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

MAMMALS

Australian Government

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2 Introduction

Whether they live in the centre of our towns and cities or on their fringes, you'll find our native mammals cohabitating all of the environments that we, as human beings, enjoy. Many of our native wildlife are cryptic, nocturnal, and globally unique. Though we may not see them, they are often brought in for medical care and rehabilitation to specialist centres across the country. In this document, while not exhaustive, we endeavour to highlight some of the more common diseases you may encounter while working with Australian mammals.

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

3 Parasitic Disease

3.1 Ectoparasites

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

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

Nycteribids and Strebilids are flat flies that commonly infest Australian bats. Nycteribids are wingless flies that look similar to a spider. These flies are haematophagous, but do not appear to harm the host or bite humans. Strebilid flies have wings and closely resemble hippoboscid flies. These flies are an incidental finding on bats, but they can bite humans.

Marsupials are often encountered with areas of skin crusting or redness, especially in the inguinal or axillary regions (Skerratt, et al., 2007), or orange crusts around the pouch, that may be due to ectoparasites. In these instances, mites or lice may be present on the skin or burrowed into the epidermis making them difficult to visualise. They may incite secondary infection. A firm skin scraping visualised under a microscope may reveal the type of parasite and dictate the best course of treatment. Treatment for animals with deeply burrowing parasites is difficult due to their position deep within the keratin crust.

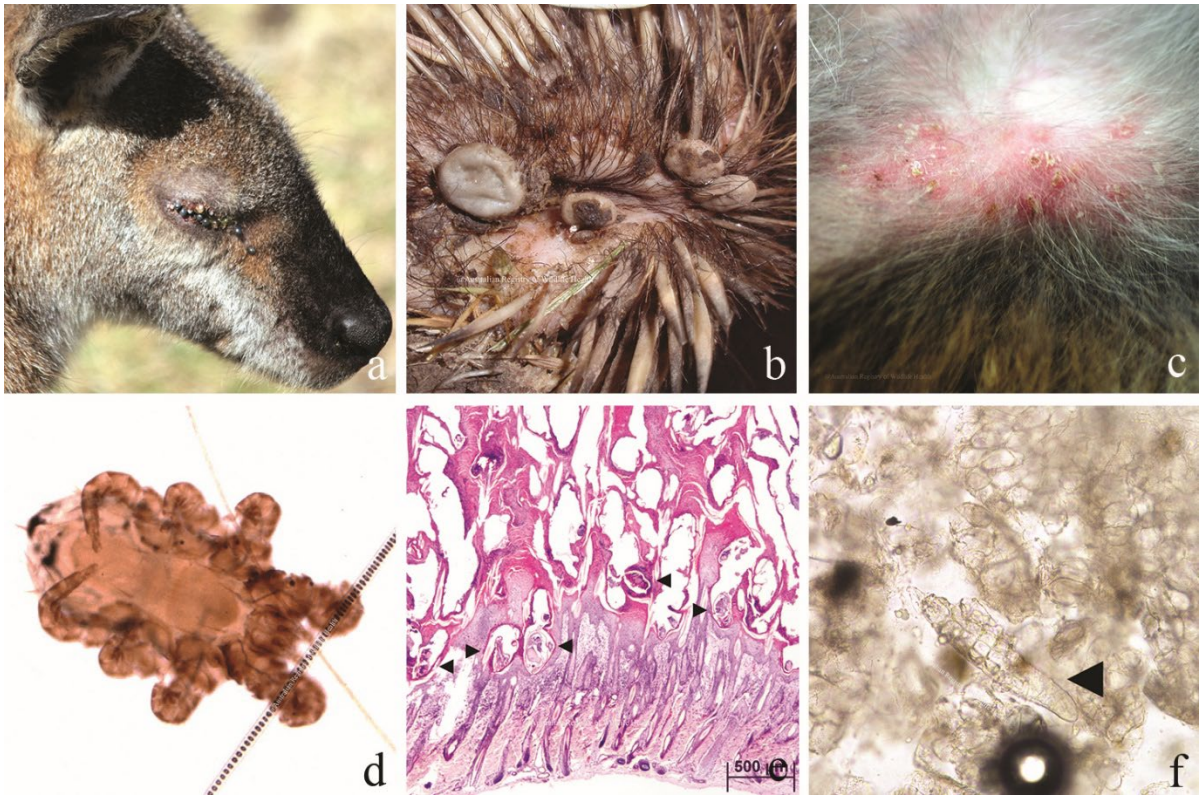

Figure 1 a) Red-necked wallaby, severe tick burden, eyes, b) short-beaked echidna, thickened skin, ticks, c) tammar wallaby, mite associated skin redness and crusting, d) common ringtail possum, mite, e) agile wallaby, sarcoptes mites in thickened keratin (arrows), f) demodex mite in skin scrape (arrow)

3.1.1 Sarcoptic Mange

Mange, caused by the *Sarcoptes scabiei* mite, has been recorded in numerous Australian species including: koala, agile wallaby, swamp wallaby, southern brown bandicoot, dingo, and both bare-nosed and southern hairy-nosed wombats (Fraser, et al., 2016). Bare-nosed wombats are the most readily reported however, and infestation with this parasite has resulted in significant population declines (Fraser, et al., 2016).

Wombats are known to suffer severe debilitation as a result of *S. scabiei* infestation. *S. scabiei* is transmitted during direct contact between animals and through sharing contaminated rubbing sites and burrows. It is an obligate skin parasite, but mites are capable of surviving for up to three weeks at low temperatures and high humidity off the host (Fraser, et al., 2016). There are reports of transmission of sarcoptic mange from wombats to humans and dogs (Booth, 1994; Skerratt & Beveridge, 1999; Fraser, et al., 2016) therefore persons handling infested animals or bedding, etc., should take precautions.

Sarcoptic mites burrow into the keratinising layers of the epidermis causing itching, thickened skin, and hair loss. Evidence of infestation may begin as thickened skin on the head, and progress to thickened skin and hyperkeratotic crust formation along the shoulders, flanks and limbs. The thick crust is composed of keratin, bacterial colonies, cellular debris, mites and degenerating neutrophils (Skerratt, et al., 1998). Deep cracks may extend through the hyperkeratotic crust into the dermis, resulting in myiasis (maggots) or invasion of opportunistic bacteria. The severe and extensive nature of skin lesions in some wombats can lead to impaired vision, impaired chewing, emaciation and abnormal activity throughout the day (Booth, 1994). Death may result from secondary infection, dehydration or starvation.

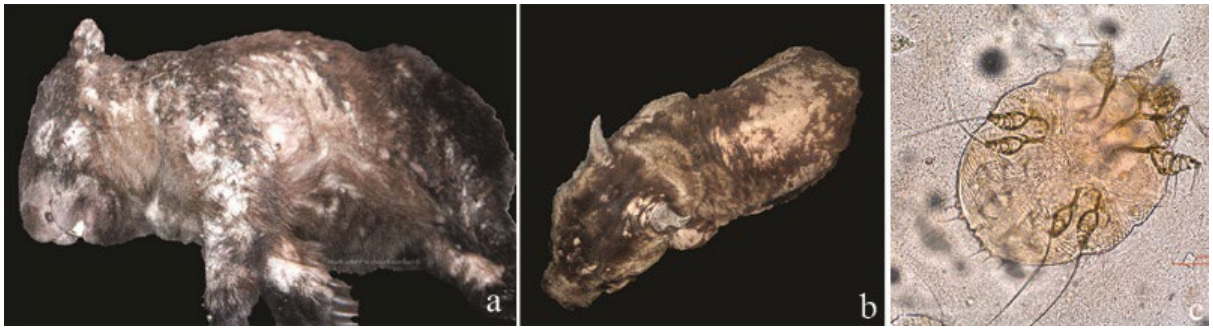

Figure 2 Patchy hair loss, thickening and crusting of skin, and emaciation in a) bare-nosed and b) southern hairy-nosed wombats with sarcoptes mange, and c) Sarcoptes scabiei

When greater than 30% of the skin surface is covered by a hyperkeratotic crust, wombats generally have marked reduction of muscle mass, and atrophy of fat. Inflammation is commonly encountered in the peripheral lymph nodes that drain severely infested portions of the skin (Skerratt, et al., 1998; Skerratt, 2003; Skerratt, 2003). Sarcoptic mange in wombats may be associated with overcrowding, habitat degradation, and a high density of foxes or feral dogs (Booth, 1998).

Diagnosis of sarcoptic mange relies on identifying the mite within skin scrapings or samples of the hyperkeratotic crust. Treatment should be initiated early in the progression of the disease whenever possible. Prior to initiating therapy, the availability of suitable habitat to release the animal should be considered. Severely infested wombats commonly become re-infected with sarcoptic mites and may transmit the mite to others in the wild population.

Euthanasia should be considered for seriously infested, emaciated and debilitated wombats. Treatment of mildly to moderately affected wombats consists of application of either a topical acaricide solution, or systemic therapy with anti-parasitic agents. Prior to the first application of topical solutions, the animal may require a bath and keratolytic agents to remove some of the hyperkeratotic crust (Booth, 1994). Rehydration and electrolyte therapy may also be required. New generation antiparasitic agents are being trialled and have been demonstrated to resolve clinical disease in captive wombats (Old, et al., 2017; Wilkinson, et al., 2021). Treatment of free ranging wombats using flaps holding doses of antiparasitic agents is becoming more common (Old, et al., 2017; Wilkinson, et al., 2021).

3.1.2 *Riouxgolvania beveridgei* (nematode)

Zoonotic: No
Reportable: No
Species records: Southern bentwing bat, eastern bentwing bat
Similar presentation to: poxvirus, white-nose syndrome, scars, mites, bacteria, yeast

Riouxgolvania beveridgei is a nematode parasite that has been recorded as causing raised, white, nodular, often vesicular or ulcerated, cutaneous lesions on unfurred skin of southern bentwing bats (McLelland, et al., 2013). Nematodes were identified by histopathology on wing biopsies, and confirmed by dissection of the nematode followed by morphological examination. In the outbreak described by McLelland et al. (2013), 45% of southern bentwing bats in a single colony in South Australia were infected, with a higher prevalence of infection in males. Nycteribiid flies were also present on all bats.

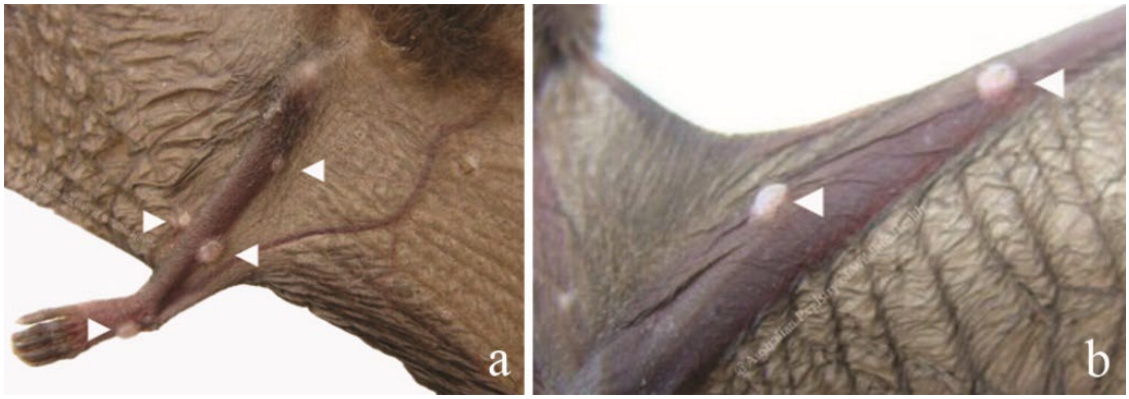

Figure 3 Raised white cutaneous nodules on unfurred skin of southern bentwing bat due to nematode Riouxglovania beveridgei (Image credit: D McLelland)

Other, similarly raised white nodular lesions have been recorded on eastern freetail-bats and grey-headed flying foxes however these are believed to be caused by intradermal mites of unknown identification. More research is needed in this area.

3.2 Endoparasites

The parasites of native species are extremely diverse. Many of these parasites are cryptic and we are only beginning to understand their diversity as molecular methods for identification become more common (Spratt & Beveridge, 2018). With over 600 species described from marsupials and monotremes (Spratt & Beveridge, 2018) and a lack of diagnostic parasitologists, specific identification of parasites found on post mortem and faecal examinations are often not pursued. The parasites of prey species can be found passing through the gastrointestinal tract of carnivorous mammals, which can cause confusion. Understanding the type and volume of parasites that should be reasonably expected in free-ranging wildlife can help us to understand when these host-parasite relationships become imbalanced and when disease may be attributed to parasite burden. The examination of animals that die suddenly from known traumatic injury can help to establish these baseline parasite diversity and intensity parameters.

3.2.1 Trematodes (flatworms)

Sheep liver fluke infection, *Fasciola hepatica*, occurs commonly in eastern grey kangaroos, red-necked wallaby, swamp wallaby, bare-nosed wombats and brushtail possums grazing on wet pastures (Spratt & Presidente, 1981). Although one does not often see adult flukes, nor large numbers of them, the immunological response is such that the entire liver may become completely fibrosed. The number of flukes is not indicative of the amount of tissue damage observed. *Lymnaea tomentose* snails function as the intermediate host for this parasite. Animals infected with *F. hepatica* may exhibit weight loss, anorexia, depression, anaemia, and death.

3.2.2 Nematodes (roundworms)

A wide variety of nematodes parasitise can be found in the gastrointestinal and respiratory tracts, and subcutaneous tissues of native mammal species. Clinical signs are rarely associated with nematode parasitism however heavy burdens and nematode migration through tissues can result in significant scarring and fibrosis, inflammation or mechanical blockages.

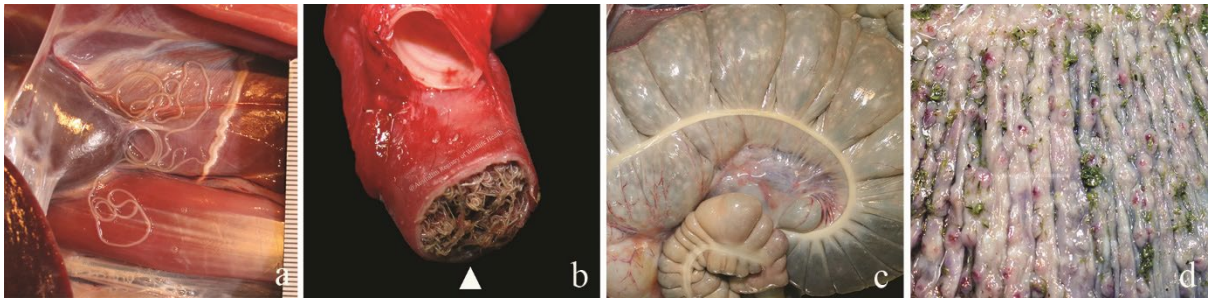

Figure 4 a) Swamp wallaby, abdominal cavity, unidentified nematodes, b) swamp wallaby, oesophagus, Cyclostrongylus wallabiae, c) eastern grey kangaroo stomach, multifocal white foci on serosal surface, and d) raised white nodular granulomas with central ulceration, unidentified nematodes

Ophidascaris robertsi, a python parasite, can often be found migrating through, or encysted within, various tissues in small mammals. While most infections are incidental, sometimes *O. robertsi* burdens are severe, causing vascular disturbance and organ distortion (Gonzalez-Astudillo, et al., 2019). The nematodes are very large, up to 15 cm, and can be easily visualised in viscera and subcutaneous tissues. As pythons are the primary host for this parasite, infection is generally restricted to small mammals, however reports have been made in larger species such as the koala (Gonzalez-Astudillo, et al., 2019) and red kangaroo. Infection is not transmissible between individuals. Infection is most likely spread through contact with python faeces or contaminated soil.

