Common Diseases of Urban Wildlife

MAMMALS

The Australian Registry of Wildlife Health

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TARONGA ZOO | WESTERN PLAINS ZOO
Zoological Parks Board of New South Wales

1 Common Diseases of the Common Wombat

1.1 Introduction

The common wombat (*Vombatus ursinus*) primarily lives within borrows located near the margins of forest and scrubland. Common wombats are fairly solitary, herbivorous and nocturnal animals. The nocturnal habits of wombats place them at risk of motor vehicle associated trauma. Wombat pouch young often survive the traumatic injury and are hand raised by wildlife rehabilitators. These hand raised sub-adult wombats are commonly presented to urban veterinarians for medical care.

1.2 Parasitic Disease

1.2.1 Sarcoptic Mange

Aetiology

Sarcoptes scabiei var wombati is a mite of uncertain host specificity. There are reports of transmission of sarcoptic mange from wombats to humans and dogs (Booth, 1994, Skerratt and Beveridge, 1999). Persons handling infested animals should take precautions.

Native animals reported to have suffered sarcoptic mange include the common wombat, koalas, agile wallabies and the common ringtail possum. Intermittent outbreaks of infestation occur within the southern hairy-nosed wombat, but the parasite is not reported to occur in the endangered northern-hairy nosed wombat (Skerratt et al., 1998).

The common wombat is known to suffer severe debilitation as a result of *S. scabiei* infestation. Sarcoptic mange occurs throughout the range of the common wombat, including Tasmania and Flinder's Island. Outbreaks of severely debilitating sarcoptic mange occur only sporadically; nonetheless, these outbreaks may pose a risk to the long-term survival of some of the smaller sub-populations of the common wombat (Martin et al., 1998).

S. scabiei is transmitted during direct contact between animals and through sharing contaminated rubbing sites and burrows. It is an obligate skin parasite and does not live longer than a few weeks in the environment. It is uncertain whether the mites responsible for sarcoptic mange in wombats were introduced to Australia with dingo, or with the European red fox (Skerratt et. al, 1998).

Clinical signs

Sarcoptic mites burrow into the keratinising layers of the epidermis causing pruritis, hyperkeratosis, epidermal thickening, and alopecia. 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 or invasion of opportunistic bacteria.


Sarcoptic mange, common wombat

The severe and extensive nature of skin lesions in some wombats can lead to impaired vision, disturbance in mastication, emaciation and abnormal activity throughout the day (Booth, 1994). Death may result from secondary infection, dehydration or starvation.

Pathogenesis

It has been proposed that wombats exhibit marked skin lesions in response to low numbers of *Sarcoptic scabiei* mites due to hypersensitivity reaction (Munday, 1988; Skerratt, 2003). Systemic effects can also be severe. 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. These severely infested wombats may

have anaemia, leucocytosis, lymphopaenia, and a reduced serum protein concentration (Skerratt, 1989). The most commonly infested locations included the anterior lateral aspect of the body, the posterior lateral surface of the body, and occasionally the ventral and dorsal midline. Lymphadenitis is commonly encountered in the peripheral lymph nodes that drain severely infested portions of the skin (Skerratt, 1998, Skerratt, 2003a and b). Sarcoptic mange in wombats may be associated with overcrowding, habitat degradation, and a high density of foxes or feral dogs (Booth, 1998).

Diagnosis

Diagnosis of sarcoptic mange relies on identifying the mite within skin scrapings or samples of the hyperkeratotic crust.

Treatment

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 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). Additional information on treatment is described by Skerratt (2003b).

1.2.2 Coccidiosis

Aetiology

Eimeria arundeli is a frequently encountered enteric coccidian of the common wombat. This parasite has been associated with enteritis in sub-adult wild and hand-reared wombats. The life cycle of the coccidian parasites of wombats has not been thoroughly investigated, but it is thought to be similar to other eimerian parasites.

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Clinical signs

Oocysts of *E. arundeli* are often found within the faeces of healthy adult common wombats. Wild and captive sub-adult wombats appear to be the most likely animals to exhibit clinical signs of enteritis associated with coccidial infection. Wombats with a severe burden of coccidia may develop mucoid to liquid green diarrhoea, progressively loose weight and become bloated. The onset 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.

Pathology

Gross post mortem examination 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.

Wombats that die with intestinal coccidiosis often have concurrent salmonellosis (ARWH).

Diagnoses

Diagnosis of coccidiosis is achieved through examination of faeces within a wet preparation using light microscopy at a magnification of 400x. Standard faecal flotation techniques are also useful in the diagnosis of coccidiosis.

Treatment

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.

1.2.3 Other Parasites

Toxoplasmosis can cause mortality in hand-raised wombats. This disease is discussed in detail in section 5.2.1

Sheep liver fluke infection (*Fasciola hepatica*) also occurs commonly in wombats. Although one often does not see adult flukes or not large numbers of them, the immunological response of the wombat to the fluke is such that the entire liver may become completely fibrosed. (David Spratt, personal communication).

1.3 Fungal Disease

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

1.4 Nutritional Disease

Captive wombats offered energy or carbohydrate-rich diets, such as lucerne and fruit, can become bloated. Bloated wombats have a distended abdomen, abdominal pain, and flatulence. Diet modification and short-term use of agents such as Milanta® can be effective remedies for these wombats.

Wombats 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 wombats; however, repeated tooth clipping is often required.

A syndrome of multisystemic mineralisation has historically been reported in captive common wombats. 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 cases of multisystemic mineralisation in wombats are present within the Australian Registry of Wildlife Health at Taronga Zoo (ARWH). Affected animals had been 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 is the most likely cause of this syndrome.

1.5 Traumatic Injury

Wombats that are injured by motor vehicles tend to suffer spinal injuries, pelvic fractures, concussion, or massive contusions.

2 Common Diseases of the Short-beaked Echidna

2.1 Introduction

The short-beaked echidna (*Tachyglossus aculeatus*) is an insectivorous monotreme that inhabits virtually every habitat-type throughout Australia. Short-beaked echidna have crepuscular habits in summer and are diurnal during winter. The normal body temperature for the short-beaked echidna ranges between 27 and 32^{o}C. Whenever environment temperatures fall below 10^{o}C and food availability declines, short-beaked

echidna will dig into the substrate and begin hibernation. During hibernation, the animal's body temperature may fall to as low as 5^{o}C (Whittington, 1993).

2.2 Parasitic Disease

2.2.1 Coccidiosis

Aetiology

Eimeria echidna, E. tachyglossi, and *Octosporella hystrix* are enteric coccidia of the short-beaked echidna (Barker et al., 1985). Coccidial oocysts are a frequent finding within the faeces of healthy echidna; however, coccidia are occasionally associated with marked enteritis or disseminated disease.

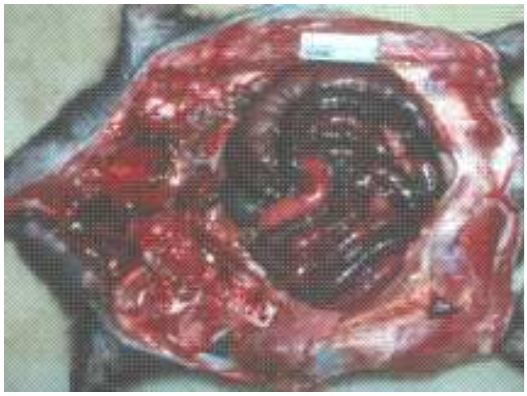
Clinical signs

Short-beaked echidna with coccidial enteritis or disseminated coccidiosis are most often found dead without premonitory clinical signs.

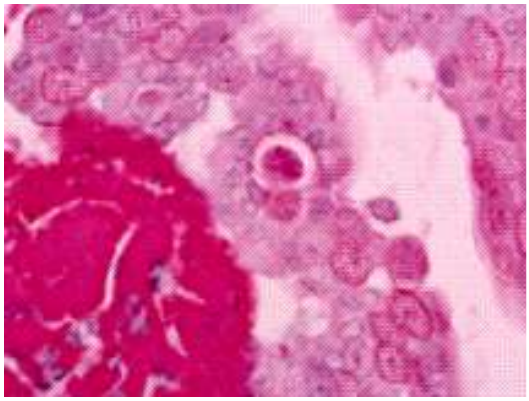
Pathology

Enteric coccidiosis:

Short-beaked echidna with enteric coccidiosis may have haemorrhagic and necrotising enteritis. The intestinal mucosa is often thickened due to the presence of large numbers of coccidial microgametocytes, macrogametocytes and oocysts. Villous atrophy has been reported in some animals with enteric coccidiosis. An intense mononuclear cell infiltration is usually evident throughout the intestinal lamina propria of echidna with coccidial enteritis.



