive care. In severe cases where birds present in complete recumbency with an inability to swallow or blink, recovery is unlikely and prognosis is considered very poor. Lorikeets with LPS do not typically have clenched-claws. While specific lesions are not found grossly in LPS, heterophilic leucocytosis and elevated muscle enzymes, uric acid, sodium, and chloride can be seen clinically.

For any lorikeets presenting with an inability to fly, hind limb weakness and ataxia during the warmer months in the region, LPS should be considered along with differential diagnoses such as trauma. If symptoms are severe, including flaccid paralysis of all limbs and the neck, inability to blink or swallow, paralysis of the tongue, and voice changes, euthanasia should be considered. When symptoms are mild, fluid and nutritional support along with nursing care can result in full recovery.

10.3 Black and White Bird Disease

Zoonotic: No
Species records: Australian Magpie, Australian Raven, Pied Currawong, Magpie-lark
Similar presentation to: *Angiostrongylus cantonensis*, intoxication, botulism, trauma

A series of unusual mortality events in “black and white birds” including Australian magpies, pied currawongs, Australian ravens and magpie larks along the east coast have defined this emerging syndrome since the first reported event in 2003. Adult and subadult birds of both sexes have been found alone or in groups, either dead or exhibiting severe weakness and paresis. Birds often appear recumbent or paralysed, but are able to stand for brief periods of time when stimulated to do so, or scoot along the ground using their wings. Birds retain good cloacal tone though some may present with diarrhoea. Birds often remain bright and alert, maintain peripheral light reflexes and withdrawal reflexes. Severely affected birds may be dyspnoeic (have trouble breathing). Ravens present with severe neurological dysfunction evidenced by head tilt, star-gazing, ataxia and circling. Presumptive intoxication has been a common concern due to the presentation of point source, mixed species mass mortality and morbidity events.

Figure 31 Australian magpie with Black and White Bird Disease exhibiting a) paralysis of wings and limbs, b) unable to right itself when placed on back though alert, and c) profound weakness and paresis

On gross examination body condition and other findings can be highly variable, presumably based on the duration of illness. Some animals may present thin and dehydrated. Affected animals may have epicardial haemorrhage, hydropericardium, or haemorrhage into the gastrointestinal tract, but often no lesions are visible to the naked eye. The syndrome is characterised by non-suppurative inflammation in the heart muscle, skeletal muscle, along the coelomic surfaces of the intestinal tract, and sometimes in the brain. Microscopically, birds may be suffering a mixture of concurrent disease such as microfilaraemia, *Leucocytozoon* spp. infection, and a host of other parasitic infections (Chang, et al., 2020). In accordance with the clinical presentation, ravens generally had more severe central nervous system lesions microscopically (Chang, et al., 2020).

We have suspected a viral aetiology for this syndrome for many years but have been unable to identify any known virus in affected bird samples. However, using meta-transcriptomic approaches in conjunction with clinical and microscopic finings, we have recovered 8 RNA viruses that may be associated with the clinical syndrome, including a divergent astrovirus which seems the most likely aetiology for this syndrome (Chang, et al., 2020). More research is required to confirm a causal association between this virus and the clinical syndrome.

There is no known treatment for black and white bird disease.