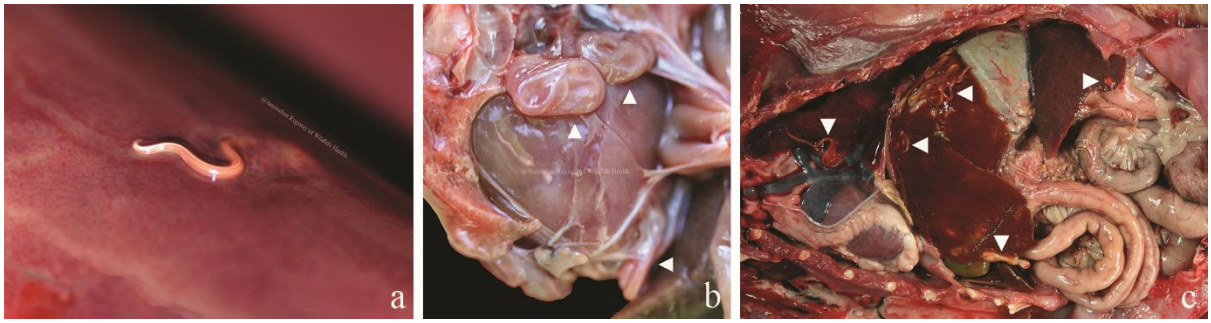

Figure 5 Ophidascaris robertsi a) red kangaroo, emerging from liver, b) southern brown bandicoot, encysted in viscera around heart, c) tiger quoll, emerging from and encysted in lung and liver lobes

Fine, long, filarial nematodes are often found in the abdominal, thoracic cavity, intravascular or lymphatic vessels of native rodents, marsupials and bats. These fine, white parasites can be easily overlooked or misinterpreted as lymphatic or blood vessels. Filarial nematodes of several genera (*Breilia, Dirofilaria, Johnstonema, Dipetalonema*) have been identified as either adult parasites, or as intravascular larval microfilaria. These parasites are generally considered to be an incidental finding, although exclusion of the dog heartworm, *Dirofilaria immitis*, infection is worth considering.

3.2.2.1 Angiostrongylus cantonensis (Rat lungworm)

Zoonotic: from slugs and snails
Species records: ringtail possum, brushtail possum, rufous bettong, black flying fox, little red flying fox, grey-headed flying fox
Similar presentation to: trauma, toxoplasmosis, *Angiostrongylus mackerrasae*

Angiostrongylus cantonensis is a metastrongylid nematode from the Pacific Islands that has fairly recently spread along the East coast of Australia. *A. cantonensis* can cause central nervous system disorder and death in wild and captive native fauna. *A. cantonensis* is a zoonotic threat. Eosinophilic encephalomyelitis has been reported to occur within people, usually children, that ingest snails or slugs. This parasite is morphologically similar to the native *Angiostrongylus mackerrasae*, which is not considered to be a pathogen.

Parasitic meningoencephalitis due to infection by *A. cantonensis* has been reported in ringtail possums, brushtail possums, rufous bettongs, black flying foxes, and little red flying-foxes, numerous bird species, and people in Queensland and New South Wales. A single outbreak of *A. cantonensis* encephalitis in captive, hand-raised, sub-adult grey-headed flying-foxes (GHFF) was reported in Sydney in 1997 (Reddacliff, et al., 1999). Ringtail possums on the same property had also previously been diagnosed with *A. cantonensis* encephalitis. Four of five captive GHFF became paretic, depressed and anorexic over a period of several weeks. Three of the GHFF died, and the fourth animal responded to oral fenbendazole therapy (Reddacliff, et al., 1999). Brushtail possums are often found to be infected with *A. cantonensis*, and may be a sentinel of high environmental parasite concentrations and zoonotic risk (Ma, et al., 2013).

Rats are the definitive host of *A. cantonensis*. Adult parasites live within the pulmonary arteries of rats and release eggs that are trapped in the pulmonary microvasculature. Larvae emerge from the eggs and migrate into the airways and then to the intestinal tract. Rats excrete first-stage larvae in their faeces, and these larvae either directly penetrate, or are ingested by terrestrial snails or slugs. Larvae develop into the infective third-stage in the snails, which are then eaten by rats. When ingested by rats, infective larvae migrate to the pulmonary artery where they mature to adulthood. In other hosts, the parasite often undergoes migration through the spinal cord, ascending to the brain causing considerable neurological deficits (Ma, et al., 2013).

Animals suffering from *A. cantonensis* meningoencephalitis may exhibit a variety of clinical signs related to the central nervous system. The most common clinical signs of infection include hind limb ataxia, paresis, intention tremors, which progress to forelimb paresis, profound central depression, coma, seizures, and often death. Gross post mortem examination is often unrewarding in animals with eosinophilic meningoencephalitis. Rarely, eosinophils are evident within CSF antemortem, or nematode parasites are visible within the subarachnoid spaces post mortem.

Nematode larvae are evident upon histologic examination of serial sections of the brain and spinal cord, but they can be difficult to spot. Examination of numerous sections of the spinal cord and brain is recommended. Often *A. cantensis* infection post mortem presents as foci with mixed inflammatory cells with few or no parasites in the spinal column, while the advancing front of parasites in the brain are unaccompanied by an inflammatory response. Inflammation most often occurs as the parasites shed their cuticle and release antigen, triggering a mixture of lymphoplasmacytic, granulomatous and eosinophilic inflammation. Foci of malacia may occur when parasite migration has damaged a blood vessel resulted in a lack of oxygenation and tissue necrosis.

Diagnosis of the infection can be very difficult, since animals do not usually develop eosinophilia in peripheral 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 *A. cantonsis* PCR to aid diagnosis via CSF ante mortem or central nervous tissue post mortem. Treatment of the infection 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 be reversed.

3.2.2.2 *Marsupostrongylus* sp.

Marsupostrongylus sp. has been reported in northern quoll, agile antechinus, Atherton antechinus, swamp antechinus, dusky antechinus, mountain brush-tailed possum, common brush-tailed possum, Herbert River ringtail possums, common ringtail possum, long-nosed bandicoot and greater gliders. Adult or larval *Marsupostrongylus* sp. are generally found within the lungs or trachea, and larvae may be found in faeces. Animals may be asymptomatic, however severe infections can result in respiratory disease and death. Lungs may appear consolidated or have pale areas on gross examination. Nematodes may be visualised by squashing a very small piece of firm lung tissue between two glass slides and observing these under the microscope. Infection is confirmed by histopathology.

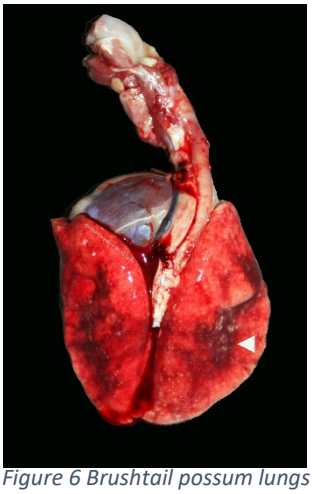

Figure 6 Brushtail possum lungs with bilateral areas of congestion and pinpoint white foci (arrow), Marsupostrongylus spp.

3.2.2.3 Toxocara pteropodis

Toxocara pteropodis is an ascarid of southeast Asian and Australian flying-foxes. The adult form of this nematode inhabits the upper gastrointestinal tract of nursing pups. The adult female flying-fox ingests eggs of *T. pteropodis* that are passed in the pup's faeces. Larvae develop in the dam's intestinal tract, penetrate into the portal circulation, and encyst in the liver. The parasite then lies dormant, but can be re-activated near the end of parturition. Re-activated larvae concentrate in the mammary tissue and are passed to the pup in milk. Adult nematodes die and are shed when juvenile flying-foxes cease nursing. Pups generally contain less than five adult *T. pteropodis* nematodes and infection is inconsequential. Unusually high burdens of this parasite, however, can result in obstruction of the gall bladder, or obstruction of airways in young flying-foxes (Prociv, 1986). This parasite is also potentially zoonotic and health and safety measures should be in place to prevent disease transmission to humans.

3.2.3 Cestodes (tapeworms)

Two of the most common parasites of the echidna are the cestodes *Echidnotaenia tachyglossi*, which occurs in northern echidna, and *Linstowia echidnae,* which occurs in southern echidna where 500 or more parasites can occur in a single host (David Spratt, personal communication). Most often these parasites are incidental to the host, but large numbers can cause space occupying lesions.

Bertiella trichosuri is an anoplocephalid cestode that parasitises the intestinal tract of brushtail possums. Many possums are infected with this tapeworm, and the parasites are dramatically large, yet clinical signs have not been attributed to infection. Possums living in suboptimal habitats appear to be most susceptible to infection with this parasite (Booth, 1994). Similar, but smaller parasite of the *Bertiella* genus inhabit the intestinal lumen of koalas, ringtail possums and other native mammals with no apparent ill-effect.

3.2.3.1 Sparganosis

Zoonotic: Yes
Species records: Short-beaked echidna, eastern quoll, spotted-tailed quoll, *Antechinus* spp., brush-tailed phascogale, common planigale, Tasmanian devil
Similar presentation to: neoplasia, bacterial infection, fungal infection

Sparganosis is the term used to describe infection with plerocercoid cestode *Spirometra eranacei* or *Diphyllobothrium dendriticumi*. The definitive hosts are canids and felids and they are transmitted by

primary and secondary intermediate hosts such as crustaceans and copepods, and amphibians and reptiles respectively.

S. eranacei is most commonly reported to cause large tumour-like masses within the subcutaneous tissues of the ventral and lateral abdomen of short-beaked echidna. The masses can be as large as 12 cm diameter and are composed of a central sparganum, surrounded by an intense non-suppurative inflammatory infiltrate, and a thick layer of fibrous tissue. Plerocercoids may replace large proportions of the pulmonary parenchyma (Whittington, et al., 1992). Presumably, the echidna ingests infected copepods and act as incidental intermediate hosts. Sparganosis is a rare, but highly unpleasant zoonotic disease (Tran, et al., 2019).

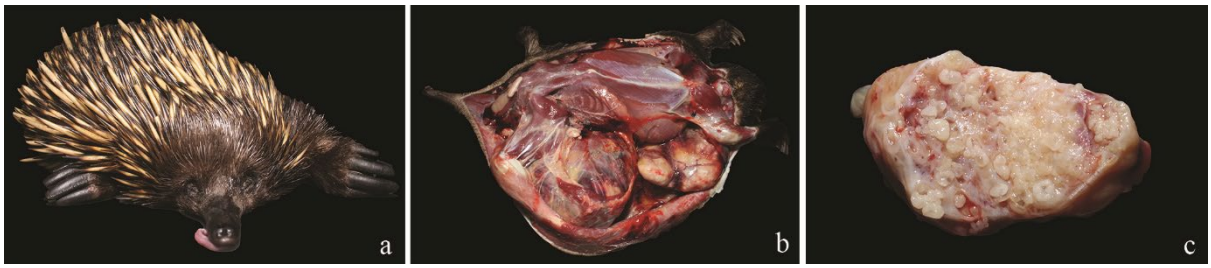

Figure 7 Short-beaked echidna with multiple large subcutaneous masses (a, b) of Spirometra eranacei encased in fibrous tissue (c) causing deformed appearance and locomotor dysfunction

3.2.4 Protozoa (single-celled parasites)

3.2.4.1 Coccidiosis

Zoonotic: No
Species records: All
Similar presentation to: bacterial infection, candidiasis, other parasites, toxins, torsion

Eimerian and isosporan coccidial oocysts are commonly identified within the faeces of healthy captive and wild mammals. There are more than 40 described *Eimeria* spp. in macropods alone. Coccidia appear to be host specific, evolving with their host over a long time. Disease associated with coccidial infection in free ranging mammals is rare. Coccidia may cause necrotising enteritis in young animals of a variety of species and clinical disease is often the result of co-morbidities including chronic stress. Microscopic examination of faecal wet preparations and flotations is recommended to monitor coccidia burdens in wildlife maintained in rehabilitation facilities. When large numbers of faecal oocysts are detected or if oocysts accompany diarrhoea, treatment is advisable.

Coccidial infections can be either enteric, affecting predominantly the gastrointestinal system, or disseminated, affecting multiple organs in addition to the intestinal tract.

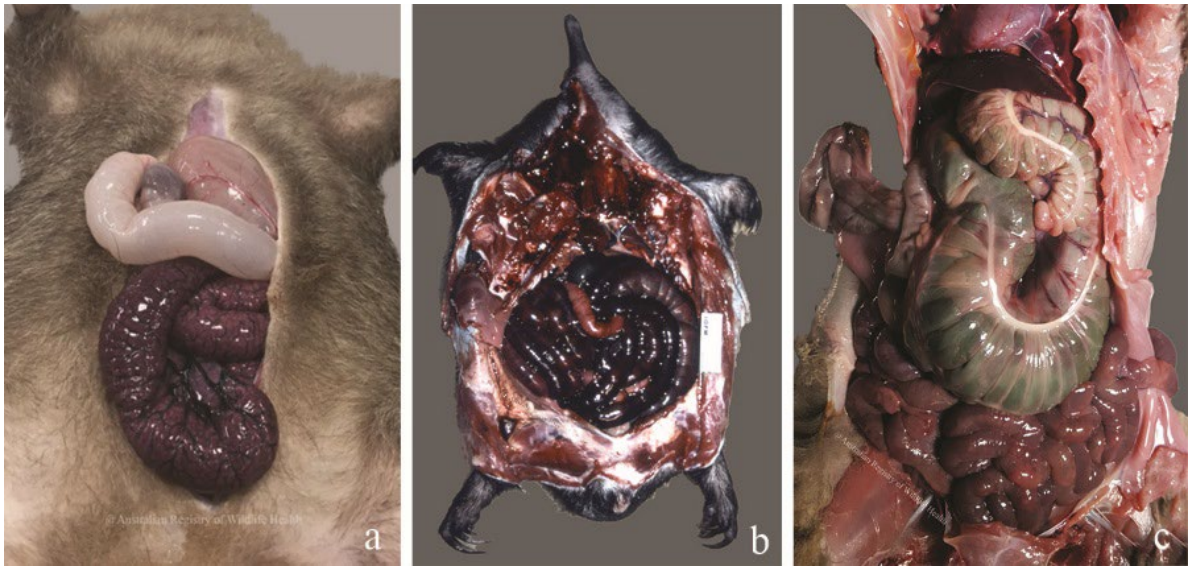

Figure 8 Necrotising enteritis caused by severe coccidial infection in a) bare-nosed wombat, b) short-beaked echidna, and c) eastern grey kangaroo

Animals with enteric coccidiosis may have haemorrhagic and necrotising enteritis. The intestinal mucosa is often thickened due to the presence of large numbers of coccidia. Villous atrophy has been reported in some animals with enteric coccidiosis, as the mucosa can slough as parasites are released. An intense mononuclear cell infiltration is usually evident throughout the intestinal lamina propria of animals with coccidial enteritis. Echidna are known to suffer from the disseminated form of coccidiosis where asexual and sexual stages of the coccidian lifecycle are scattered throughout the endothelium and parenchyma of the lung, liver, heart, kidney, spleen, lymph nodules and gastrointestinal tract (Dubey & Hartley, 1993) associated with *Eimeria echidnae* – as described below (Slapeta, et al., 2017).