Haemorrhagic enteritis caused by disseminated coccidiosis, short-beaked echidna.

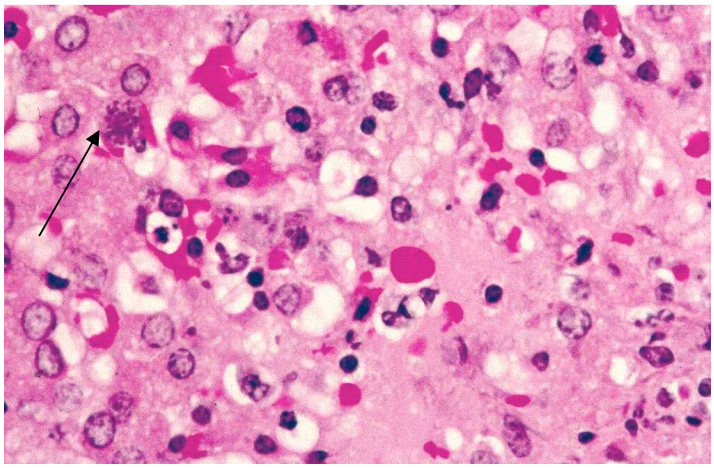


Intestinal coccidiosis, small intestine, short-beaked echidna. Note formation of oocysts with eosinophilic

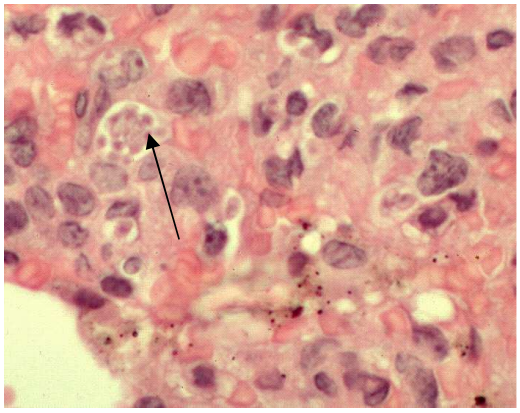
amylopectin bodies.

Disseminated coccidiosis:

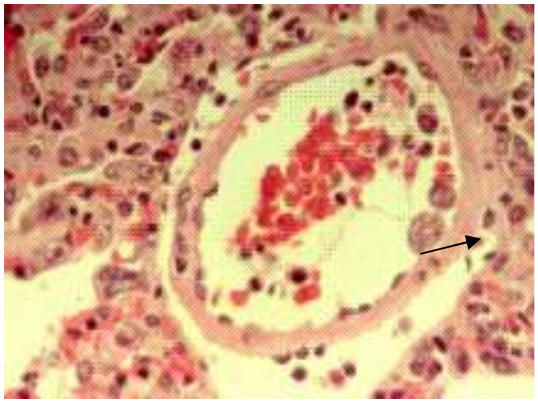
Echidna with disseminated coccidiosis also have microgametocytes, macrogametocytes and oocysts, within host cells of the intestinal mucosa and lamina propria. Desquamation of the superficial enterocytes, hypertrophy of the villous epithelium, and a mononuclear cell infiltrate throughout the lamina propria are also evident within affected segments of the intestine (Dubey and Hartley, 1993). In addition, the disseminated form of coccidiosis is characterised by asexual and sexual stages of the coccidian lifecycle scattered throughout the endothelium and parenchyma of the lung, liver, heart, kidney, spleen and gastrointestinal tract (Dubey and Hartley, 1993).



Hepatic necrosis, disseminated coccidiosis (see arrow).



Coccidial schizogeny (arrow), lung, short-beaked echidna.



Coccidial schizogeny (arrow) in the endothelium of pulmonary blood vessel, short-beaked echidna.

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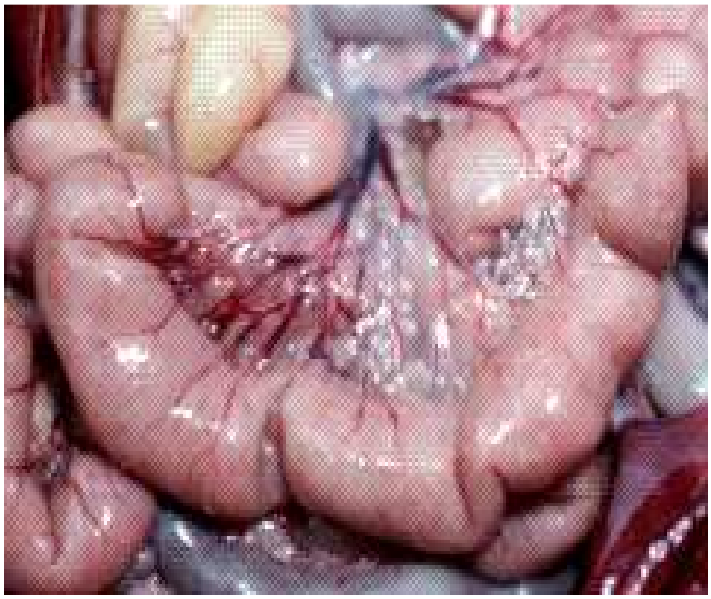
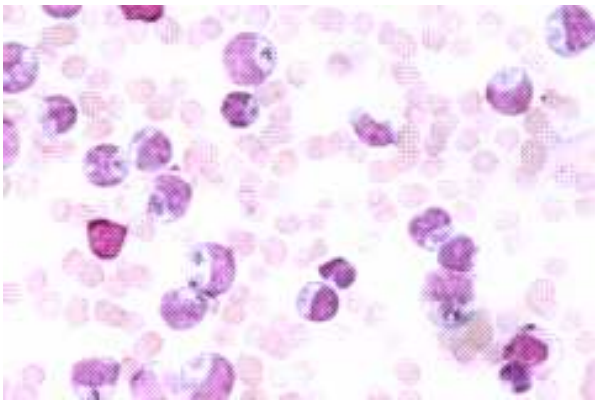


Mesenteric lymph nodes, short-beaked echidna

Monotremes have a diffuse lymphoid system composed of nodules, which 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 had 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.



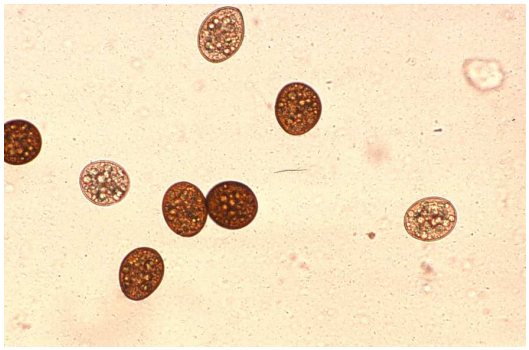
Systemic coccidiosis, short-beaked echidna (blood smear). Note organisms within mononuclear cells

Several short-beaked echidna with multisystemic coccidiosis have had *Atoxoplasma*-like blood parasites circulating within monocytes. Examination of blood films from short-beaked echidna can be used to diagnose clinical systemic coccidiosis. Many echidna have very small number of circulating organisms, but large numbers of organisms evident in monocytes is associated with clinical disease (ARWH).

Echidna that die with systemic coccidiosis often also have acute *Salmonella* species septicaemia (ARWH).

Diagnosis

Diagnosis of coccidiosis is achieved through examination of faeces within a wet preparation using light microscopy at a magnification of 400x. Standard faecal flotation techniques are also useful in the diagnosis of coccidiosis. 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.



Coccidial oocysts, faeces, short beaked echidna, 400x

Treatment

Short-beaked echidna with coccidial enteritis or systemic coccidiosis are most commonly presented moribund or dead. Treatment methods have not been thoroughly investigated.