11 Species mentioned in text

Australasian gannet (*Morus serrator*)
Australian bustard (*Ardeotis australis*)
Australian hobby falcon (*Falco longipennis*)
Australian king parrot (*Alisterus scapularis*)
Australian magpie (*Gymnorhina tibicen*)
Australian raven (*Corvus coronoides*)
Australian white ibis (*Threskiornis moluccus*)
Barking owl (*Ninox connivens*)
Bar-shouldered dove (*Geopelia humeralis*)
Black-faced cuckoo shrike (*Coracina novaehollandiae*)
Black-shouldered kite (*Elanus axillaris*)
Blue-faced parrot finch (*Erythrura trichroa*)
Bourke's parrot (*Neopsephotus bourkii*)
Brown goshawk (*Accipiter fasciatus*)
Brown-headed honeyeater (*Melithreptus brevirostris*)
Brown treecreeper (*Climacteris picumnus*)
Budgerigar (*Melopsittacus undulatus*)
Carnaby's cockatoo (*Calyptorhynchus latirostris*)
Channel-billed cuckoo (*Scythrops novaehollandiae*)
Chestnut teal (*Anas castanea*)
Crested pigeon (*Ocyphaps lophotes*)
Crimson rosella (*Platycercus elegans*)
Domestic duck (*Anas platyrhynchos domesticus*)
Domestic chicken (*Gallus gallus*)
Eclectus parrot (*Eclectus roratus*)
Fiordland penguin (*Eudyptes pachyrhynchus*)
Forest raven (*Corvus tasmanicus*)
Galah (*Cacatua roseicapilla*)
Gouldian finch (*Erythrura gouldiae*)
Grey fantail (*Rhipidura fulginosa*)
Helmeted guineafowl (*Numida meleagris*)
House sparrow (*Passer domesticus*)
Laughing kookaburra (*Dacelo novaeguineae*)
Little corella (*Cacatua sanguinea*)
Little eagle (*Hieraaetus morphnoides*)
Little penguin (*Eudyptula minor*)
Long-billed corella (*Cacatua tenuirostris*)
Lord Howe woodhen (*Hypotaenidia sylvestris*)
Magpie lark (*Grallina cyanoleuca*)
Metallic starling (*Aplonis metallica*)
Musk lorikeet (*Glossopsitta concinna*)
Orange-bellied parrot (*Neophema chrysogaster*)
Pacific black duck (*Anas superciliosa*)
Pacific swallow (*Hirundo tahitica*)
Painted button-quail (*Turnix varius*)
Painted firetail finch (*Emblema pictum*)
Peaceful dove (*Geopelia placida*)
Peregrine falcon (*Falco peregrinus*)
Pied butcherbird (*Cracticus nigrogularis*)
Pied currawong (*Strepera graculina*)

Powerful owl (*Ninox strenua*)
Purple-crowned lorikeet (*Glossopsitta porphyrocephala*)
Rainbow bee-eater (*Merops ornatus*)
Rainbow lorikeet (*Trichoglossus haematodus*)
Red wattlebird (*Anthochaera carunculata*)
Red-bellied parrot (*Poicephalus rufiventris*)
Red-collared lorikeet (*Trichoglossus haematodus rubritorquis*)
Red-tailed black cockatoo (*Calyptorhynchus magnificus*)
Red-whiskered bulul (*Pycnonotus jocosus*)
Regent bowerbird (*Sericulus chrysocephalus*)
Regent honeyeater (*Anthochaera phrygia*)
Rock (feral) pigeon (*Columba livia*)
Rock parrot (*Neophema petrophila*)
Rufous whistler (*Pachycephala rufiventris*)
Satin bowerbird (*Ptilonorhynchus violaceus*)
Scaly-breasted lorikeet (*Trichoglossus chlorolepidotus*)
Short-tailed shearwater *(Puffinus tenuirostris*)
Silver gull (*Larus novaehollandiae*)
Silvereye (*Zosterops lateralis*)
Southern boobook (*Ninox novaeseelandiae*)
Spotless crake (*Porzana tabuensis*)
Spotted harrier (*Circus assimilis*)
Spotted dove (*Spilopelia chinensis*)
Square-tailed kite (*Lophoictinia isura*)
Straw-necked ibis (*Threskiornis spinicollis*)
Sulphur crested cockatoo (*Cacatua galerita*)
Superb fairy-wren (*Malurus cyaneus*)
Superb lyrebird (*Menura novaehollandiae*)
Tawny frogmouth (*Podargus strigoides*)
Variegated fairy-wren (Malurus lamberti)
Wandering albatross (*Diomedea exulans*)
Wedge-tailed eagle (*Aquila audax*)
White-bellied sea eagle (*Haliaeetus leucogaster*)
Yellow-tailed black cockatoo (*Calyptorhynchus funereus*)
Zebra finch (*Taeniopygia guttata*)

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