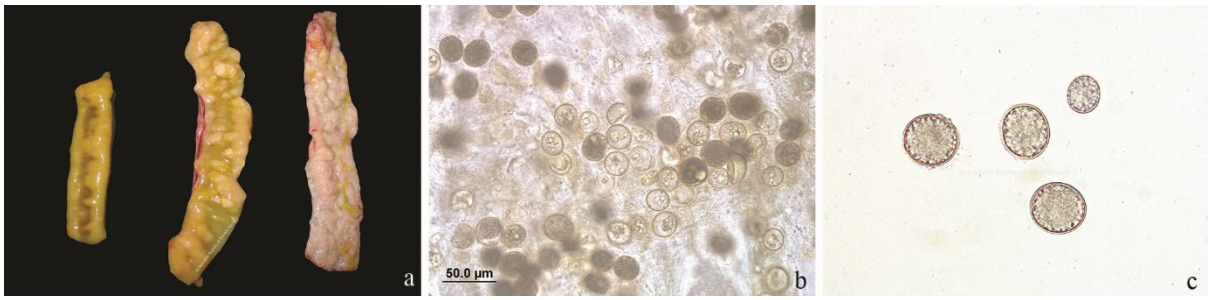

Figure 9 Enteric coccidiosis in a short-beaked echidna a) sections of small intestine (jejunum) showing increased thickening of the mucosa, b) mucosal scrape showing masses of coccidial ova, c) coccidian parasites in faecal float

Gross post mortem examination of animals with coccidiosis often reveals a gastrointestinal tract diffusely distended with fluid and gas. The character of the luminal fluid varies from yellow, mucoid to green/grey and thick. Fibrin strands are often scattered throughout the luminal fluid. The wall of the proximal small intestine is often segmentally thickened and bears a reticular pattern created by small yellow foci raised above the more normal mucosa. Intestinal villi may visibly hypertrophic (Hum, et al., 1991).

Histological examination of sections of the affected intestine typically reveals congestion of the mucosa, and large masses of coccidial gametocytes within distended host cells in the mucosa and lamina propria. The abundance of these coccidial gametocytes results in the hypertrophic villi seen upon gross examination (Hum, et al., 1991). The presence of the coccidial organisms has been associated with oedema and an inflammatory cell infiltrate throughout the lamina propria.

Diagnosis of coccidiosis is achieved through examination of faeces in a wet preparation using light microscopy at a magnification of 400x. Standard faecal flotation techniques are also useful in the diagnosis of coccidiosis. Histology, serology, immunohistochemistry and PCR from serum and tissues is also diagnostic.

3.2.4.1.1 Wombat coccidiosis

Eimeria arundeli is a frequently encountered enteric coccidian of the bare-nosed wombat. This parasite has been associated with enteritis in sub-adult wild and hand-reared wombats, but is often considered an incidental parasite of adult animals. Wombats with a severe burden of coccidia may develop mucoid to liquid green diarrhoea, progressively lose weight and become bloated. The presentation of enteritis is often associated with the onset of grazing, which occurs at approximately 10 months in wild wombats, but is often earlier in hand raised wombats. Presumably animals immunologically naïve to coccidia are suddenly exposed to large numbers of infective oocysts present on contaminated pasture. Once clinical signs of enteritis have developed, treatment becomes very difficult. Fluid therapy can be difficult to deliver to wombats, and response to anti-coccidial therapies that are used in other species is often poor.

3.2.4.1.2 Echidna Coccidiosis

Eimeria echidna, *E. tachyglossi*, and *Octosporella hystrix* are enteric coccidia of the short-beaked echidna (Barker, et al., 1985; Slapeta, et al., 2017). Coccidial oocysts are a frequent finding within the faeces of healthy echidna; however, coccidia are occasionally associated with marked enteritis or disseminated disease. Short-beaked echidna with coccidial enteritis or disseminated coccidiosis are most often found dead without premonitory clinical signs.

Monotremes have a diffuse lymphoid system composed of nodules that are suspended by a pedicle dispersed throughout the lymphatic vessels. The lymphoid nodules within the intestinal mesentery are grossly visible as small white, raised foci near the attachment of the mesentery to the intestinal serosa. Echidna with disseminated coccidiosis often have foci of necrosis throughout the spleen and the disseminated lymph nodules. Presumably, these foci of necrosis represent sites of schizogony, where cell damage is associated with the release of merozoites from ruptured schizonts.

Short-beaked echidna with multisystemic coccidiosis may have *Atoxoplasma*-like blood parasites circulating within monocytes. Examination of blood films from short-beaked echidna can be used to diagnose clinical systemic coccidiosis. Many echidnas have very small number of circulating organisms, but large numbers of organisms evident in monocytes is associated with clinical disease (Registry). Collection of a blood sample and examination of the blood film is encouraged to identify systemic coccidiosis. Buffy coat preparations concentrate the leucocytes and can aid in the identification of intracellular parasites. The systemic form of coccidiosis has been associated with the presence of *Eimeria echidnae* (Slapeta, et al., 2017).

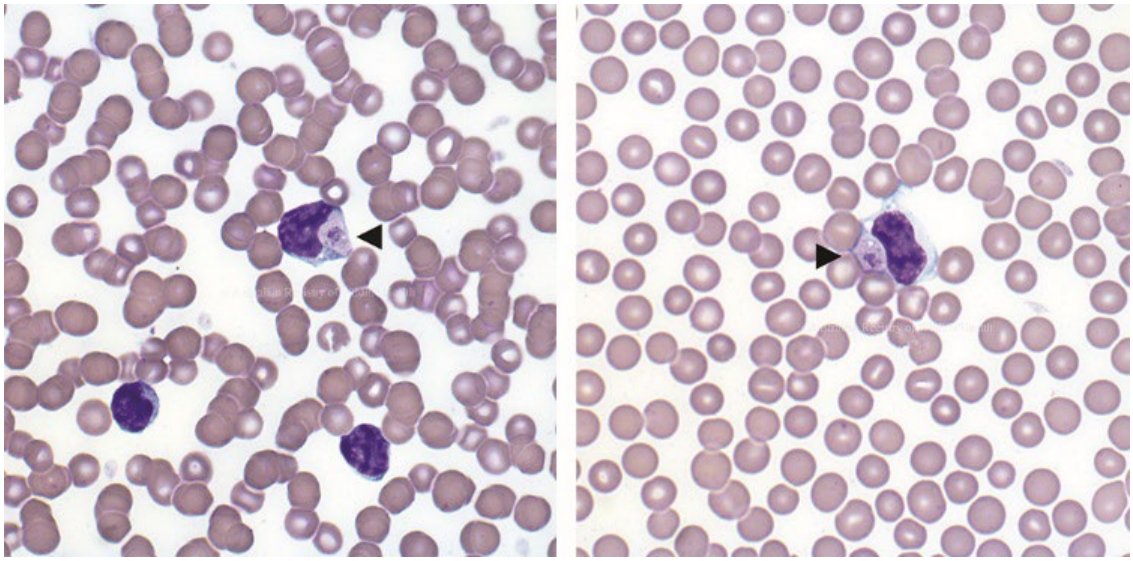

Figure 10 Atoxoplasma-like blood parasites circulating within monocytes (arrows), blood smear, indicative of clinical systemic coccidiosis

3.2.4.2 Toxoplasmosis

Zoonotic: Yes
Species records: All
Similar presentation to: other protozoa, trauma, cryptosporidiosis, Australian bat lyssavirus, *Angiostrongylus cantonensis*, paralysis tick, systemic bacterial infection, trauma

Toxoplasma gondii is a single-celled protozoan parasite with worldwide distribution and a broad host range. All vertebrates may be infected with the asexual stages of this organism; however, some taxonomic groups are more susceptible to clinical illness. Marsupials are remarkably susceptible to developing toxoplasmosis.

Asexual (merogony) and sexual (gametogony) stages of the *T. gondii* lifecycle take place within the intestinal mucosa of cats, which are the definitive hosts. Unsporulated oocysts are shed in the faeces and become infective when they sporulate, 24 to 96 hours after leaving the host. Cats may shed millions of oocysts in their faeces during their first infection with *T. gondii*. Cats are usually infected when they eat birds or rodents infected with *T. gondii* asexual tissue cysts.

Marsupials are primarily infected with *T. gondii* when they ingest food or water contaminated with sporulated oocysts from cat faeces, or ingest encysted parasites in prey. Transplacental transmission, and transmission via ingestion of infected earthworms, flies and cockroaches has been reported. Cysts are identified most commonly in the tissues of the brain, liver, muscles and retina. Cysts may remain dormant for prolonged periods, but are capable of releasing their bradyzoites, which become tachyzoites and re-initiate active infection. The organism has a high affinity for the central nervous system, lung, pancreas, lymphoid system, myocardium, adrenal gland and ocular tissues. Necrosis and inflammation accompany the replication of tachyzoites in these tissues. Oocysts are highly resilient in marine environments and can biomagnify in filtering species, resulting in health threats to marine mammals.

Clinical illness associate with *T. gondii* occurs primarily in animals that are immunosuppressed, hand raised or exposed to a high infective dose or organisms. Clinical signs of toxoplasmosis are primarily associated with lesions in the central nervous system, lungs, and liver. Animals clinically affected by toxoplasmosis may be depressed, weak, anorexic, have a fever, difficulty breathing, lack of muscle control or coordination, comatose, convulsive, or may exhibit muscle stiffness, diarrhoea, vomiting,

eye inflammation or cataract formation. Slow growth rates have been reported in some hand raised animals in association with *Toxoplasma gondii* infection. Infection or expression of disease may occur at any time of the year.

Gross post mortem lesions may be inapparent, however, pulmonary congestion, oedema, consolidation, gastric ulceration, hepatosplenomegaly, lymphadenopathy, myocardial haemorrhage, or multisystemic pale foci may be evident. Infection can be localised to the spinal cord or adrenal gland, so microscopic examination of a broad range of tissues is recommended. Individual tissue cysts are commonly found within the muscle and nervous tissue of animals during histologic examination. Fulminating infection can be associated with the presence of pseudobradyzoites, which appear similar to dormant tissue cysts, except that they are larger, lack a cyst wall and can be found with free zoites and foci of inflammation. In possums, muscle tissue may undergo calcification and necrosis. Unless cysts are associated with tissue necrosis or inflammation, they most likely represent subclinical infection.

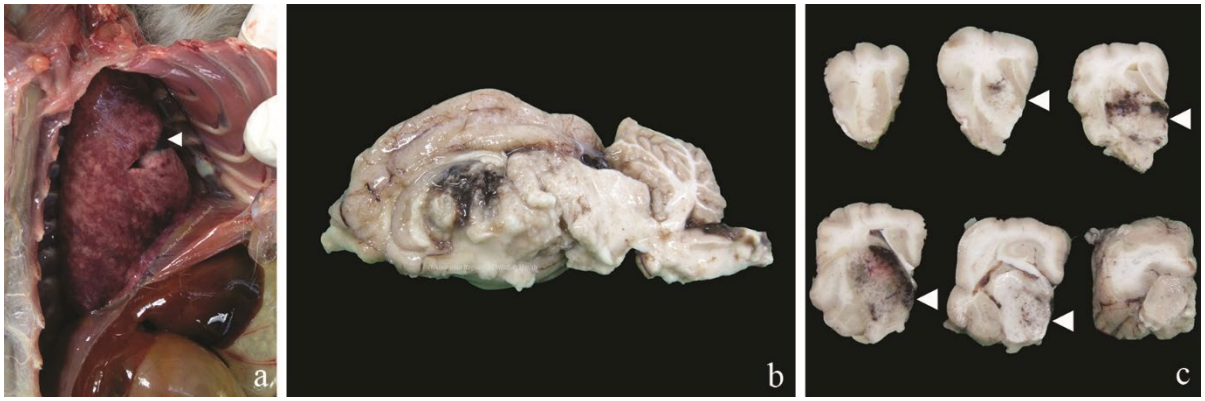

Figure 11 a) Red-necked wallaby with congested and consolidated lungs (arrow), systemic toxoplasmosis, b, c) brain and brain in section, haemorrhage and necrosis (arrows), systemic toxoplasmosis, bare-nosed wombat (Image courtesy of D. Phalen)

Ante mortem diagnosis of toxoplasmosis relies on serological testing to detect rising IgG *T. gondii* concentrations. Serial four-fold increases in *T. gondii* IgG titres represent active infection. IgM antibodies are more indicative of active infection than IgG antibodies, and a single high *T. gondii* IgM concentration in serum may reflect active infection. Post mortem diagnosis of toxoplasmosis usually occurs through histopathologic examination of tissues, or cytological examination of impression smears of the spleen, liver, or lung. Immunohistochemical staining, tissue antigen ELISA, or PCR are used to detect small numbers of *T. gondii* organisms in tissues, and differentiate *T. gondii* from other protozoa (Donahoe, et al., 2015).

Treatment of toxoplasmosis is based upon the use of drugs that arrest replication of the parasite. A drug that will eliminate the organism from tissues has not yet been discovered. Thus, a competent host immune system is required to effect recovery. Response to therapy may vary depending on the degree of tissue damage already sustained. If therapy is likely to be effective, clinical improvement should be noticed within the first seven days.

Toxoplasmosis prevention relies on decreasing the opportunity for immunologically naïve or otherwise susceptible hosts to be exposed to large numbers of infective oocysts. Oocysts can survive for up to 18 months in the environment. Effective cat control and proper storage of animal foodstuffs are the cornerstones of toxoplasmosis prevention. Clinical trials using commercial *T. gondii* vaccines, developed for use in sheep, have resulted in the development of fatal toxoplasmosis in Tammar wallabies (Lynch, et al., 1993). The management of stressors, or other immunosuppressive factors, are important in reducing the expression of clinical disease.

3.2.4.3 *Sarcocystis* spp.

Although typically an incidental finding, *Sarcocystis* spp. may be clinically significant if the parasite burden is high. Infection has been reported in a number of species, however, due to definitive diagnoses being difficult this may be either over or under reported. Gross lesions are rarely reported and diagnosis is typically made using histopathology to describe cyst morphology and immunohistochemistry to rule out Toxoplasma and Neospora spp. infections. *Sarcocystis* sp. has a predilection for muscle tissue and there is rarely an inflammatory response by the host. Immunohistochemistry, electron microscopy and PCR can be used to definitively identify *Sarcocystis* cysts in tissue, but these tests can be difficult to find in Australia.

3.2.5 Protozoan blood parasites

Zoonotic: No
Reportable: *Anaplasmia marginale, Theileria parva, Theileria annulata, Theileria equi*
Species records:
Similar presentation to:

Theileria sp. and *Babesia* sp. are common incidental haemoprotozoa of monotremes (Slapeta, et al., 2017). An *Anaplasma marginale*-like organism has also been identified within circulating red blood cells of clinically healthy echidna (Whittington, 1993).

Hepatozoon spp are occasionally found in the erythrocytes of possums and gliders, with uncertain clinical significance.

Hepatocystis levinei is an intraerythrocytic protozoon of the family *Plasmodiidae*. This protozoan infects grey-headed flying foxes and is transmitted by *Culicoides nubeculosus*, a biting midge.

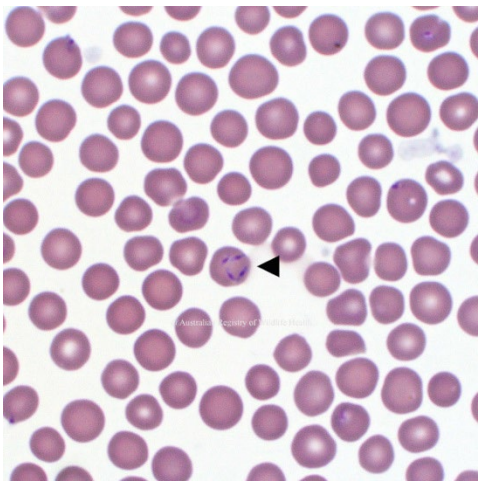

Figure 12 Theileria parasites within the red blood cell of a platypus

3.2.5.1 Leishmania spp.

Zoonotic: undetermined for *L. australiensis*
Reportable: Yes
Species records: red kangaroo, northern wallaroo, black wallaroo, agile wallaby
Similar presentation to: other trypanosomes, ticks, dermatitis, trauma, pox and papilloma virus

Cutaneous leishmaniosis has been described in multiple macropod species in the Northern Territory (Dougall, et al., 2009). Lesions appeared primarily on the pinnae and other extremities as a granulomatous dermatitis defined by areas of thickened skin with hair loss and central papules that progressed to ulceration. Molecular studies have confirmed the organism and research by Dougall, et al (2009) showed that transmission is via a day-feeding midge.