2.2.2 Other Parasites

Short-beaked echidna are commonly infested with ticks. *Aponomma concolor* is the "echidna tick", which infests both captive and wild short-beaked echidna. Large tick burdens can result in anaemia and dermatitis. Ticks are also capable of transmitting a number of haemoprotozoa and viruses.

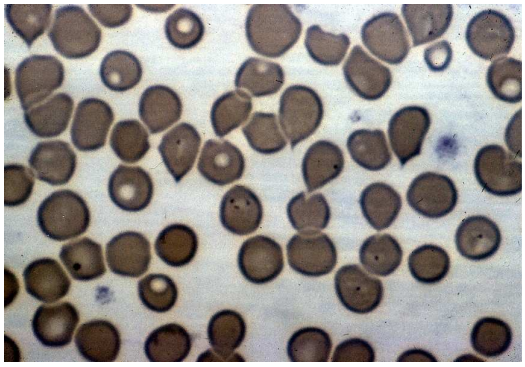
Sparganosis due to the plerocercoid *Spirometra eranacei* has been reported to cause large tumour-like masses within the subcutaneous tissues of the ventral and lateral abdomen of short-beaked echidna. The masses can be as large as 12 cm diameter and are composed of a central sparganum, surrounded by an intense non-suppurative inflammatory infiltrate, and a thick layer of fibrous tissue. Several infected echidna have had numerous plerocercoids replacing a large proportion of the pulmonary parenchyma (Whittington et al., 1992, ARWH). Presumably, the echidna ingest

infected copepods and act as incidental intermediate hosts. The definitive hosts of *Spirometra eranacei* are introduced felids and canids.

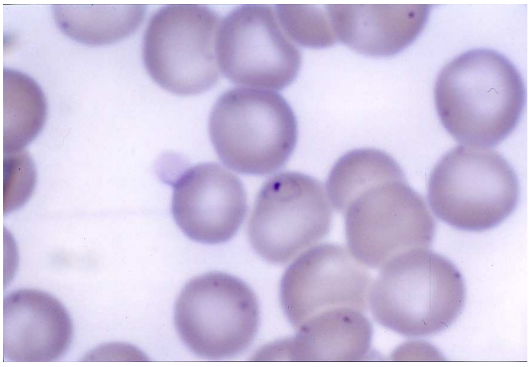
A wide variety of nematodes parasitise the gastrointestinal tract, pulmonary tissues, and subcutaneous tissues of the short-beaked echidna, including: *Parastrongyloides* spp., *Nicollina* spp., *Tachynema* spp., *Tasmanema* spp, *Ophidascaris* sp., and *Dipetalonema* sp. Clinical signs are rarely associated with nematode parasitism.

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 and in which upwards of 500 individuals can occur in a single host (David Spratt, personal communication).

Theileria tachyglossi, and *Babesia tachyglossi* are common incidental haemoprotozoa of the short-beaked echidna. An *Anaplasma marginale*-like organism has also been identified within circulating red blood cells of normal echidna (Whittington, 1993).



Theileria tachyglossi in Howell-Jolly body



Babesia tachyglossi, short-beaked echidna

2.3 Bacterial Disease

Many species of *Salmonella* have been isolated within healthy short-beaked echidna, yet several species of *Salmonella* have been identified as the cause of haemorrhagic enteritis and septicaemia.

Multisystemic mycobacteriosis is a fairly common post mortem finding in short-beaked echidna. Granulomatous lesions occur in a variety of tissues, but are most

common in the liver. Beaded, red bacilli are easily demonstrated within the granulomas upon acid fast staining.

Staphyloccocus spp., *Streptococcus spp, Aeromonas spp.*, and *Proteus spp*. have been associated with septicaemia in the short-beaked echidna. *Edwardsiella sp.* has been isolated from the pulmonary tissue of echidna suffering from suppurative bronchopneumonia. *Edwardsiella* sp. may be a primary pathogen of the short-beaked echidna (Whittington, 1993).

2.4 Viral Disease

Multisystemic herpes virus infection and inclusion body hepatitis associated with herpes virus have been reported in short-beaked echidna (Whittington, 1992).

Poxvirus is occasionally found within proliferative dermal lesions in juvenile short-beaked echidna. It is uncertain whether poxvirus infection is the cause of a fairly common seborrheic condition of hand reared juvenile short-beaked echidna (Whittington, 1993).

Cytomegalo-like adenovirus inclusion bodies have been identified within the renal epithelium of short-beaked echidna, but were considered to be an incidental finding (Whittington, 1993).

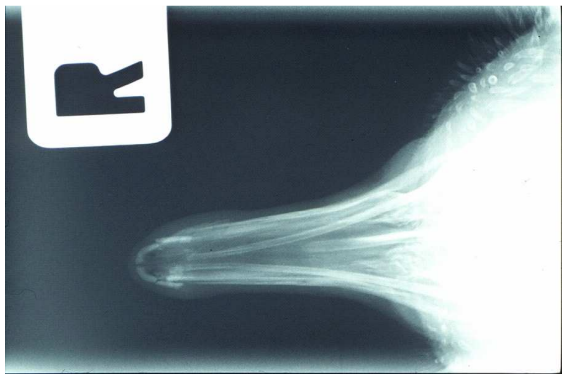
2.5 Fungal Disease

Oesophagitis and gastritis are frequently diagnosed in short-beaked echidna 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. *Corynebacterium, Candida* and *Fusobacterium* spp. have been isolated from these ulcerative lesions (Whittington, 1993). Among short-beaked echidna presented to Taronga Zoo for post mortem examination (ARWP), *Candida albicans*, or one of its telomorphs, is most commonly identified in microbial culture, and during histologic examination of these lesions.

Microsporum gypseum has been isolated from short-beaked echidna with fractured quills and *Cryptococcus neoformans* has been isolated within pulmonary granulomas of the short-beaked echidna.

2.6 Traumatic Injury

The oronasal structure of the short-beaked echidna is a sensitive and fragile organ. Unfortunately, the bones of the beak often sustain comminuted 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 is often indicated for severe damage.



Fractured beak, *Tachyglossus aculeatus*

External examination is not a sensitive tool for the detection of traumatic injuries in short-beaked echidna. Radiography is warranted when any echidna presents with evidence of traumatic injury, since the presence of cutaneous spines prevent thorough palpation of the skeletal system.

3 Common Diseases of Possums

3.1 Introduction

The common ringtail possum (*Pseudocheirus peregrinus*) and the common brushtail possum (*Trichosurus vulpecula*) are widespread throughout the east coast of Australia and southwest Australia. These herbivorous marsupials have nocturnal habits and nest in tree hollows, logs, and roof spaces. While brushtail possums are solitary and territorial, ringtail possums often have overlapping home ranges.

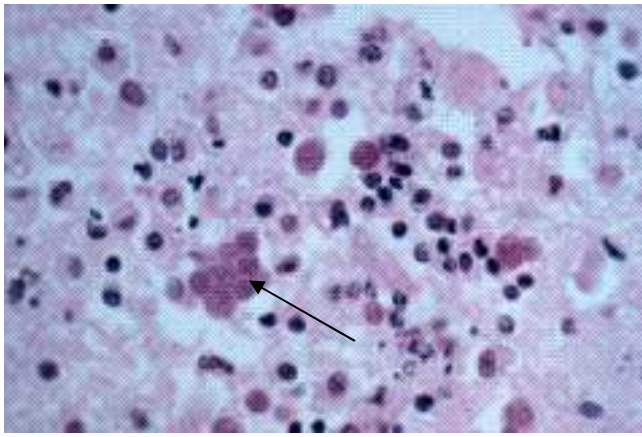
*3.2 **Parasitic Disease***

3.2.1 Toxoplasmosis

Aetiology

Toxoplasma gondii is a coccidian parasite with worldwide distribution and a broad host range. All vertebrates may be infected with the asexual stages of reproduction; however, some taxonomic groups are more susceptible to suffering 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 felids, 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. Felids may shed millions of oocysts in their faeces during their first infection with *T. gondii*. Felids are usually infected when they eat birds or rodents infected with *T. gondii* tissue cysts.