3.2.5.2 Babesiosis

Zoonotic: No
Reportable: *Babesia bovis, B. bigemina, B. divergens, B. caballi, B. equi*
Species records: eastern grey kangaroo, agile wallaby, woylie, short-beaked echidna
Similar presentation to: anthrax, trauma, toxins, candidiasis, toxoplasmosis

Babesia macropus has been described in both free-ranging and rehabilitated hand-reared eastern grey kangaroos. Babesiosis in eastern grey kangaroos is more commonly recognised in the rehabilitation setting where infection is usually limited to pouched young that may have been in care for several months before exhibiting clinical signs such as refusing food, weakness, depression, convulsion, or sudden death. However, in rare mass mortality events in free ranging eastern grey kangaroos in NSW, all age class of animals have been recorded with clinical signs including weakness, lethargy, hunched posture, drooling, bleeding from nose and cloaca, and sudden death. These mass mortality events share a similar history of artificially increased animal density due to being fed by humans, or due to habitat alteration through fire or other event.

Macropods with clinical babesiosis are often anaemic with pale mucous membranes and low PCV, are of variable body condition, and exhibit petechial haemorrhage and widespread oedema. Blood smears are rarely diagnostic since the organisms congregate in capillaries. Microscopic examination of kidney impression smears and small sections of brain squashed between two slides, stained with Diff Quik, can provide a rapid diagnosis at time of necropsy. Electron microscopy and molecular studies have been described by Donahoe, et al. (2015).

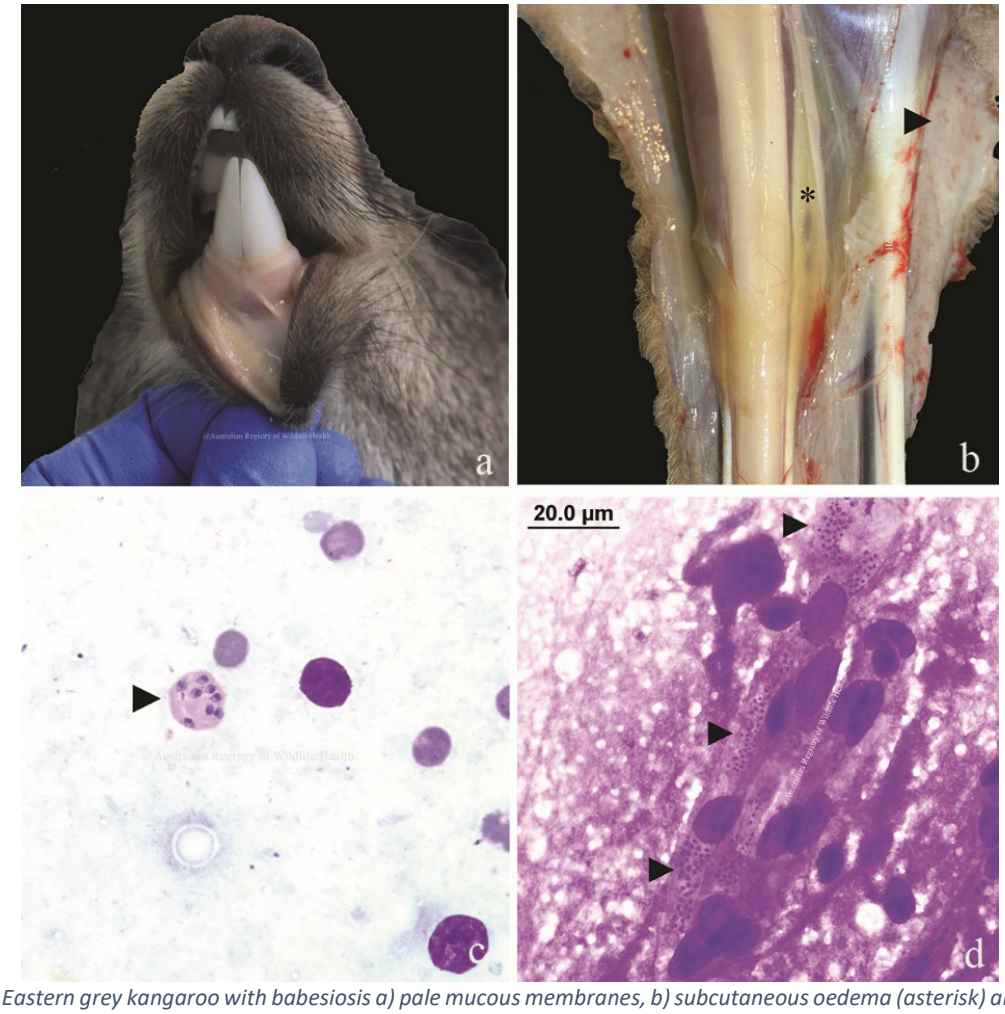

Figure 13 Eastern grey kangaroo with babesiosis a) pale mucous membranes, b) subcutaneous oedema (asterisk) and pinpoint haemorrhages (arrow), c) babesia in red cell, kidney impression smear, d) intravascular parasites, brain squash prep (arrows)

The significance of infection with *Babesia* spp. in other species is undetermined and infection is generally considered incidental.

4 Bacterial Diseases

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

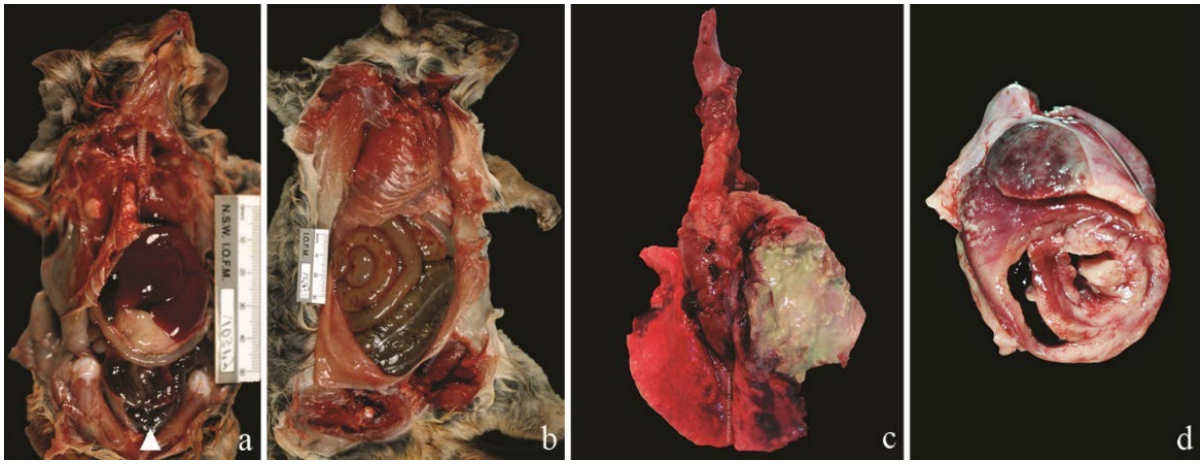

Figure 14 a) ringtail possum, haemorrhagic enteritis (arrow), Salmonella typhimurium, b) ringtail possum, intestinal haemorrhage, Streptococcus bovis, c) red-necked wallaby, lung abscess, Fusobacterium necrophorum , d) brushtail possum, pale foci, heart, Listeria monocytogenes

4.1 Salmonellosis

Zoonotic: Yes
Reportable: *Salmonella abortus-ovis, S. abortus-equi, S. pollarum, S. gallinarum*
Species records: All, especially possums and long-nosed bandicoots
Similar presentation to: may be no presenting symptoms, coccidia, toxins

Salmonellosis is primarily a disease of captive animals, and disease often occurs in hand raised animals or those animals subject to stressful events. Outbreaks of salmonellosis have been recorded in young ringtail possums being hand reared. Rapidly fatal, haemorrhagic enteritis and septicaemia are the manifestations of salmonellosis in young possums. Foci of hepatic necrosis and paratyphoid nodule formation are also reported in possums (Booth, 1994).

Many species of *Salmonella* have been isolated within healthy wild and captive native species. Some types of *Salmonella* may cause little or no harm to their natural host, such as *Salmonella enterica* paratyphi B, variant Java in long-nosed bandicoots (Staff, et al., 2012), but infection in people, especially children, can cause severe and debilitating diarrhoea and sequela. As the most direct route of transmission is faecal-oral, suitable hygiene practices should be employed by anyone working with wildlife both in the rehabilitation setting, and in the field.

4.2 Leptospirosis

Brushtail possums throughout south-eastern Australia and the North Island of New Zealand have been recorded as being infected with *Leptospira interrogans* serovars including: Balancia (Victoria and NZ), Medanensis (north Qld), Hardjo (Sydney, NSW and NZ), and Arborea (Sydney, NSW) (Bender, et al., 2019). Leptosporosis has also been recorded in bandicoots (Bender, et al., 2019), sheep, cattle, and people. This organism does not appear to be host specific. Transmission between possums occurs primarily via urine; however, sexual transmission has also been reported. Infection is most often

inapparent causing mild, transient clinical signs of malaise in possums, and can be associated with inflammation within the kidneys.

4.3 Mycobacteriosis

Zoonotic: Potential
Reportable: *M. bovis, M. caprae, M. tuberculosis*
Species records: various dasyurids, common brushtail possum, numbat, feathertail glider
Similar presentation to: other bacterial diseases

Mycobacteriosis of agricultural or human zoonotic concern has not been identified in free-living wildlife in Australia, however feral brushtail possums in New Zealand are a reservoir for *M. bovis* in areas where the cattle and possums share habitat at the forest/pasture margin.

Multisystemic mycobacteriosis is a fairly common post mortem finding in many species, especially those that spend a lot of time digging or foraging in soil. Granulomatous lesions may occur in a variety of tissues, but are most common in the lungs, liver or skin. Beaded, red bacilli are easily demonstrated within the granulomas upon acid fast staining. If tissues are collected for microbiological culture, the tissue that spans both healthy and granulomatous areas will be most fruitful.

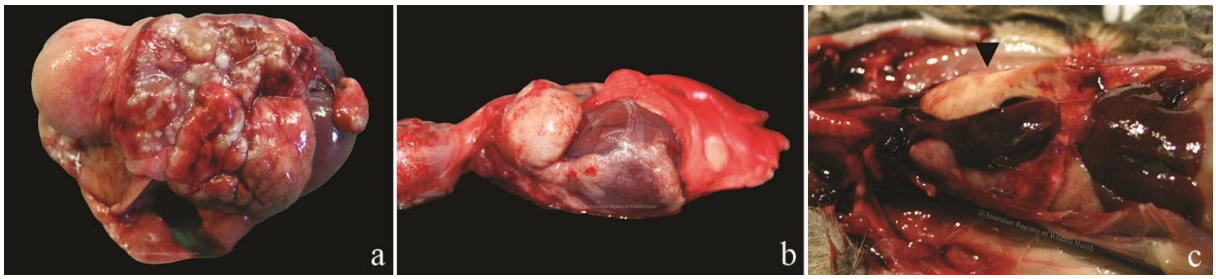

Figure 15 a) Brush-tailed bettong, lung, Mycobacterium avium, b) long-nosed potoroo, lung and thoracic lymph node, M. fortuitum, c) brush-tailed phascogale, lung, Mycobacterium spp.

4.3.1 Buruli ulcer (*Mycobacterium ulcerans*)

Zoonotic: Unknown
Reportable: in Victoria
Species records: common ringtail possum, common brushtail possum, mountain brushtail possum, koala, long-footed potoroo, northern brown bandicoot, as well as feral, pet and agricultural species
Similar presentation to: other bacterial diseases, leishmaniasis, viral disease, trauma, brushtail possum ulcerative dermatitis, swollen paw syndrome in ringtail possums

Also called Bairnsdale ulcer in VIC, and Daintree ulcer in Qld, *Mycobacterium ulcerans* is a globally recognised disease of people and other animals living in tropical areas, and infection in Australia appears to be geographically spreading. Australia is the only country where this is also a recognised disease of wildlife. *M. ulcerans* has been recovered from clinically well animals and animal faeces for many years, however, clinical case of infection in free-ranging wildlife have more recently been recorded (Wildlife Health Australia, 2021).

Adult ringtail possums appear to be most commonly clinically affected by this bacterium. In wildlife, the most common presentation is an ulcerative lesion on the nose, ear, paw or tail, and lethargy. Similarly, non-healing ulcers are typical in humans and domestic species (Wildlife Health Australia, 2021). *M. ulcerans* produces a toxin that causes tissue necrosis and supresses the host's immune response, resulting in ulcers that are often quite extensive, and fail to heal. Minor infections may be

self-limiting and heal spontaneously without treatment, however in severe cases of infection euthanasia may be worthy of consideration. A direct smear of the ulcer stained for acid-fast bacilli is a good first step, however diagnosis should be based upon culture, histopathology, and PCR.

Considered an environmental pathogen, modes of transmission of this disease are unknown however in people, exposure is likely to be via contaminated water, vegetation, soil or arthropod vectors. There is no indication that this particular microbe is zoonotic, however infected animals and people occupy the same environment.

4.4 Yersiniosis

Yersinia pseudotuberculosis is a ubiquitous gram-negative bacterium commonly identified within the faeces of wild birds and mammals. *Y. pseudotuberculosis* can persist in the environment for several years (Bender, et al., 2019). Transmission of this bacterium occurs via the faecal-oral route.

Although many animals will harbour *Y. pseudotuberculosis* within the intestinal tract without effect, the organism is capable of causing multisystemic illness. Yersiniosis results in either rapidly fatal enteritis and septicaemia, or subacute to chronic multisystemic abscessation. Clinical signs in animals experiencing the rapid, septicaemic form of the disease may include, profound depression, dehydration, diarrhoea and faeces containing partially digested blood. Animals suffering from the multisystemic form will have clinical signs associated with the organs infected. Outbreaks of yersiniosis are thought to be associated with stress, immunosuppression or poor hygiene.

Enteritis associated with yersiniosis is evident as foci of mucosal necrosis, while the more chronic form will have pale foci scattered throughout many organs upon gross post mortem examination. These pale areas represent foci of necrosis, which are infiltrated with neutrophils and macrophages. Diagnosis of yersiniosis is achieved by isolating the organism within lesions. *Y. pseudotuberculosis* can be difficult to isolate in microbial culture.

Successful treatment is best achieved based on microbial culture and antimicrobial sensitivity testing; however, once clinical signs are apparent, animals may respond poorly to therapy. High standards of husbandry and hygiene are used to prevent yersiniosis. It is important to protect food and water supplies from contamination with the faeces of wild birds. Minimising stress may also assist in the prevention of yersiniosis.

4.5 Tyzzer's Disease

Tyzzer's disease is the clinical syndrome caused by infection with the bacterium *Bacillus piliformis. B. piliformis* is a gram negative aerobic, spore forming rod shaped bacterium. Tyzzer's disease has been reported in koala, wombat, dasyurids, and native rodents and there have been reports of fatal necrotising hepatitis and myocarditis in young possums (Canfield & Hartley, 1991). In laboratory animals, mortality associated with *B. piliformis* usually occurs subsequent to stressful situations such as weaning, poor sanitation, overcrowding, transport, or concurrent disease. The only means of preventing Tyzzer's disease is to attempt to reduce the stressors placed on young hand raised possums, and adherence to the highest standards of hygiene. This organism does not grow in culture, so a presumptive diagnosis is achieved through the identification of long bacilli within heart and liver lesions that stain with silver. The diagnosis is confirmed via PCR, which is available from commercial veterinary pathology labs that specialise in rodent diagnostic investigations.

4.6 Tularaemia (Francisella tularensis holarctica)

Zoonotic: Yes
Reportable: Yes
Species records: Ringtail possum
Similar presentation to: mass mortality or sudden death, other bacterial infection, viral infection

Tularaemia is the name given to the disease caused by the bacterium, *Francisella tularensis*. It is commonly found in wildlife in the northern hemisphere with rabbits and rodents being important reservoirs for disease (Eden, et al., 2017). Overseas, mass mortality events have been recorded in rabbits, beavers and muskrats, and infection can modulate population densities (Wildlife Health Australia, 2020). Humans may become infected following contact with infected wildlife, being bitten by infected haemophagous vectors such as ticks, or via contact with contaminated environmental sources including water.

Tularaemia was first described in Australia in 2017 despite sporadic unconfirmed cases having been reported in humans since 2003. Eden, et al. (2017) described the first confirmed case of tularaemia in Australia, with the bacterium, *Francisella tularensis holarctica* biovar *japonica*, identified through genomics and culture of ringtail possum tissues from Sydney that died during separate mass mortality event in 2002 and 2003.

Tularaemia is highly infectious and care should be taken when handling ringtail possums to avoid contact with blood or tissue, inhalation of aerosols or particles, or ingestion of contaminated water (Wildlife Health Australia, 2020). Tularaemia should be considered as a differential diagnosis for individual or mass mammal mortality events where there is microscopic evidence of septicaemia, including intracellular fine bacteria, particularly within the liver and spleen. These bacteria will not grow in routine bacterial culture, and PCR is the preferred method of diagnosis. As this is a notifiable disease, diagnostic assistance is available through your state agriculture lab.

5 Fungal Diseases

5.1 Ringworm

Ringworm, or dermatophytosis, is a fungal infection of the skin, hair or nails, typically caused by superficial infection with *Microsporum* sp. or *Trichophyton* sp. It is not a parasite as the name would suggest, but is rather named after the ring-like appearance that is indicative of infection in people. In animals, lesions are not always ring shaped, but the infection is zoonotic and can pass from animals to people, especially children. The fungi responsible for ringworm may also be soil-borne and infection appears spontaneously through contact with the environment. Immunocompromised or immature animals are more likely to become infected.

Ringworm can infect a wide variety of species, appearing as a circular or irregular area of alopecia which may heal in the centre as infection expands. Skin may be inflamed or scaly, and secondary bacterial infection may occur. Pulling hair from the margin of the lesion for microscopic examination and culture is a useful diagnostic exercise, however culture of fungus may take several weeks. Infection, and lesions are generally self-limiting and heal without treatment. Care should be taken not to spread infection by reducing contact with other animals. Sound hygiene is also recommended for animals in rehabilitation care.

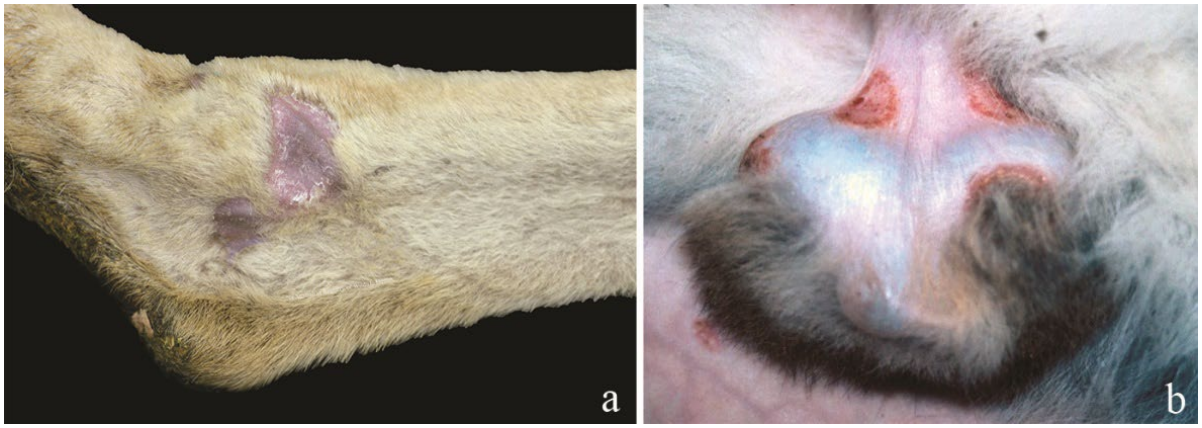

Figure 16 Ringworm a) eastern grey kangaroo, leg, b) pademelon, testicles

5.2 Candida spp.

Candida spp are yeasts that are commensal within the upper gastrointestinal tract. Disease associated with an overabundance of this organism occurs most commonly in young, hand-raised possums, particularly under conditions of poor hygiene, inappropriate hand rearing formulas, antibiotic therapy, or significant stress. Oesophagitis and gastritis are frequently diagnosed in native species that have been hand raised or have concurrent disease. Affected animals may experience a short course of diarrhoea, but are often found dead. Microscopic examination of the oesophagus and squamous portions of the stomach reveal foci of mucosal necrosis, inflammation and erosion.

Among short-beaked echidna, *Candida albicans*, or one of its telomorphs, is most commonly identified in microbial culture and during histologic examination of lesions. A small number of wild, young ringtail possums are reported with a moist dermatitis caused by *Candida* sp. Adult brushtail and ringtail possums that are administered broad-spectrum antibiotics orally, or those possums that have concurrent systemic illness, are also susceptible to candidiasis on the skin or in the gastrointestinal tract.

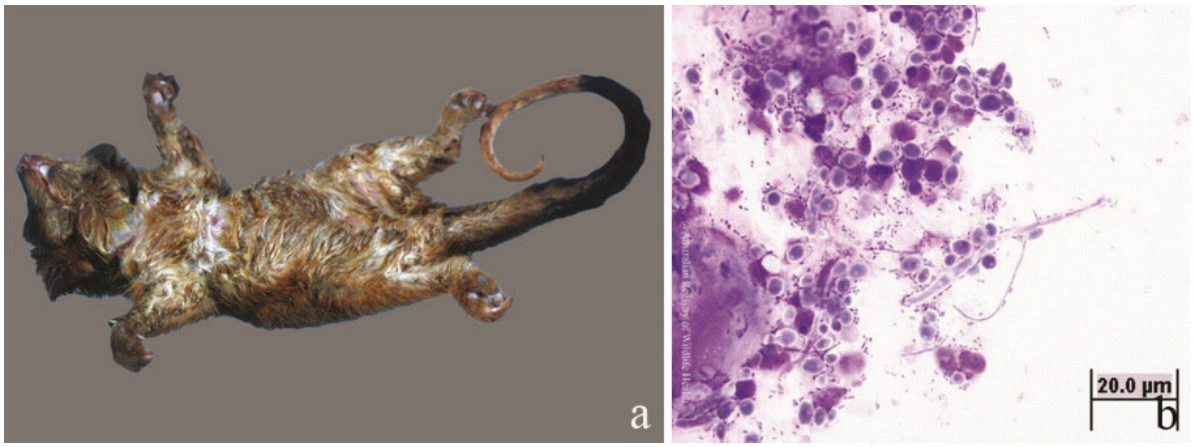

Figure 17 a) Young ringtail possum with moist dermatitis, Candida albicans, b) budding yeasts and hyphae of Candida albicans

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

Diagnosis of candidiasis relies upon microscopic examination of smears made from oral lesions, moist skin lesions, or wet preparations of faeces, and standard fungal culture techniques. Gram stains and Diff Quik® stains are useful to illustrate the presence of yeast within smears. *Candida* spp. are commensal within the gastrointestinal tract, and scattered yeast cells within tissue smears or faeces

are not unusual. The presence of large numbers of budding yeast, and pseudohyphae reflect active infection with *Candida* sp.

5.3 Cryptococcus sp.

Zoonotic: Yes
Species records: koalas, short-beaked echidna, eastern barred bandicoot, Gilbert's potoroo, quokka, feathertail glider, stick nest rat, sugar glider, leadbeater's possum, ringtail possum, yellow-bellied glider, mountain pygmy possum, numbat
Similar presentation to: neoplasia, lumpy jaw, other fungal disease, bacterial infection, parasitic infection, herpesvirus

Cryptococcosis is the term used to describe clinical infection by either *Cryptococcus neoformans* or *C. gattii*, a yeast-like organism with a thick capsule that does not stain with H&E on routine histopathology. While infection is most commonly associated with koalas, cryptococcosis can cause sporadic disease in a wide range of Australian species, including humans. Transmission is likely from inhalation of contaminated soil or other organic matter.

Clinical signs of infection with *Cryptococcus* sp. can include neurological signs such as head tilt, circling, incoordination, or dilated pupils, respiratory signs, abdominal distension, and sudden death. On gross post mortem examination, round, white-tan masses may be seen within, or adherent to affected organs. Lesions are most commonly found in brain and meninges, sinuses, lungs, and lymph nodes.

Figure 18 Eastern barred bandicoot, Cryptococcus sp. a) left mandibular and maxillary swelling, b) enlarged gelatinous submandibular lymph nodes, c) mottled lungs with discrete white lesions

Cryptococcosis can be diagnosed using serology, histopathology with PAS or silver stains, immunohistochemistry and culture. In live animals, serial serology testing should be utilised to understand infection status. In rehabilitation centres where koalas are housed, enclosures should be thoroughly disinfected between animals, and branches should be regularly removed and replaced to decrease environmental load. Fungal culture should only be attempted by experienced laboratories, as pure cultures of this organism pose a substantial zoonotic risk. Always communicate your suspicion of cryptococcal infection when submitting samples to a laboratory.

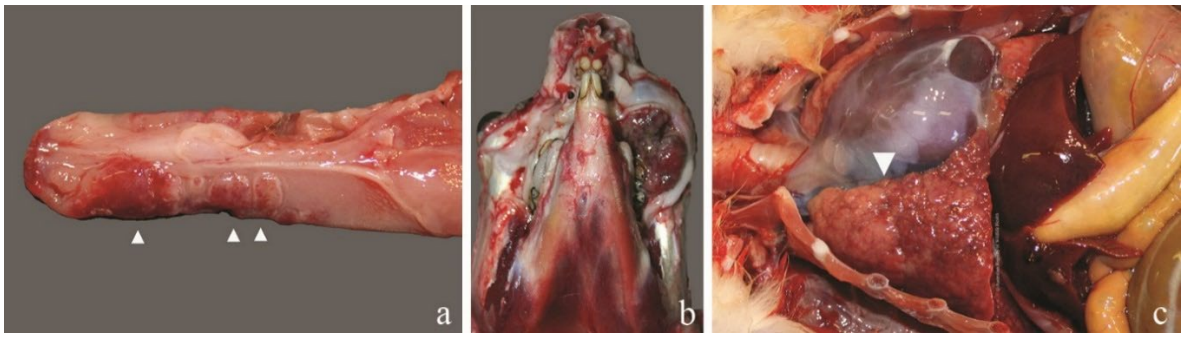

Figure 19 Koala, Cryptococcus gattii a) ulceration ventral aspect of tongue (arrows), b) necrotising sinusitis and facial osteomyelitis with friable maxillary mass, c) coalescing nodular changes in the lungs

5.4 Histoplasma capsulatum

Zoonotic: Yes
Reportable: *Histoplasma farciminosum*
Species records: horses
Similar presentation to: steatitis, neoplasia, lumpy jaw, other fungal disease, bacterial infection, parasitic infection

Although not reported to be a pathogen of Australian bats, *Histoplasma capsulatum* has an affinity for substrates containing large concentrations of nitrogen, such as the faeces of birds and bats. This is an environmental fungus capable of causing severe disease in humans, who are usually infected with *H. capsulatum* through inhalation. Granulomatous enteritis, osteomyelitis, and endophthalmitis often develop after the initial pulmonary infection, and the disease is usually fatal. Care needs to be taken when working in environments where there are abundant bird or bat faeces. Rare cases of multisystemic granulomatous disease are documented in Australian mammals (Registry).

5.5 Emmonsia spp.

Spores of the fungi *Emmonsia parvum* and *E. crescens* are frequently encountered during histologic examination of wombat pulmonary tissue. These fungi are most often localised within the alveoli at the periphery of the lung, and are most often unaccompanied by significant inflammation. These organisms appear to be an incidental finding in burrowing species.

6 Viral Diseases

6.1 Poxvirus

Figure 20 Pox-like lesions on the feet of a juvenile eastern grey kangaroo (Image courtesy of Slade Macklin).

Poxvirus lesions are occasionally found on the extremities of marsupials, most commonly juvenile eastern grey kangaroos. Western grey kangaroos, red kangaroos, common wallaroos, tammar wallabies, agile wallabies, swamp wallabies, Tasmanian pademelons, quokka, common brushtail possums and common ringtail possums, and short-beaked echidnas may also be affected (Sarker, et al., 2017; Bender, et al., 2019). In eastern grey kangaroos, the virus has been described as eastern grey kangaroopox virus (Sarker, et al., 2017). An orthopox virus was characterised following an epizootic in captive ringtail possums (Bender, et al., 2019). It is unsure whether these same poxviruses affect other species.

Lesions are generally raised proliferative round dermal nodules which can be found on feet, tail or face. Lesions have also been described on the tongue of ringtail possums (Bender, et al., 2019). The mode of transmission is unknown, however close contact and vector spread are likely (Sarker, et al., 2017). Lesions are generally self-limiting and will heal over time without treatment.

6.2 Papillomavirus

Papillomaviruses can be found in a number of animal species, including humans, however these viruses are highly host specific and are not considered zoonotic. The virus may be present in healthy

animals, or it can present as epithelial proliferations (wart-like lesions) on the skin. Papillomavirus has been described from koalas, eastern-grey kangaroos, short-beaked echidnas and brushtail possums (Antonsson & McMillan, 2006). In addition, the Registry hold records for numerous other cases of papilloma-like cases including long-nosed potoroo, bilby, and tiger quoll. A syndrome described in both wild and captive western barred bandicoots in WA caused by infection with bandicoot papillomatosis carcinomatosis virus type 1 (BPCV1) has hampered conservation of the species, and highlighted the importance of conducting thorough disease risk assessments prior to translocation of any species (Woolford, et al., 2009).

In most species, clinical symptoms are usually spontaneous and generally associated with wart-like skin lesions. Lesions are usually self-limiting however they may present on any mucous membranes including the mouth, anus or cloaca and persistent lesions can become debilitating.

BPCV1 is characterised by cutaneous and mucocutaneous lesions that resemble typical papilloma lesions, to larger tumour-like carcinoma and squamous cell carcinomas (Woolford, et al., 2009). Lesions may cause the animals to have ambulatory problems and difficultly seeing or eating. Secondary infections may cause further issues.

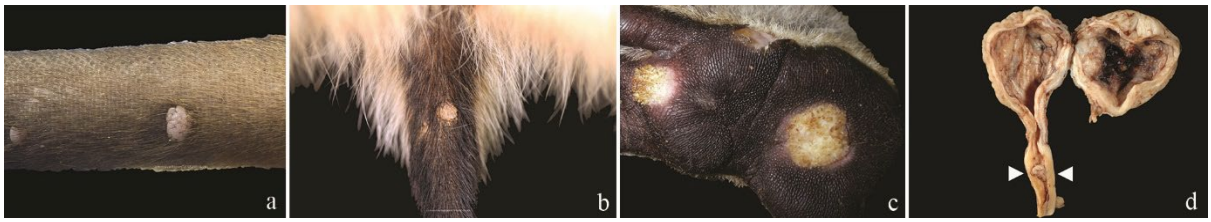

Figure 21 Papilloma like lesions a) long-nosed potoroo, tail, b) bilby, tail, c) koala, foot (Image courtesy of Dr V Nicholson), d) tiger quoll, urethra causing bladder rupture

6.3 Encephalomyocarditis Virus

Encephalomyocarditis virus (EMCV) is carried by rodents and a variety of species may become exposed via contact with rodent urine or faces. The virus is hardy in the environment and can survive in puddles for up to 150 days. Registry records contain macropods and southern hairy-nosed wombats affected by EMCV. Infections are more frequently reported in captive animals but rehabilitators should be aware of this disease.