Toxoplasma gondii. (see arrow)

Marsupials are primarily infected with *T. gondii* when they ingest vegetation contaminated with felid faecal material containing sporulated oocysts; however, transplacental transmission of the parasite has been reported. Sporozoites are released from ingested oocysts and replicate in the tissues of the intestine and associated lymphoid tissue. The organisms are referred to as tachyzoites as they undergo several generations of rapid replication, before spreading through the circulatory system to numerous tissues and forming cysts. Cysts, which contain bradyzoites, 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.

Both ringtail and brushtail possums share their habitat with pet and feral cats, and as a consequence toxoplasmosis is not uncommon in these species.

Clinical signs

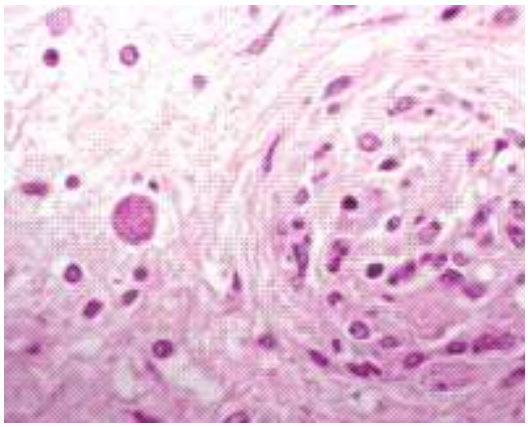
Toxoplasma gondii infection is often inapparent. Clinical illness associate with *T. gondii* occurs primarily in animals that are immunosuppressed or hand raised.

Clinical signs of toxoplasmosis are primarily associated with lesions in the central nervous system, lungs, and liver. The severity of the illness is highly variable, ranging from mild malaise to peracute mortality. Animals clinically affected by toxoplasmosis may be depressed, weak, anorexic, pyrexic, dyspnoeic, ataxic, hemiplegic, quadriplegic, comatose, convulsive, or may exhibit muscle stiffness, diarrhoea, emesis, uveitis, retinitis 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.

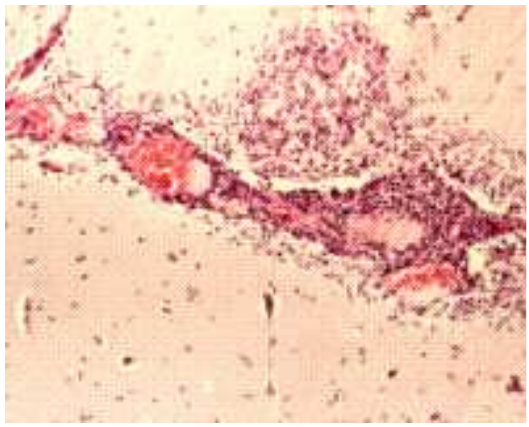
Serum concentrations of AST, CK and alanine amino transferase (ALT) are often elevated in animals suffering from toxoplasmosis but this depends upon the organs affected.

Pathology

Non-suppurative inflammation and necrosis are the most prevalent morphologic changes associated with active *T. gondii* infections. Although bradyzoites can remain dormant with little or no host inflammatory response, tachyzoites of *T. gondii* are highly immunogenic, inciting a strong humeral and cell mediated immune response in the host. Lymphocyte, plasma cell and macrophage infiltration is commonly associated with the presence of trophozoites. The cause of tissue necrosis associated with *T. gondii* trophozoites is uncertain (Canfield et al., 1990, Dubey and Hartley, 1991).



Toxoplasma – subclinical infection, brain, wombat.



Toxoplasma encephalitis, wombat

Gross post mortem lesions may be inapparent, however, pulmonary congestion, oedema, consolidation, hepatosplenomegaly, lymphadenopathy, or multisystemic pale foci may be evident. Histologic examination of tissues from clinically affected animals may reveal any of the following lesions: retinitis, lymphadenitis, hepatitis, myositis, pneumonia, pancreatitis, myelitis and encephalitis.

Individual tissue cysts are commonly found within the muscle and nervous tissue of animals during histologic examination. Unless these cysts are associated with tissue necrosis or inflammation, they most likely represent subclinical infection.

Diagnosis

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. Most commercial veterinary labs offer indirect haemagglutination inhibition tests for the detection of *T. gondii* IgG. Indirect fluorescent antibody tests may be used to determine serum IgM concentrations.

Post mortem diagnosis of toxoplasmosis usually occurs through histopathologic examination of tissues, or cytological examination of impression smears of the spleen, liver, or lung. Immunoperoxidase staining, tissue antigen ELISA, or PCR are used to

detect small numbers of *T. gondii* organisms in section, and differentiate *T. gondii* from other protozoa.

Treatment

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 affect 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.

Prevention

Toxoplasmosis prevention relies on decreasing the opportunity for immunologically naïve or otherwise susceptible hosts to be exposed to large numbers of infective oocysts. 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). An oral vaccine consisting of *Hammondia hammondi,* has effectively prevented the development of clinical disease in Tammar wallabies challenged with *T. gondii* (Reddacliff, 1993*). Hammondia hammondi* is a non-pathogenic coccidian parasite of cats that is very similar to *T. gondii*. Clinical trails of vaccines using temperature sensitive strains of *T. gondii* are under investigation. Additionally, the management of stressors, or other immunosuppressive factors, are important in reducing the expression of clinical disease.

3.2.2 *Angiostrongylus cantonensis*

Aetiology

Angiostongylus cantonensis is a metastrongylid nematode that has for some time been a cause of eosinophilic meningoencephalitis in mammals and birds in north-eastern Australia. It is extending its range along the central to south eastern seaboard and cases are becoming common in Sydney.

Parasitic meningoencephalitis due to infection by the metastrongylid nematode, *Angiostrongylus cantonensis*, has been reported in ringtail possums, brushtail possums, primates, flying foxes, yellow tailed black cockatoos, and tawny frogmouths in Queensland and New South Wales. An outbreak of disease was also reported in captive rufous bettongs in Queensland (Higgins et al., 1997). *A. cantonensis* can cause central nervous system disorder and death in wild and captive native fauna. This parasite was originally thought to be geographically restricted to Queensland, but recent reports of the infection in dogs, zoo and wild animals indicate that the parasite is now widespread in New South Wales (Carlisle et al., 1998, Collins and Rothwell, 1992, Reddacliff et al., 1999). *A. cantonensis* is a zoonotic threat. Eosinophilic encephalomyelitis has been reported to occur within children that ingest snails or slugs (Paul Prociv, personal communication).

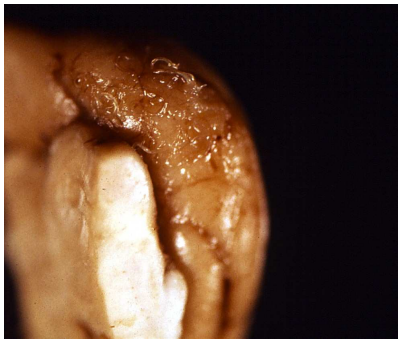
Rats are the definitive host of *A. cantonensis*. Adult parasites live within the pulmonary arteries and release eggs that are trapped in the pulmonary microvasculature. Larvae emerge from the eggs and migrate into the airways and then to the intestinal tract. Rats excrete first-stage larvae in their faeces, and these larvae either directly penetrate, or are ingested by terrestrial snails or slugs. Larvae develop into the infective third-stage in the molluscs, which are then eaten by rats. When ingested by rats, infective larvae undertake a prolonged migration through the central nervous tissue before they mature to adulthood in the pulmonary artery. In non-adapted hosts, the parasite often undergoes ongoing migration through the central nervous tissues, and rarely matures and migrates to the pulmonary arteries (Carlisle et al., 1998).

Clinical signs

Animals suffering from *A. cantonensis* meningoencephalitis may exhibit a variety of clinical signs related to focal lesions within 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.

Pathology

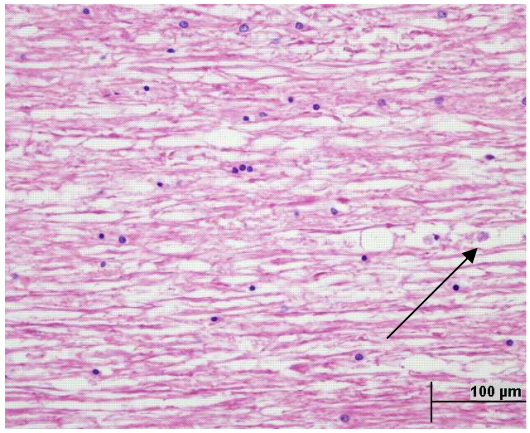
Gross post mortem examination is often unrewarding in animals with eosinophilic meningoencephalitis. Occasionally nematode parasites are visible within the subarachnoid spaces.