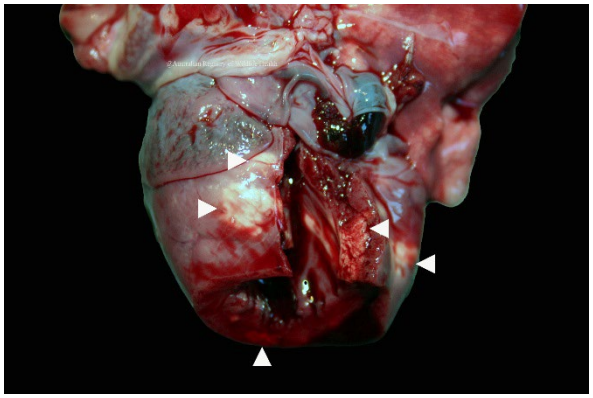

Figure 22 Tammar wallaby, heart, multifocal white lesions throughout myocardium, Encephalomyocarditis virus

Manifestations can range from sudden death with no grossly visible changes to subacute or chronic disease with marked visible necrosis inflammation or fibrosis in the heart. Some animals may develop antibodies to the virus with no clinical signs. Animals may present as anorexic, lethargic, have trouble breathing, have a fever, trembling, staggering, having trouble standing or acutely dead.

Serological testing can be performed on live animals to investigate exposure. Serum collected post mortem may contain antibodies, unless the animal died acutely. Histopathology, PCR, and virus isolation are useful to achieve a diagnosis post mortem. Good hygiene, including securing feed and bedding in rodent proof containers, is important to avoid infection.

6.4 Australian Bat Lyssavirus

Zoonotic: Yes
Reportable: Yes
Species records: All flying-foxes and insectivorous microbats
Similar presentation to: may be no clinical symptoms, trauma, *Angiostrongylus cantonensis*

Australian bat lyssavirus is a member of the family *Rhabdoviridae*. The Rhabdoviruses include classic rabies viruses and genetically related viruses. Nucleotide sequencing has indicated that Australian bat lyssavirus is closely related to European bat virus and classic rabies virus, sharing 92% nucleotide homology with classic rabies virus (Fraser, et al., 1996).

Lyssavirus has been isolated within both frugivorous and insectivorous bats throughout Australia. First discovered in flying-foxes in 1996, retrospective investigations identified the first known case in a black flying-fox that died in 1995. The first human death associated with Australian bat lyssavirus occurred in October 1996 in Rockhampton. A woman who cared for a variety of wild animals, including micro and megachiroptera, developed numbness, fever, headaches, and became comatose within a period of ten days. The woman was found to be seropositive for lyssavirus, and PCR tests identified lyssavirus antigen in her cerebrospinal fluid. Her illness progressed rapidly after the diagnosis was established, resulting in her death (Allworth, et al., 1996).

Many of the bats infected with Australian bat lyssavirus are non-symptomatic. Non-suppurative encephalitis occurs primarily in young bats, and is associated with an inability to fly, hindquarter paresis, weakness, and potentially with aggressive behaviour.

Rhabdoviruses are enveloped viruses that do not persist well in the environment. The viruses are rapidly inactivated with exposure to ultraviolet light, strong acids or bases, or detergents. Thus, human exposure to the virus occurs primarily through direct contact with saliva or nervous tissue of infected animals. Bite wounds are the most effective means of viral transmission. Animal handling protocols are required that will reduce or eliminate the risk of being bitten during any attempt to handle bats. Even with these protocols in place, any individual that has direct contact with bats should be vaccinated against lyssavirus.

Advice regarding vaccination, post-exposure therapy, and frequency of serological testing should be sought from your doctor, local public health agency, or state or territory health department. Individuals who regularly have direct contact with species known to carry lyssavirus should be monitored serologically every two years. Booster vaccinations may be required to ensure vaccination efficacy.

If bitten by a bat, wounds should be thoroughly and continuously scrubbed with soap and water for 15 minutes. The incident must then be immediately reported to public health officials who will decide whether post exposure immunisation is warranted. When a bat has bitten a person, the bat should be euthanased and its tissues submitted for rabies diagnosis however we do not recommend handling the bat further for this purpose unless trained to do so, and with the appropriate personal protective equipment to do so safely. Individuals who are bitten by an animal suspected to have lyssavirus will usually receive vaccination if previously unvaccinated, and vaccine boosters if previously vaccinated (Wildlife Health Australia, 2019).

Diagnostic testing to confirm Australian bat lyssavirus is conducted at Australian Centre for Disease Preparedness in Geelong, and can be accessed through your state agriculture department. Testing should be undertaken on any bat that bites or scratches a person, even if that person is vaccinated. Ensure that you collect and share contact details of the bitten or scratched persons along with the bat.

6.5 Hendra Virus - paramyxovirus

Zoonotic: Yes
Reportable: Yes
Species records: All pteropids, horses
Similar presentation to: may be no clinical symptoms

Hendra virus was discovered after two outbreaks of fatal respiratory disease in racehorses were experienced one month and 1,000 km apart in the spring of 1994. During a 16-day period beginning September, 1994 in Brisbane, 14 of 21 ill horses died or were euthanased due to acute and severe respiratory disease. Two people who worked very closely with the ill horses developed flu-like illness, and one of these people died (Murray, et al., 1995). Retrospective examinations conducted in October 1995 identified an outbreak of acute, severe respiratory disease that occurred one month before the incident described above. One person and two horses died of respiratory disease in this event in Mackay, 1000 km north of Brisbane (Hooper et al., 1996). The index case in both outbreaks involved heavily pregnant thoroughbred mares at pasture.

Virus neutralising antibody tests conducted in April 1996 identified Hendra virus antibodies in all four species of Australian flying-fox (spectacled, black, little red and grey-headed flying-fox) with a prevalence of approximately 9 to 12% (Field, et al., 1997).

Clinical signs of disease are not reported in flying-foxes infected with Hendra virus, although the virus has been isolated in various tissues from three of the four species of Australian flying-foxes (Field, et al., 1997). Human cases of Hendra virus infection have all been contracted from infected horses. Hygiene and husbandry protocols sufficient to prevent Australian bat lyssavirus should protect bat caregivers from active Hendra virus infection. However, bat caregivers should be aware of Hendra virus and should report the occurrence of severe flu-like illness to their medical practitioner.

Recently variant strains of Hendra virus have been identified in sick or dead horses that had previously tested negative for Hendra virus infection. Research is underway to further characterise Hendra virus diversity in Australia.

7 Nutritional Disease

7.1 Macropod progressive periodontal disease (Lumpy jaw)

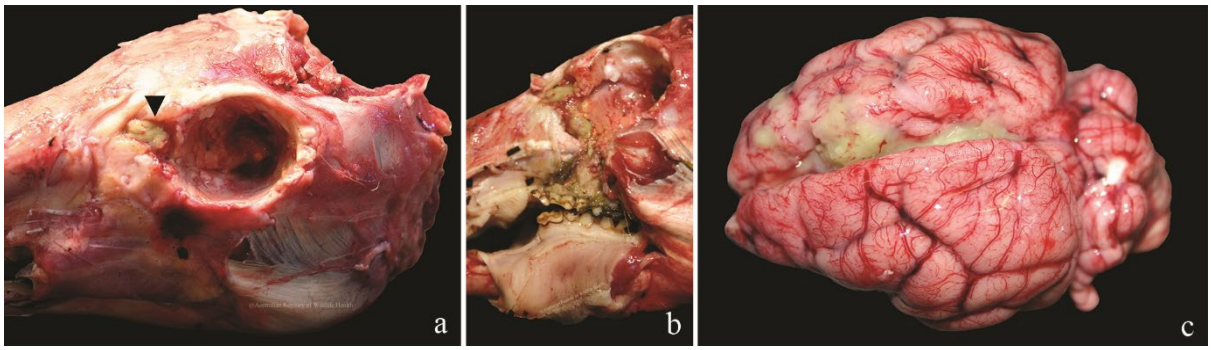

Figure 23 Eastern grey kangaroo with chronic macropod progressive periodontal disease (lumpy jaw) that has progressed to a draining tract near the eye (a, b), and abscessation in the brain (c)

Macropod progressive periodontal disease (MPPD), commonly called lumpy jaw, is a periodontal disease that affects both wild and captive macropods, and is often associated with inappropriate diet. Infection of the jaw is most commonly caused by the bacterium *Fusobacterium necrophorum* or *Bacteroides* sp. Infection may spread, creating a draining tracts toward sinuses, orbital bone, cranial

bones or the chin where secondary abscess may occur. Pneumonia and septicaemia are also common concurrent findings. MPPD is a chronic, progressive disease that is difficult to treat, and often fatal. MPPD should be differentiated from facial trauma, which most often affects the base of the incisors. Differentiating the two syndromes should assist prevention and mitigation.

7.2 Dental malocclusion

Wombats and native rodents have continuously growing teeth. Teeth that are fractured during trauma may be filed or clipped. Malocclusion of incisors and molars may result from irregular tooth wear, insufficient tooth wear or traumatic injury. Elongated teeth are filed or clipped while the animal is under general anaesthesia. Provision of sufficient browse should help to alleviate malocclusion in captive animals; however, repeated tooth clipping is often required. Euthanasia for malocclusion in rehabilitated or hand-raised wombats should be considered depending on severity as without continuous care this dental issue could result in oral trauma sufficient to lead to starvation in the wild.

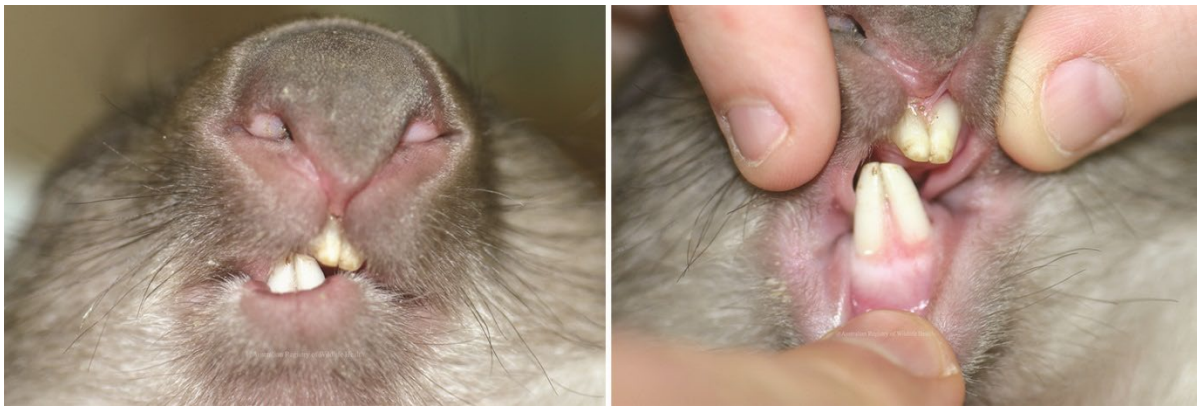

Figure 24 Bare-nosed wombat with malocclusion, lower jaw deviated to the right

7.3 Vitamin D intoxication / Calcinosis circumscripta

A syndrome of multisystemic mineralisation has historically been reported in captive bare-nosed wombats (Griner, 1983). Each wombat examined had significant arteriosclerosis and medial mineralisation of the aorta, pulmonary artery, and sub-endocardial tissue of the right atrium. Many of the animals examined also had marked calcification of the footpads. Chronic interstitial nephritis with multifocal interstitial mineralisation was also evident upon post mortem examination. Mineral deposits within the renal tubular lumina or within the interstitium consisted of concentric rings of basophilic, mineralised material (Griner, 1983). Several historical cases of multisystemic mineralisation in wombats are present within the Australian Registry of Wildlife Health. Affected animals had been hand raised and/or maintained on a diet composed primarily of dog food. Based on the gross and microscopic post mortem findings, in conjunction with the known diet, vitamin D intoxication or calcinosis circumscripta (unusual deposits of calcium salts often associated with sites of previous trauma) are the most likely cause of this syndrome. Both of these aetiologies are poorly understood in domestic species and little is known for wildlife.

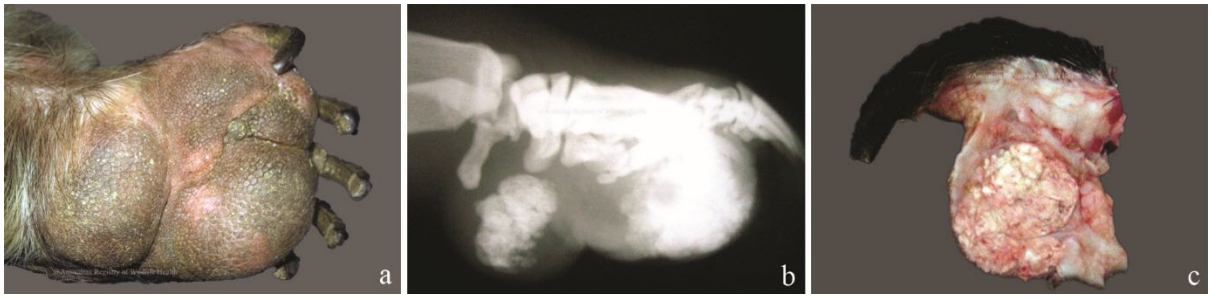

Figure 25 Bare-nosed wombat with calcinosis circumscripta: the most striking physical symptoms include muscle wastage and swelling and ulceration of foot pads (a), mineral deposits visible on radiographs (b), and caseous deposits in the soft tissue of the foot pads on cross section (c). Images courtesy of South Penrith Veterinary Clinic

7.4 High lactose diet

Occasionally hand raised possums are presented with concurrent enteritis and bilateral cataracts. These lesions are highly suggestive that the animal has been fed cow's milk or some other milk substitute containing high concentrations of lactose and galactose (sugar). The marsupial gastrointestinal system is not able to effectively digest these sugars and diarrhoea occurs as a result of the osmotic effect of undigested sugar within the small intestine. Cataract formation in these animals presumably occurs due to osmotic effects caused by the conversion of galactose to dulcitol. Dulcitol is a sugar alcohol that has the ability to draw fluids into the lens. Toxoplasmosis should be considered as a differential diagnosis in any young possum with cataracts.

7.5 Steatitis

Steatitis, or 'yellow fat disease', is a poorly understood condition whereby there is an unusual inflammatory response associated with adipose tissue, i.e. fat. Vitamin E and/or selenium deficiency and a high fat diet are thought to be contributory. Steatitis is generally an issue for captive animals fed a non-natural diet. Clinical signs may include anorexia, depression, difficulty breathing, fever and sudden death.

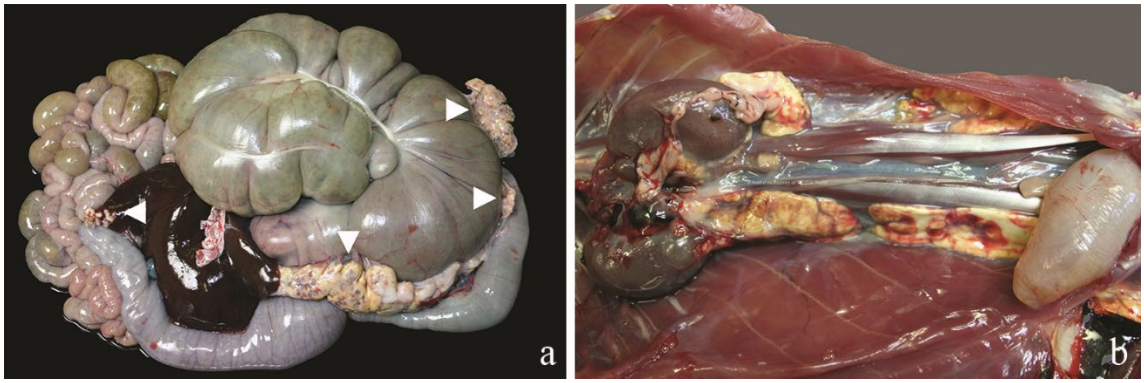

Figure 26 Yellow fat disease, or steatitis, in a) quokka with distinct firm, mottled yellow/red mesenteric fat lining the serosal surface of the intestinal tract and liver (arrows), and b) ringtail possum with affected dorsal abdominal and pelvic fat

8 Toxins

8.1 Lead Poisoning

Lead poisoning and organophosphate toxicity have been reported in flying-foxes; however, with the introduction of lead-free petrol, clinical disease from lead poisoning in flying-foxes is now rare. Heavy metal intoxication may still occur however and recent surveillance has confirmed while there is reduced exposure and uptake of lead in Australian flying-foxes, increased tissue concentrations of the heavy metal cadmium were documented (Pulscher, et al., 2020).

Flying-foxes with high concentrations of tissue lead may be unable to fly, have uncoordinated movements, tremors, vomiting, diarrhoea, constipation, anaemia, salivation, signs of secondary trauma, or they may abort (Sutton & Wilson, 1983; Sutton & Hariano, 1987; Wilson, 1988). The haemogram may demonstrate microcytic, hypochromic anaemia. The basophilic stippling of erythrocyte cytoplasm that is reported in ruminants suffering from lead poisoning is rare in other species, including bats.

Gross post mortem examination of flying-foxes with lead poisoning is unrewarding. Histologic examination usually reveals intranuclear eosinophilic inclusion bodies within the proximal convoluted tubules of the kidney. Intranuclear inclusion bodies have also been reported to occur in the brain, and liver (Wilson, 1988). These inclusion bodies stain positively with acid-fast stains. Flying-foxes suffering lead poisoning are treated with calcium disodium edetate (Booth, 1994). A second course of therapy may be required.

Chronic cadmium toxicity may be associated with renal damage, altered bone growth or strength, altered reproduction, neurological or cardio-respiratory damage (Pulscher, et al., 2021).

8.2 Chronic fluoride toxicoses

Fluoride exposure can happen naturally in the environment, but excessive or chronic exposure can occur due to anthropogenic sources such as industrial processes, mining and manufacturing (Death, et al., 2017). Fluoride has a high affinity for bone and teeth and animals with chronic exposure to high levels of fluoride may exhibit bony changes and subsequent debility. Lesions due to fluorosis have been described in eastern-grey kangaroos, *Pteropus* bats, swamp wallabies, red-necked wallabies, common brushtail possums, common ringtail possums, and koalas (Death, et al., 2017). Research has shown that the presence of lesions differs depending on how species eat and move, with lesions being more prominent in high impact areas.

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 and oxygenation. An animal is severely dehydrated when the eyes are sunken, the capillary refill time is very slow, the mucous membranes are dry and tacky, and the skin has lost its elasticity.

The neuroendocrine cascade that is initiated during shock is initially protective, but over time energy reserves are depleted and peripheral vasoconstriction contributes to hypoperfusion of tissues. The heart, lungs, liver, gastrointestinal tract, pancreas, and central nervous system are most susceptible to damage induced by low 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 low oxygen concentrations. Mucosal ulceration and decreased mobility occur with reduced blood flow and oxygenation of the gastrointestinal tract. 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 in the tissue 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, gastrointestinal ulceration or sepsis three to five days later. If reduced blood flow continues, pancreatic damage through low oxygenation can result in the release of vasoactive substances and myocardial depressant factor. Ultimately, reduced blood and oxygen flow to the brain causes nerve cell death.

9.2 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. 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 fur over the shoulders. Canid-inflicted bite wounds do not necessarily break the skin. The mild outward appearance of predator-induced lesions often masks very serious internal injuries. Feline bite wounds can puncture deep into the tissues, and felids have the potential to break bones or reduce the underlying muscle to pulp. Canine bite wounds are most often associated with circular subcutaneous contusions 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 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).

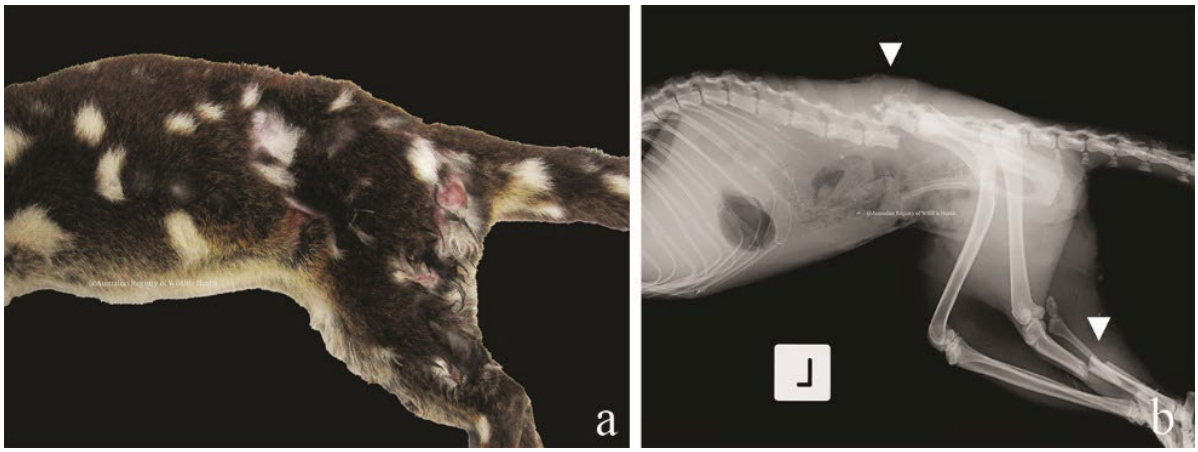

Figure 27 Tiger quoll with dog predation wounds a) superficial wounds to hind area, and b) radiograph showing complete fracture of the lumbar spine, tibia/fibula, and ileal crest, and hip luxation (Images courtesy of South Penrith Veterinary Clinic)

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.

9.3 Soft tissue injury in bats

Wing web lacerations are a common traumatic event in bats. These injuries usually heal well if necrotic tissue is debrided and the wound is kept clean with topical antiseptic agents. Surgical intervention in the treatment of wing web injuries often results in greater contraction and scar tissue formation than conservative wound management. Wound infection with *Candida spp*. and *Psuedomonas aeruginosa* occur primarily when poor husbandry prevails or when tight wing bandages are applied (Booth, 1994).

Abrasion of the wing tips occurs commonly in captive flying-foxes. These injuries can be slow to heal, and amputation of exposed bone may be required.

Flying-foxes lick and chew bandage material, sutures, and external fixators; thus, the use of Elizabethan collars, and buried sutures are recommended.

9.4 Exertional myopathy

Zoonotic: No
Species records: All, primarily macropods
Similar presentation to: nutritional, toxic, infectious and parasitic myopathies, trauma, *Angiostrongylus* sp.

Exertional myopathy, capture myopathy, or exertional rhabdomyolysis, are terms used to describe a syndrome characterised by damage to skeletal or cardiac muscle following a period of intense physical activity. Exertional myopathy occurs in a wide variety of mammals; however, some species appear to be more susceptible to developing clinically apparent disease than others. There is no association between sex or age and the development of exertional myopathy; however, elevated environmental temperature may increase the likelihood of an animal developing exertional myopathy (Williams and Thorne, 1996). Exertional myopathy is well documented in Australian macropods.

The clinical signs associated with exertional myopathy will vary depending on the types of muscles that have been damaged and the degree of damage sustained. The onset of this syndrome is usually precipitated by activities such as chemical or physical restraint, transport, or chase. Peracute death may follow such an incident, or clinical signs may begin days to weeks after the incident. Initial clinical signs include: increased heart rate, increased respiratory rate, and increased body temperature. The animal may then go on to develop depression, ataxia, fever, pulmonary oedema, unsteady or stiff gait, muscle tremors, and urine may become red to brown in colour. Recumbency and death may ensue in severely affected animals.

Clinical pathological changes that are associated with this syndrome include those related to systemic metabolic acidosis stemming from lactic acid release from muscles. Blood urea nitrogen concentration is elevated when substantial renal impairment has occurred. Renal damage occurs as a result of low blood and oxygen flow, or due to the toxic effect that myoglobin has after it is released from damaged muscles and is filtered into the kidneys. Both low oxygen and myoglobin result in acute renal tubular necrosis. Creatine kinase (CK), aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) concentrations are also markedly elevated. Serum concentrations of CK increase rapidly in response to cardiac or skeletal muscle damage, yet the enzyme has a short half-life and tends to persist in very high concentrations only in the face of ongoing muscle damage. Serum concentrations of AST and LDH are less specific to muscle damage, since they may originate from multiple organs. These analytes have a longer half-life and persist longer after a single point of muscle injury. Serial monitoring of AST and CK may provide some insight into the duration and degree of muscle damage.

Muscle damage as a result of exertional myopathy is primarily due to a combination of reduced blood perfusion, acidosis and depletion of ATP reserves. Damage to the muscle cells is degenerative rather than inflammatory. Delayed peracute death syndrome occurs when an animal dies suddenly after a second episode of exertion or stress. This syndrome may occur due to the presence of pre-existing cardiac lesions, increasing the sensitivity of the animal to the effects of potassium released from damaged muscle cells and lactic acidosis associated with a second exertional episode. Skeletal muscle cells can regenerate if the cell membrane remains intact. Cardiac muscle cells have poor regenerative capacity.

Compartment syndrome occurs primarily in muscles that are bound by a tight fascia such as those in the legs of macropods. When oedema occurs, secondary to lactic acid build up in these muscles, the internal pressure can be so great that it compresses the muscle's venous supply. The combination of arterial integrity and venous blockage result in massive oedema, congestion, haemorrhage and ischemic necrosis throughout the affected muscle.

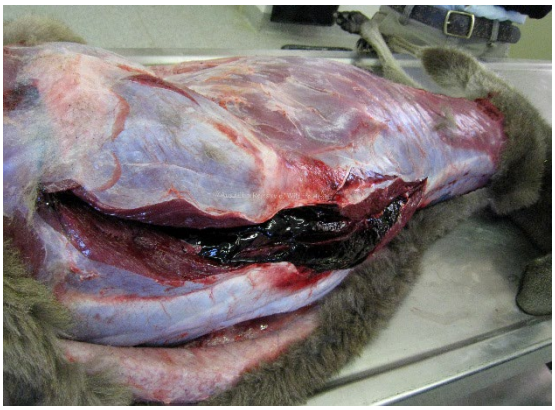

Figure 28 Western grey kangaroo, capture myopathy, extensive haemorrhagic necrosis to dorsal, brachial, and gluteal muscles

Bloody or dark brown urine often occurs in animals with myopathy as muscle cells break down and liberate myoglobin into the blood stream to be filtered into the urine. Shock often occurs in animals suffering exertional myopathy due to the compounding effects of acidosis, reduced cardiac function, dehydration and uraemia.

Lesions evident on gross post mortem examination of an animal with exertional myopathy may include: haemorrhage or oedema throughout muscles, muscle pallor or pale streaking, gritty or chalky muscle texture (mineralisation of sub-acute to chronic injuries), swollen kidneys, with a dark cortex, dark brown urine, pulmonary congestion and oedema, and/or haemorrhage and congestion in the adrenal cortex. Any skeletal muscle may be affected; however, the hind limb adductor muscles are particularly prone to post-exertional degeneration. Muscles may appear swollen and haemorrhagic or soft, dry, pale and friable. Muscles affected by compartment syndrome are often markedly swollen, haemorrhagic, congested and muscle fibres are friable as a result of ischaemic necrosis.

Microscopic lesions in muscles will vary depending on the severity and duration of the insult. The primary pathological process evident is cell degeneration, with a regenerative response visible several days to weeks after the initial damage. Lesions may be evident within the cardiac musculature, skeletal musculature, and in severe cases in the striated muscle of the tongue, diaphragm, and oesophagus.

The objectives of therapy for exertional myopathy are to reverse concurrent shock or hyperthermia, reverse metabolic acidosis, and stabilise cellular membranes. Fluid therapy and correction of acidosis through administration of sodium bicarbonate are initiated to increase tissue perfusion and prevent myoglobinuric nephrosis. Administration of steroids may contribute to stabilising cell membranes to prevent ongoing or irreversible cell degeneration.

Myopathies are considered to be extremely painful, and associated mortality rates are very high. Animals that survive the initial episode of myopathy may go on to relapse upon subsequent exertion. Preventive planning is paramount to reducing the risk of exertional myopathy. The prevention of exertional myopathy should be a consideration each time a wild animal is to be handled. Factors such as increased environmental temperature, inexperienced staff members, traumatic restraint devices,

and prolonged duration of restraint increase the risk of the animal developing exertional myopathy. The administration of vitamin E and selenium at the time of an exertional episode has been proven, in some species, to reduce the risk of subsequent myopathy (Williams & Thorne, 1996). Supplementation of the diet with these nutrients in advance of a stressful event may be warranted. Diazepam has been shown to reduce the prevalence of myonecrosis in macropods when administered intramuscularly after capture (Shepherd, 1982).

9.5 Electrocution

Flying-foxes and brushtail possums are often reported as having died due to electrocution on power lines, however other events involving native species have also been recorded including entanglement in electric fences and lightning strike. The Registry has documented a number of cases where lightning strike may have caused mass mortality events in gliders, and other small mammals. There may be no lesions apparent to account for these events, however in the absence of other lesions or intoxicants, electrocution is a reasonable differential diagnosis.

Most animals that are electrocuted die acutely as a result of ventricular fibrillation and pulmonary oedema (Cooper, 1996). Animals that are electrocuted may have lesions that can be traced along the flow of the current, often with a distinct entry and exit wound. Thus, simultaneous wing and foot injuries are common in electrocuted bats. Electrocuted animals also tend to have a characteristic odour. If the animal survives the acute effects of electrocution, they will usually have thermal injuries that become progressively more severe and exudative as the affected tissues begin to die. It is not unusual for electrocuted animals to be found with debility and an unusual odour, but few lesions. Then over a course of two to five days, ulcerative wounds develop along the path of the current. This progression of clinical signs is most likely the result of ischaemic tissue necrosis stemming from thermal injury to blood vessels.

9.6 Skeletal injuries

Native animals that are injured by motor vehicles tend to suffer spinal injuries, pelvic fractures, concussion, or massive contusions. Radiography is warranted when any animal presents with evidence of traumatic injury.

External examination is not a sensitive tool for the detection of traumatic injuries in short-beaked echidna since the presence of cutaneous spines prevents thorough palpation of the skeletal system. The oronasal structure of the short-beaked echidna is a sensitive and fragile organ. Unfortunately, the bones of the beak often sustain comminuted or paired fractures, and soft tissues are severely damaged during motor vehicle induced injury. Internal fixation of these bones is not possible, and the animals will not tolerate external fixation. In addition, the senses necessary for effective foraging are often permanently damaged. Cage rest may help minor injuries to the beak resolve, but euthanasia may be indicated for severe damage.

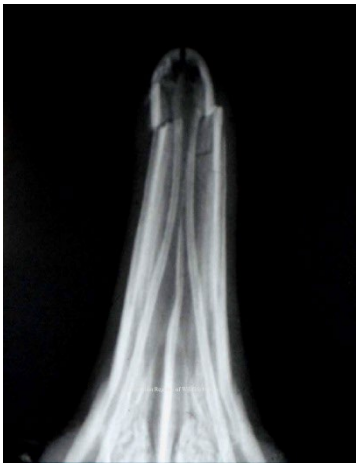

Figure 29 Short-beaked echidna with severe fractures of the beak

Ringtail and brushtail possums have prehensile tails and can incur tail pull injuries resulting in spinal cord damage. Significant trauma to the tail renders most ringtail possums in a condition unfit to be released into the wild. Ringtail possums also frequently suffer traumatic unilateral luxation of the coxofemoral joint. Treatment of this condition has not been successful.

Humeral fractures are the most common skeletal injury in flying-fox presented to wildlife care centres. The humerus is highly curved and bone repair is accomplished through the use of intramedullary pins. Radial fractures are repaired through intramedullary pins inserted retrograde at the elbow. A fractured phalanx can be cast, amputated or pinned, depending upon the site and degree of associated soft tissue damage. Fractured phalanges can be difficult to repair due to the paucity of surrounding soft tissue structures, and the difficulty in adequately immobilising the area. Fractured distal femoral epiphyses can result when excessive force is used to pull a young flying-fox from a wire enclosure. These fractures are repaired surgically with the use of cross pins (Heard, 1999).

Vertebral luxations and compression fractures are not uncommon in flying-fox that fly into stationary objects. These injuries primarily occur at the junction of the most caudal thoracic and the first lumbar vertebra. The location of these vertebral injuries is similar to birds. Both birds and bats have a rigid thoracic skeleton and vertebral injury may be more likely to occur at the first site of flexibility, the thoraco-lumbar junction.