A. cantonensis within the meninges

Nematode parasites are evident upon histologic examination of serial sections of the brain and spinal cord. Parasites may be unaccompanied by an inflammatory response, but are often associated with a mixed inflammatory cell infiltrate. This infiltrate is composed of lymphocytes, macrophages, plasma cells and scattered eosinophils. Foci of malacia may occur when parasite migration has resulted in ischaemic tissue necrosis.

Cross-sections of A. cantonensis , spinal cord, brushtail possum.



Wallerian degeneration, spinal cord, brushtail possum. Note axonal swelling and digestion chambers(phagocytic cells within axonal chambers - arrow)

Diagnosis

Ante mortem diagnosis of *A. cantonensis* is often difficult. Eosinophilia within the cerebrospinal fluid is highly suggestive of parasitic meningoencephalitis; however, infected animals often only show non-suppurative inflammation, which does not assist in the differentiation from viral or protozoal infections (ARWH). Infected animals rarely have systemic eosinophilia. ELISA has been used to detect infection in humans and dogs, and preliminary work has been carried out to develop an ELISA for use in marsupials (Smaller, 2004).

Treatment

Corticosteroids are employed in the treatment of angiostrongyliasis to reduce inflammation within the central nervous system. Some researchers suggest that the use of antiparasitic drugs can result in increased inflammation throughout the central nervous system as a result of massive release of antigen from degenerating parasites. Fenbendazole, has been used to successfully treat *A. cantonensis* infection in a grey-headed flying-fox (Reddacliff et al., 1999). The prognosis for infected animals, however, is generally poor.

Prevention

Prevention of eosinophilic meningoencephalitis caused by *A. cantonensis* relies on eradicating intermediate hosts from the enclosures of captive mammals.

3.2.3 Other Parasites

Ectoparasites

The paralysis tick, *Ixodes holocyclus*, is commonly found in the pelage of brushtail possums. Anecdotal reports have described tick paralysis in brushtail possums.

Notoedres muris, the mange mite of rats, has been associated with thick crusty dermatitis of the ears, tail, snout and periocular tissues of brushtail possums in Victoria (Booth, 1994b).

Cestodes

Bertiella trichosuri is an anoplocephalid cestode that parasitises the intestinal tract of brushtail possums. Many possums are infected with this tapeworm, 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, 1994b).

Trematodes

Fasciola hepatica is the liver fluke of sheep. This parasite enjoys a wide host range, and has been known to infect brushtail possums grazing on wet pastures. Snails function as the intermediate host for this parasite.

Nematodes

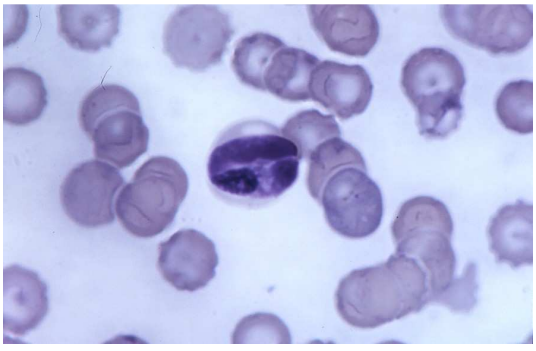
A variety of nematode parasites infect the intestinal tract of possums; however, parasitism rarely results in clinical disease.

Marsupiostrongylus minesi is most often an incidental lungworm found in possums and gliders. On occasion, this parasite may occur in the airways and causes pulmonary consolidation or bronchopneumonia (Booth, 1994b).

Brushtail possums grazing pastures inhabited by sheep may develop diarrhoea, dehydration and eventually die as a result of heavy intestinal burdens of *Trichostrongylus axei* and *T. colubriformis.*

Protozoa

A variety of protozoa are observed within the faeces or intestinal lumen of brushtail possums and ringtail possums. Enteritis associated with the presence of these parasites has not been described.



Hepatozoon spp, ringtail possum

Hepatozoon spp are occasionally found in the erythrocytes of possums and gliders. Their clinical significance is unknown.

3.3 Bacterial Disease

3.3.1 Yersiniosis

Aetiology

Yersinia pseudotuberculosis is a ubiquitous gram-negative bacterium of the family Enterobactericeae that is commonly identified within the faeces of wild birds and mammals. Transmission of this bacterium occurs via the faecal-oral route.

Clinical signs

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 melaena. 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 or immunosuppression.

Pathology

An outbreak of yersiniosis in captive brushtail possums occurred in Melbourne Zoo in 1987. The incidence of disease was between 13 and 15 percent in this population, and seven deaths were recorded (*Y. pseudotuberculosis* was isolated in 6 of 7 animals). Gross post mortem examination conducted on these animal revealed multifocal abscesses throughout the liver, spleen, and kidneys (Booth, 1994b).

Enteritis associated with yersiniosis is evident as foci of mucosal necrosis. Histological examination of affected portions of the small intestine confirms the mucosal necrosis and reveals mats of bacterial colonies along the mucosal surface and within the necrotic debris. Animals with the more chronic form of yersiniosis 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

Diagnosis of yersiniosis is achieved by isolating the organism within lesions. *Y. pseudotuberculosis* can be difficult to isolate in microbial culture, however, cooling tissue samples briefly may increase the likelihood of isolating the organism.

Treatment

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.

Prevention

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 experienced by captive wildlife may assist in the prevention of yersiniosis.

3.3.2 Salmonellosis

Many *Salmonella* species have been isolated from the tissues or faeces of wild and captive possums. Rarely have *Salmonella* spp. been responsible for disease in wild possums. Salmonellosis is primarily a disease of captive possums, and disease often occurs in hand raised animals or those animals subject to stressful events. Outbreaks of samonellosis have been recorded in young ringtail possums being reared in aviaries (ARWH). Rapidly fatal, haemorrhagic enteritis and septicaemia are the manifestations of salmonellosis in young possums. Foci of hepatic necrosis and paratyphoid nodule formation are reported in possums (Booth, 1994b).

3.3.3 E. coli

Escherichia coli infection is not uncommon in captive ringtail possums and the findings are as described above for salmonellosis. A single wild ringtail possum has

had severe granulomatous lymphadenitis associated with *E. coli* infection (coligranulomas - ARWH).

3.3.4 Leptospirosis

Brushtail possums throughout New South Wales, Victoria, and the North Island of New Zealand are often infected with *Leptospira interrogans balcanica*. This organism does not appear to be host specific, and can infect sheep and cattle. Infection in these species, however, is most often inapparent. Transmission between possums occurs primarily via urine; however, sexual transmission has also been reported. This organism is most often responsible for mild, transient clinical signs of malaise in possums.

3.3.5 Mycobacteriosis

Mycobacteriosis has not been identified in free-living possums in Australia. Since 1967, feral brushtail possums in New Zealand have been a reservoir for *Mycobacterium bovis* in areas where the cattle and possums share habitat at the forest/pasture margin.

3.3.6 Tyzzer's Disease

Aetiology

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 and dasyurids, but it most often occurs as a fatal necrotising hepatitis and myocarditis in young possums (Canfield and 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.

Clinical signs

Most possums will die acutely or exhibit non-specific signs of illness 24 to 48 hours before death (Canfield and Hartley, 1991). Clinical signs of illness in laboratory animals may include diarrhoea, jaundice, and elevated CK, AST, and ALT.

Pathology

Pale foci throughout the hepatic parenchyma and myocardium are often visible upon gross post mortem examination of animals suffering from Tyzzer's disease. Microscopic examination of the hepatic and myocardial lesions reveals foci of coagulation necrosis. When lesions are more chronic, neutrophils may be scattered throughout the necrotic tissue. Slender bacilli may be evident packeted within hepatocyte cytoplasm at the margins of the necrotic foci. Silver stains render the organisms more visible using light microscopy

Diagnosis

Presumptive diagnosis of Tyzzer's disease is based upon visualising intracytoplasmic bacilli within hepatocytes or cardiac myocytes adjacent to necrotic foci. Definitive diagnosis, however, relies on isolation of *B. piliformis* from the lesions.