9.7 Burns

Wild animals sustain thermal injuries from contact with any number of hot items in an urban environment. Wildlife may also sustain severe thermal injury during bush fires. Chemical burns have been suspected as the cause of melting ulcerative wounds in wildlife, where the wounds were localised to parts of the body where contact with caustic agents may occur.

Clinical signs of burn wounds may vary from charred fur to widespread necrosis and secondary suppurative inflammation. Animals may suffer concurrent inhalation pneumonia or traumatic injury. When greater than 25% of the animal's skin surface is burnt the animal is highly likely to succumb to systemic illness, such as shock, sepsis, renal failure, and anaemia. Australian fauna with less than 15% of their body affected by burns are considered to have a reasonable prognosis. The prognosis deteriorates when greater than 15 % of the skin surface area is burnt, and when the head and joints are affected.

Figure 30 Southern brown bandicoot with burns to tail, hind feet, and fore feet (insert) following a bushfire

Burns occurring in animals are most often categorised as first degree, second degree or third degree burns. First-degree burns involve incomplete destruction of the epidermis, and healing is achieved rapidly through re-epithelialisation. Second-degree burns are defined as damage to the epidermis and variable quantities of dermis. These injuries heal primarily through re-epithelialisation from adnexal remnants. The degree of residual alopecia will depend upon the number of follicles completely damaged. Third degree burns involve the complete destruction of the epidermis, adnexa, and nerve endings. Healing of full-thickness burns occurs slowly through wound contraction, and migration of epidermal cells from the wound margins. Animals suffering second or third degree burns to greater than 50% of the skin surface have a very poor prognosis for recovery and euthanasia may be warranted.

Burn injuries break the barrier protection offered by skin and provide a route for either localised or systemic invasion by bacteria. These wounds are often initially colonised by gram-positive bacteria; however, over the course of three to five days, gram-negative bacteria tend to invade. *Pseudomonas aeruginosa* is a prevalent pathogen involved in the opportunistic infection of burns. Wearing latex or nitrile gloves when handling burnt animals may reduce the likely transmission of *Staphylococcus Streptococcus* spp. and other bacteria from our skin to the open wounds.

10 Diseases of Uncertain Aetiology

10.1 Swollen Paw Syndrome of ringtail possums

Ringtail possums within the Sydney region have been presented to wildlife rehabilitation facilities with swollen and gangrenous paws since 1990. The apparently high prevalence of possums exhibiting similar clinical signs quickly lead to the description of a syndrome known as Swollen Paw Syndrome. These days, possums presenting with this syndrome are less common.

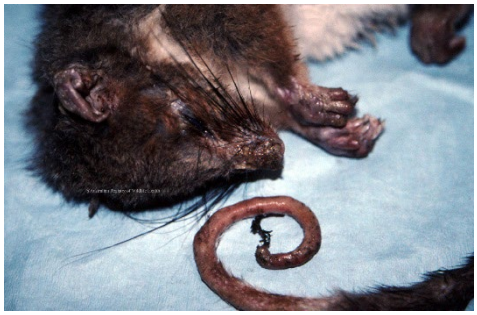

Figure 31 Ringtail possum with swollen paw syndrome exhibiting swollen and ulcerated paws, moist dermatitis, and curling and necrosis of the ear pinnae and tail tip

Animals with Swollen Paw Syndrome are bright, alert and continue to eat and drink. Clinical signs begin with oedema of the front paws. Animals with more advanced disease exhibit many of the following lesions: swollen and ulcerated paws, moist or ulcerative dermatitis of the bridge of the nose, alopecia, curling and necrosis of the tips of the pinnae, or moist dermatitis of the tips of the pinna, and/or multifocal dry, encrusted, or ulcerated lesions on the tail tip. Ulcerative lesions occur on either the ventral, dorsal or medial surfaces of the paws. Suppurative tenosynovitis may occur in ringtail possums with deep ulceration, primarily in the paws of the hind limbs. Some animals will have complete avascular necrosis of the paws. Ringtail possums are occasionally observed in the wild missing one or more of the distal extremities, and deformed ears. Other possums are found with alopecia along the sites described above. It has been speculated that these animals have suffered avascular necrosis associated with swollen paw syndrome.

Ringtail possums affected by Swollen Paw Syndrome usually have reduced fat stores, but have reasonable muscle mass. Gross post mortem examination does not reveal significant findings in addition to those seen on external examination. The pathogenesis of this disease is poorly understood and an effective treatment regime has not yet been established.

10.2 Exudative Dermatitis of brushtail possums

Zoonotic: Unknown
Species records: brushtail possum
Similar presentation to: trauma, Buruli ulcer

Severe and extensive exudative dermatitis is a somewhat common finding in brushtail possums admitted to urban wildlife care centres. Brushtail possums with this syndrome are found in an emaciated state with full thickness ulcerative lesions along the skin of the lumbosacral region, hips, flanks, tail base, and lateral thighs. The skin covering the head and forelimbs is less often affected with lesions however lesions can be severe. Lesions occurring early in the progression of the disease are characterised by alopecia, matting of the hair, and thickening of the skin. In more advanced lesions the skin is ulcerated and exudative.

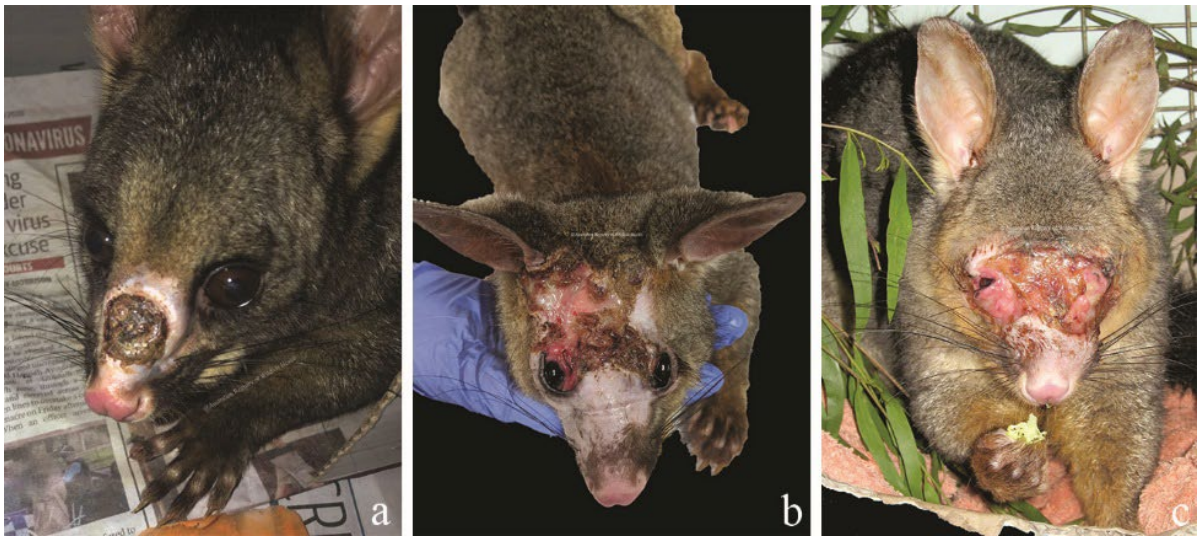

*Figure 32 Common brushtail possum with progressive facial lesions of exudative dermatitis syndrome from a) small ulcer over nose, b) lesion expanding over face including alopecia and ulceration, c) severe exudative lesion involving eyes (*not the same animal)*

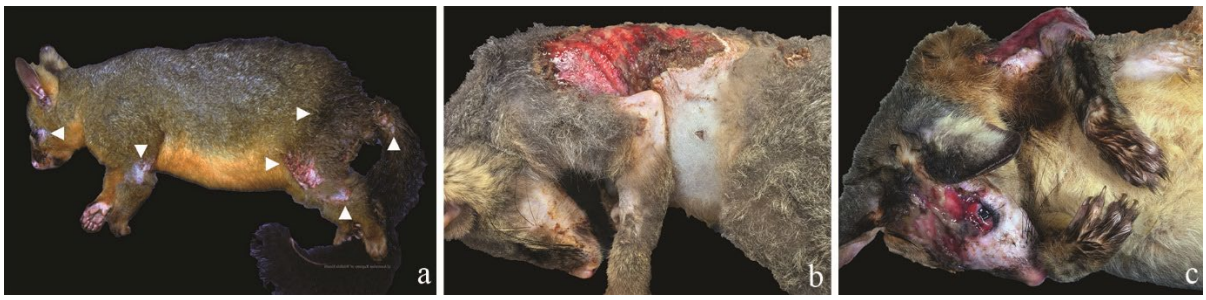

Figure 33 Common brushtail possum with ulcerative dermatitis body lesions including a) matting of fur around rump with associated ulcerative lesions, including face and limbs (arrows), b) large severe ulcerative lesion over thorax, c) severe facial and forelimb ulceration

The histologic appearance of these ulcerative lesions is highly variable. Presumably this variability is a result of both the timing of sample collection with respect to the stage of disease, and the role of secondary infection. Microscopic lesions may range from proliferation and thickening of the epidermis and adnexal glands to marked ulcerative dermatitis with dermal oedema and neutrophilic infiltrates (Bender, et al., 2019).

The exact aetiologic agent responsible for exudative dermatitis is uncertain, and it may be that initiation of this syndrome relies upon multiple factors. Proposed aetiologies include: *Trichosurolaelaps crassipes*, the common mite of brushtail possums, hypersensitivity reactions, bacterial infection, fungal infection, fighting wounds, and other traumatic injuries that become infected (Reddacliff, 1981; Bender, et al., 2019). A wide variety of bacteria have been isolated within the exudative wounds. Exudative dermatitis has been reported to occur most commonly in animals subject to social stress. High population densities, heavy rains, and high relative humidity have been proposed as risk factors in the development of exudative dermatitis.

Administration of broad-spectrum antibiotics and topical wound therapy may result in clinical improvement of the skin lesions. Bacteria isolated in microbial culture are often susceptible to most antibiotics, but culture is still recommended to guide our understanding of the syndrome and the treatment of individual animals. Wound contracture may result in limited mobility of limbs in severely affected animals. It is important to ensure that suitable habitat is available to release the animal prior to undertaking both intensive and long term wound therapy.

10.3 Wobbly possum disease

Chronic meningoencephalitis and optic neuritis occur in brushtail possums in eastern Australia and Tasmania in a syndrome often referred to as wobbly possum disease. A similar syndrome occurs in brushtail possums in New Zealand which has been attributed to a virus coined artevirus (Arteriviridae) wobbly possum disease virus (WPDV-NZ) (Chang, et al., 2019). A recent retrospective study into wobbly possum disease in Australia found genetically distinct arteriviruses, named AU1 and AU2 in mainland brushtail possums (Chang, et al., 2019). This research suggests that the mainland Australian, and New Zealand viruses that cause this syndrome are similar but distinct.

In Australia, adult possums are most often affected; however, this disease has also been reported in sub-adult animals and pouch young. Clinical signs of disease may progress over a period of weeks to months. Depression, blindness, ataxia, incoordination and abnormal gait are the most consistent clinical findings. Many possums will also have ophthalmological abnormalities consisting of foci of tapetal discoloration, a pale optic disc, or a disc that lacks the normal vascular tuft. These animals are clinically blind, and have dilated pupils.

Gross post mortem examination of animals with chronic meningoencephalitis does not reveal significant lesions, and microscopic examination of a range of tissues is required to achieve a presumptive diagnosis. Histologically, brushtail possums with wobbly possum syndrome have inflammation of the meninges and parenchyma of the brain characterised by perivascular accumulations of lymphocytes, plasma cells and smaller numbers of macrophages. These inflammatory changes can also be demonstrated in the optic nerve and choroid (vascular lining at the back of the eye) of blind possums. Degeneration of the axons in the optic tract occurs in many of the blind possums with this syndrome. Possums with chronic meningoencephalitis may have atrophy of the cerebellar folia or retinal atrophy.

Brushtail possums from New Zealand and from Tasmania that have wobbly possum disease also have lymphoplasmacytic inflammation in their livers, kidneys and sometimes other organs. These changes are not seen in mainland Australian wobbly possums. New Zealand was populated with brushtail possums from Tasmania, so the viruses in those locations may be different from those on the mainland. Investigations to compare viruses in Tasmanian possum to those in New Zealand and mainland Australia are underway at CSIRO.

Mainland Australian wobbly possums were often found co-infected with a hepacivirus and the contribution of this virus to the disease syndrome is uncertain (Chang, 2019).

A variety of therapeutic efforts have been unsuccessful.

11 Animals mentioned in text

Agile antechinus (*Antechinus agilis*)
Agile wallaby (*Macropus agilis*)
Atherton antechinus (*Antechinus godmani*)
Bare-nosed (common) wombat (*Vombatus ursinus*)
Bilby (*Macrotis lagotis*)
Black flying fox (*Pteropus alecto*)
Black wallaroo (*Macropus bernardus*)
Brush-tailed bettong (Woylie - *Bettongia penicillata*)
Brush-tailed phascogale (*Phascogale tapoatafa*)
Common brushtail possum (*Trichosurus vulpecula*)
Common planigale (*Planigale maculate*)
Common ringtail possum (*Pseudocheirus peregrinus*)
Common (northern) wallaroo (*Macropus robustus*)
Dingo (*Canis lupus dingo*)
Domestic dog (*Canis familiaris*)
Domestic horse (*Equus caballus*)
Domestic sheep (*Ovis aries*)
Dusky antechinus (*Antechinus swainsonii*)
Eastern barred bandicoot (*Perameles gunnii*)
Eastern freetail-bat (*Mormopterus norfolkensis*)
Eastern grey kangaroo (*Macropus giganteus*)
Eastern quoll (*Dasyurus viverrinus*)
European red fox (*Vulpes vulpes*)
Feathertail glider (*Acrobates pygmaeus*)
Feral rats *Rattus* sp.
Gilbert's potoroo (*Potorous gilbertii*)
Greater glider (*Petauroides volans*)
Grey-headed flying fox (*Pteropus poliocephalus*)
Herbert River ringtail possum (*Pseudochirulus herbertensis*)
Humans (*Homo sapiens*)
Koala (*Phascolarctos cinereus*)
Leadbeater's possum (*Gymnobelideus leadbeateri*)
Little red flying fox (*Pteropus scapulatus*)
Long-footed potoroo (*Potorous longipes*)
Long-nosed bandicoot (*Perameles nasuta*)
Mountain brush-tailed possum (*Trichosurus cunninghami*)
Mountain pygmy possum (*Burramys parvus*)
Northern brown bandicoot (*Isoodon macrourus*)
Northern quoll (*Dasyurus hallucatus*)
Numbat (*Myrmecobius fasciatus*)
Quokka (*Setonix brachyurus*)
Red kangaroo (*Osphranter rufus*)
Red-necked wallaby (*Macropus rufogriseus*)
Rufous bettong (*Aepyprymnus rufescens*)
Short-beaked echidna (*Tachyglossus aculeatus*)
Southern bent-wing bat (*Miniopterus orianae bassanii*)
Southern brown bandicoot (*Isoodon obesulus*)
Southern hairy-nosed wombat (*Lasiorhinus latifrons*)
Spotted-tailed quoll (tiger quoll, *Dasyurus maculatus*)
Stick nest rat (*Leporillus apicalis*)

Sugar glider (*Petaurus breviceps*)
Swamp antechinus (*Antechinus minimus*)
Swamp wallaby (*Wallabia bicolor*)
Tammar wallaby (*Macropus eugenii*)
Tasmanian devil (*Sarcophilus harrisii*)
Tasmanian pademelon (*Thylogale billardierii*)
Tiger quoll (Spotted-tailed quoll, *Dasyurus maculatus*)
Western grey kangaroo (*Macropus fuliginosus*)
Woylie (Brush-tailed bettong - *Bettongia penicillata*)
Yellow-bellied glider (*Petaurus australis*)

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