Prevention

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.

3.4 Nutritional Disease

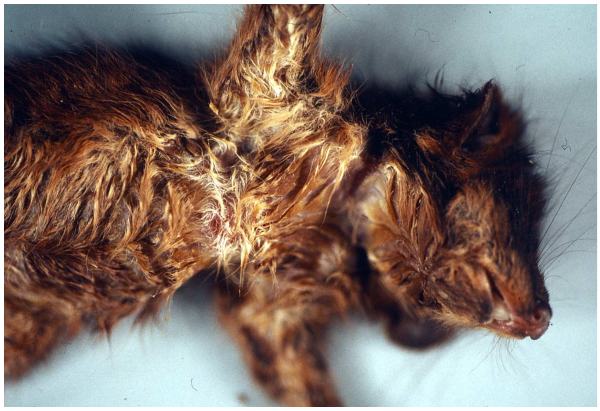
Occasionally hand raised possums are presented with concurrent enteritis and bilateral cataracts. These lesions are highly suggestive that the animal has been fed cow's milk or some other milk substitute containing high concentrations of lactose and galactose. The milk of many marsupials contains scant lactose, and the intestinal tract of young marsupials contains very low lactase activity (Jenness et al., 1964). Diarrhoea occurs as a result of the osmotic effect of undigested lactose 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.

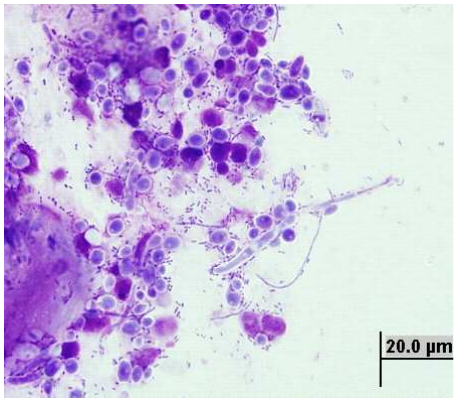
3.5 Fungal Disease

Ringtail possums being hand reared can be susceptible to overgrowth of *Candida* sp. yeast within their gastrointestinal tracts. A small number of wild, young ringtail possums have also been found with a moist dermatitis caused by *Candida* sp.

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. 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.



Young, wild ringtail possum with moist dermatitis, Candida albicans



Budding yeasts and hyphae. *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. 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. *Candida* sp can also be identified through standard fungal culture techniques.

3.6 Traumatic Injury

Possums are subject to a variety of traumatic injuries. Vehicular trauma often results in multisystemic injuries. Ringtail possums and young brushtail possums are also highly susceptible to cat bite injuries.

Ringtail 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.

Brushtail possums that sustain severe traumatic injury frequently have hypersecretion of fluids into the stomach. Subacute painful injuries in this species can be accompanied by gastric ulceration. Ringtail or brushtail possums that are trapped within the confines of a building will often be found emaciated, with scant intestinal content, and severe gastric ulceration (ARWH).

3.7 Diseases of Uncertain Aetiology

3.7.1 Swollen Paw Syndrome

Clinical syndrome

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 (SPS).

Ringtail possums with SPS most often originate from the northern suburbs of Sydney and they are discovered throughout the year. SPS affects sub-adult and adult possums of both sexes. This is primarily a disease of wild ringtail possums, but SPS occasionally develops in captive possums.

Animals with SPS are bright, alert and continue to eat and drink. Clinical signs of SPS begin with oedema of the front paws. Animals with more advanced disease exhibit many of the following lesions:

Swollen paw syndrome

- swollen and ulcerated paws,
- moist or ulcerative dermatitis of the bridge of the nose,
- alopecia, curling and necrosis of the tips of the pinna, 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 within deep ulceration, primarily in the paws of the hind limbs. Some animals with SPS will have complete avascular necrosis of the paws. Ringtail possums are occasionally observed in the wild missing one or more of the distal extremities. 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.

Pathology

Ringtail possums affected by SPS 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.

Microscopic examination of the tail and feet of possums with SPS reveal vast areas of coagulation necrosis, with variable degrees of epidermal ulceration. The dermis is usually markedly oedematous and contains scattered fibrin. Inflammatory infiltrates consisting of a mixture of lymphoid cells and neutrophils are present within the dermis underlying foci of ulceration. Cocci are evident within the superficial necrotic material covering the ulcerated epidermis. *Staphylococcus aureus* is the most common organism isolated from wound sites. *Aeromonas sobria, E. coli, Klebsiella pneumoniae,* and *Alcaligenes faecalis* have also been isolated from swabs of these ulcerative lesions.

The pathogenesis of this disease is poorly understood. Microcirculatory compromise or photosensitization are the most plausible processes responsible for such acute, severe necrosis of the extremities. Although the pattern of skin lesions and pathological findings are reasonably consistent with photosensitization, it would be highly unusual for this disease to occur in a nocturnal animal. Additional aetiologic agents that have been considered during investigation of SPS include bacterial or viral infection, thermal injury, electrocution, mycotoxicosis (ergot poisoning), and toxicosis from plant derived alkyloids (Henry Collins, personal communication, ARWH). Elucidation of the aetiologic agent responsible for SPS is likely to be a difficult task. Based on the character of the lesions, exposure to the inciting factor occurs a significant time prior to the animal's presentation with clinical signs.

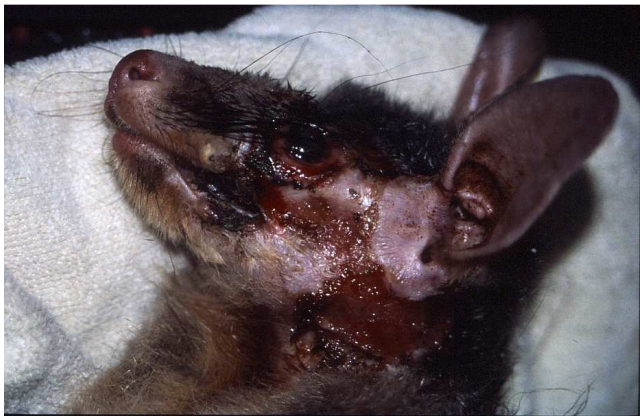
Treatment

An effective treatment regime has not yet been established.

3.7.2 Exudative Dermatitis

Clinical syndrome

Severe and extensive exudative dermatitis is a common finding in brushtail possums



Exudative dermatitis, brushtail possum

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. 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 (Munday, 1988, Reddacliff, 1981).

The haemogram of possums with exudative dermatitis often demonstrates non-regenerative anaemia, leukocytosis, neutrophilia and lymphopaenia (Hemsley and Canfield, 1994).

Exudative dermatitis, brushtail possum.

Pathology and pathogenesis

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.

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 secondarily infected (Reddacliff, 1988). A wide variety of bacteria have been isolated within the exudative wounds of brushtail possums: Coliforms, *Staphylococcus* spp, and *Streptococcus* spp. are among those most commonly identified (Reddacliff, 1981). 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. Disease may also be more common in sub-adult males, who disperse in search of their own territories.

Treatment

Administration of broad-spectrum antibiotics and topical wound therapy will usually result in clinical improvement of the skin lesions. 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.

3.7.3 Chronic meningoencephalitis

Clinical syndrome

Chronic meningoencephalitis and optic neuritis occur in brushtail possums in eastern Australia and Tasmania in a syndrome often referred to as Wobbly possum. A similar syndrome occurs in brushtail possums in New Zealand (O'Keefe et al 1997; Thompson et al 1999; Perrott et al 2000); however, it appears likely that different aetiologic agents are involved**.**

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, and ataxia 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 (Hartley et al., in preparation).

Between 1985 and 1993, 540 live brushtail possums were submitted to the wildlife clinic at Taronga Zoo. Thirty of these animals had clinical signs of depression and blindness. None of the animals recovered from their neurological defects to be suitable for release back to the wild. Tissues from these animals and an additional 12 brushtail possums from New South Wales, Victoria and Tasmania were examined histologically. Twenty three of the animals had chronic non-suppurative meningoencephalitis (Hartley et al., in preparation).

Pathology

Gross post mortem examination of animals with chronic meningoencephalitis does not reveal significant lesions.

Brushtail possums with chronic meningoencephalitis consistently have moderate to marked non-suppurative inflammation within the meninges and surrounding the blood vessels in the parenchyma of the brain. Wallerian degeneration and mild non-suppurative inflammation is observed in the optic tract of many possums with this syndrome. Possums with chronic meningoencephalitis may have atrophy of the cerebellar folia or retinal atrophy (Hartley et al., in preparation).

A viral agent is considered to be the most likely aetiology of chronic meningoencephalitis in brushtail possums in Australia, but as yet no aetiological agent has been identified.

Treatment

A variety of therapeutic efforts have been unsuccessful.

4 Common Diseases of Megachiroptera

4.1 Introduction

The grey-headed flying-fox (*Pteropus poliocephalus*), little red flying-fox (*Pteropus scapulatus*), and black flying-fox (*Pteropus alecto*) are occasionally presented to

wildlife care centres on Australia's east coast. These large frugivorous and insectivorous bats roost in trees during the day and become active at dusk.

4.2 ***Parasitic Disease***

4.2.1 *Angiostrongylus cantonensis*

Parasitic encephalitis has been documented in black flying-fox and little red flying-foxes in Queensland since 1992 (Reddacliff et al, 1999; Barrett et al, 2002). A single outbreak of parasitic encephalitis in captive grey-headed flying-foxes (GHFF) was reported in Sydney in 1997 (Reddacliff et al., 1999). The parasite associated with the neurological lesions was verified as *Angiostrongylus cantonensis*. The GHFF that suffered from parasitic encephalitis were hand raised sub-adult animals. Ringtail possums on the same property had 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).

Megachiroptera with parasitic encephalitis often do not exhibit any morphologic lesions on gross post mortem examination. Histologic lesions within the brain may include: parasites in cross section in the nervous tissue or subdural spaces, and foci of haemorrhage, necrosis, or polymorphonuclear cell infiltration.

For further information regarding *Angiostrongylus* infections please refer to section 5.2.2.

4.2.2 *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 lifecycle 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).

4.2.3 Incidental Parasitism

Megachiroptera often present with a burden of various ectoparasites, however, most of these infestations appear to be incidental to the host.

Nycteribids and Strebilids are flat flies that commonly infest Australian megachiroptera. 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.

Hepatocystis levinei is an intraerythrocytic protozoon of the family Plasmodiidae. This protozoan infects GHFF and is transmitted by *Culicoides nubeculosus*.

4.3 Viral Disease

4.3.1 Australian Bat Lyssavirus

Aetiology

Lyssavirus has recently been isolated within a wide variety of frugivorous and insectivorous bats throughout Australia. Greater than 40 lyssavirus isolates have been discovered within the tissues of bats that originated from Melbourne to Darwin. Australian bat lyssavirus (previously named pteropid bat virus, or pteropid lyssavirus) is the term now used to describe this virus, which is a member of the family Rhabdoviridae.

Lyssavirus was first discovered in pteropids in May 1996, during investigations into the role of megachiroptera as a reservoir of equine morbillivirus (Hendra virus). A

five month old, female black flying-fox was discovered unable to fly. The animal was euthanased and submitted for gross and microscopic post mortem examination. Histologic examination of the brain revealed marked non-suppurative encephalitis. Small, eosinophilic, intracytoplasmic inclusion bodies (Negri bodies) were evident within some neurons. Nervous tissue from this bat was examined using electron microscopy, and tissues were inoculated into mice (Fraser et al., 1996). The inoculated mice developed acute encephalitis within two to three weeks. Antigen derived from the virus in cell culture and in rodent brain tissue reacted with reagents used for rabies diagnosis (Fraser et al., 1996).

Retrospective investigations identified the first known case of Australian bat lyssavirus in a juvenile black flying-fox, which died in 1995. The bat was being hand raised, and was euthanased due to unusually aggressive behaviour. Mild non-suppurative encephalitis, and intracytoplasmic inclusion bodies were found upon histologic examination of the brain. Immunoperoxidase staining techniques using antirabies monoclonal antibody was used to identify lyssavirus within the brain (Fraser et al., 1996).

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 polymerase chain reaction (PCR) tests identified lyssavirus antigen in her cerebrospinal fluid. Her illness progressed rapidly after the diagnosis was established, and she died (Allworth et al., 1996).

The Rhabdoviruses include classic rabies viruses and genetically related viruses. These viruses are classified into five groups based upon serological characteristics:

- Classic rabies virus
- Logos bat virus
- Mokola virus
- Duvenhage virus

- European bat virus.

Initial research to further characterise the virus isolated from the black flying-fox included histopathology, immunofluorescent testing, immunoperoxidase staining, PCR, electron microscopy, and virus isolation with subsequent monoclonal antibody testing, and genetic sequencing. The result of each test was consistent with the virus belonging to the family Rhabdoviridae. 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).

The prevalence of Australian bat lyssavirus is uncertain; however, investigations conducted in April 1997 determined that 5% of 500 chiroptera tested with indirect immunofluorescent tests were positive for Australian bat lyssavirus (Field et al., 1997). The broad geographic range of the virus and ecological complexity are highly suggestive that the virus has been present in Australia for a significant period of time (Field et al., 1997).

Clinical Signs

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.

Diagnosis

Diagnostic testing to confirm Australian bat lyssavirus is conducted at Australian Animal Health Laboratories in Geelong. None of the diagnostic tests used to detect lyssavirus is 100% sensitive, thus, a variety of tests are conducted to identify the presence of the virus. Viral culture, serology, histopathology, immunoperoxidase staining and immunofluorescent antibody testing are most often employed to establish a diagnosis of lyssavirus infection.

Prevention

Rhabdoviruses are enveloped viruses, which do not persist well in the environment. The virus is 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.

Research was conducted at the Centre for Disease Control in Atlanta to evaluate the use of currently available human and animal sub-unit vaccines in the protection against Australian bat lyssavirus. Sera from humans immunised with commercial human rabies vaccine neutralised Australian bat lyssavirus to the same extent that classic rabies virus was neutralised. Rodents vaccinated with commercial animal vaccines were protected against challenge with Australian bat lyssavirus, but not protected from developing Mokolo virus, a non-rabies lyssavirus (Rupprecht, 1999).

Human lyssavirus prophylaxis consists of pre-exposure vaccination with human diploid cell vaccine, purified chick embryo vaccine or rabies vaccine adsorbed. Human diploid cell rabies vaccine is most commonly used in rabies prophylaxis. Vaccines are administered on the following schedule: days 0, 7, and 21 or 28. Chloroquine or mefloquine antimalarial medication can interfere with the vaccination response, and should not be administered until after the 28 day vaccination period. Immunity should be monitored through serological testing at frequencies dependent upon the risk of exposure. It is recommended that individuals with continuous exposure to lyssavirus, such as individuals working in lyssavirus research labs, be monitored serologically every six months. Individuals who regularly have direct contact with species known to carry lyssavirus should be monitored serologically every two years (Rupprecht, 1999).

If bitten by a bat, wounds should be thoroughly cleaned with soap and water. 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. Individuals who are bitten by an animal suspected to have lyssavirus will usually receive human rabies immune globulin and serial human diploid cell vaccinations if previously unvaccinated, and human diploid cell vaccine boosters if previously vaccinated (Rupprecht, 1999).

4.3.2 Hendra Virus

Aetiology

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 Mackay, which is 1000 km north of Brisbane (Hooper et al., 1996).

The index case in both outbreaks involved heavily pregnant thoroughbred mares at pasture. A paramyxovirus, initially named equine morbillivirus (EMV), was isolated from one person and four horses during investigations into the outbreak in Brisbane. Initial investigations focused on the single outbreak in Brisbane. Serological testing did not reveal any evidence of EMV in a large variety of domestic species, and greater than thirty species of rodents, marsupials, birds, amphibians and insects. Once the outbreak in Mackay was identified, and PCR testing revealed that the same virus was responsible for both outbreaks, investigations focused upon wild species that have contact with horses, and are common to both sites, or migrate between the sites.

Virus neutralising antibody tests conducted in April, 1996 identified antibody towards EMV 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). A paramyxovirus was isolated in September 1996 within the uterine fluid of a bat that aborted subsequent to being injured in a wire fence. This virus was initially named bat paramyxovirus (BPV). Further research, however, revealed that isolates of EMV and BPV were indistinguishable when fragments of the matrix protein gene were subjected to sequencing (Field et al., 1997). Hendra virus is the term now used to describe this virus.

Additional non-fatal cases of respiratory disease have been reported in horses in Townsville, and in a veterinarian in Cairns, 2004 (Promed, 20041214.3307, 2004).

Clinical Signs

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).

Exposure to Hendravirus has not been serologically detected within greater than 100 bat caregivers. Each of the three human cases of Hendra virus infection was 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.

4.3.3 Others

Serological surveys indicate that flying-foxes have also been exposed to Ross River virus, Murray Valley encephalitis; however, they do not appear to act as a reservoir host for these viruses (Prociv, 1986).

4.4 Fungal Disease

Although not reported to be a pathogen of Australian Megachiroptera, *Histoplasma capsulatum* has an affinity for substrates containing large concentrations of nitrogen, such as the faeces of colonial 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.

4.5 Toxicity

Lead poisoning and organophosphate toxicity have been reported in flying-foxes; however, lead poisoning has been the only commonly reported intoxication of flying-foxes. With the introduction of lead-free petrol, clinical disease in flying foxes is now rare.

4.5.1 Lead Poisoning

Aetiology

Lead poisoning has been reported on multiple occasions in flying-foxes. Megachiroptera have been found to have renal lead concentrations as high as 65 ppm (Wilson, 1988).

Engine emissions from vehicles that use lead based petrol are proposed as a source of environmental contamination (Wilson, 1988). Bats presumably ingest foliage and fruit that has been contaminated with lead. During feeding, the bat's pelage is contaminated with lead, which is ingested during subsequent grooming. Bats that have elevated concentrations of lead in their kidneys have also been shown to have high concentrations of lead in their fur and in fur washings (Sutton and Hariano, 1987). Investigations have demonstrated that bats living in a rural environment had lower concentrations of tissue lead than bats living in urban environments (Sutton and Hariano, 1987). Flying-fox fur has a porous scalation pattern that is thought to trap pollen grains for ingestion during grooming. This hair structure, in conjunction with fastidious grooming habits, may predispose flying-foxes to lead toxicity (Sutton and Hariono, 1987).

Clinical signs

Flying-foxes with high concentrations of tissue lead are often found unable to fly. Upon closer examination these bats exhibit ataxia, tremors, vomiting, diarrhoea, constipation, anaemia, salivation, signs of secondary trauma, or they may abort (Wilson, 1988, Sutton and Wilson 1983, Sutton and Hariano, 1987). 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.

Pathogenesis

Lead toxicity occurs as a result of interference in enzymes involved in several metabolic pathways. Altered delta aminolevulenic acid dehydrogatase and haeme synthetase activities lead to anaemia. Increased concentrations of lead within tissues may result in altered cellular oxidative function, and impaired release of impulses at synapses within the nervous system (Locke and Thomas, 1996).

Pathology

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.

Diagnosis

Diagnosis of lead poisoning is confirmed when blood lead concentrations exceed 5 ppm (*u*mol/L) (Locke and Thomas, 1996). Inhibition of serum delta aminolevulenic acid, and increased blood protoporphyrin concentrations can also be used to confirm lead poisoning.

Treatment

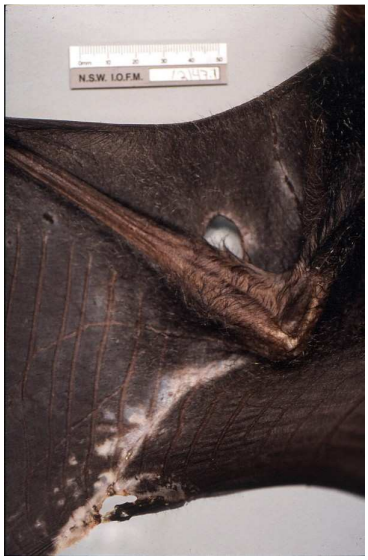
Flying-foxes suffering lead poisoning are treated with calcium disodium edetate (Booth, 1994b). A second course of therapy may be required, since lead stored in the

tissues may be mobilised into the blood stream, especially under conditions of acidosis.

4.6 Traumatic Injury

4.6.1 Soft tissue injury

Wing web lacerations are a common traumatic event in megachiroptera. 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, 1994b).



Electrocution, grey-headed flying fox

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.

4.6.2 Skeletal injuries

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 multiple fine 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 (Damien Higgins, personal communication).

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 (Bill Hartley, personal communication).

Fractured distal femoral epiphyses can result when excessive force is used to pull a flying-fox from a wire enclosure. These fractures are repaired surgically with the use of cross pins (Heard, 1999).

5 Animals mentioned in text

5.1 Mammalia

Short-beaked echidna (*Tachyglossus aculeatus*)
Koala (*Phascolarctos cinereus*)
Common wombat (*Vombatus ursinus*)
Southern hairy-nosed wombat (*Lasiorhinus latifrons*)
Northern hairy-nosed wombat (*Lasiorhinus krefftii*)
Agile wallaby (*Macropus agilis*)
Tammar wallaby (*Macropus eugenii*)
Common ringtail possum (*Pseudocheirus peregrinus*)
Common brushtail possum (*Trichosurus vulpecula*)
Rufous bettong (*Aepyprymnus rufescens*)
Dasyurids (*Dasyuridae*)
Eastern grey kangaroo (*Macropus giganteus*)
Grey-headed flying-fox (*Pteropus poliocephalus*)
Little red flying-fox (*Pteropus scapulatus*)
Black flying-fox (*Pteropus alecto*)

Black rat (*Rattus rattus*)

Brown rat (*Rattus norvegicus*)

Dingo (*Canis familiaris dingo*)

Domestic dog (*Canis familiaris*)

European red fox (*Vulpes vulpes*)

Domestic cat (*Felis catus*)

Domestic horse *(Equus caballus*)

Domestic cattle (*Bos taurus*)

Domestic sheep (*Ovis aries*)

Humans (*Homo sapiens*)

6 References

Allworth A, Murray K, Morgan J (1996) A human case of encephalitis due to a Lyssavirus recently identified in fruit bats. Comm. Dis. Intell. 20: 504.

Barker IK, Beveridge I, Munday BL (1985) Coccidia (*Eimeria tachyglossi* nsp., *E. echidnae* nsp., and *Octosporella hystrix* nsp.) in the Echidna, *Tachyglossus aculeatus*. J. Protozool. 32(3): 523 - 525.

Barrett JL, Carlisle MS, Prociv P (2002) Neuro-angiostrongylosis in wild Black and Grey-headed flying foxes (Pteropus spp.). Aust. Vet. J. 80: 554 - 558.

Booth R (1994) Medicine and husbandry: Monotremes, wombats and bandicoots. In: Wildlife. Proc 233. The Post Graduate Committee in Veterinary Science. Pp. 395 - 413.

Booth R (1994b) Medicine and husbandry: Dasyurids, possums and bats. In: Wildlife. Proc 233. The Post Graduate Committee in Veterinary Science. Pp. 423 - 442.

Canfield PJ, Hartley WJ (1991) Tyzzer's disease (*Bacillus piliformis*) in Australian marsupials. J. Comp. Path. 105: 167 - 173.

Canfield PJ, Hartley WJ, Dubey JP (1990) Lesions of toxoplasmosis in Australian marsupials. J. Comp. Path. 103: 159 - 167.

Carlisle MS, Prociv P, Grennan J, et al. (1998) Cerebrospinal angiostrongyliasis in five captive tamarins (*Sanguinus* spp). Aust. Vet. J. 76 (3): 167 - 170.

Collins GH, Rothwell TLW, Malik R, Church DB, Dowden MK (1992) Angiostrongylus in dogs in Sydney. Aust. Vet. J. 69: 170 - 171.

Collins H (1999) Personal communication.

Dubey JP, Hartley WJ (1993) Disseminated coccidiosis in short-beaked echidnas (*Tachyglossus aculeatus*) from Australia. J. Vet. Diag. Invest. 5: 483 - 488.

Field H, Halpin K, Young P (1997) Serological surveillance of wildlife for emerging viral diseases. Australian Association of Veterinary Conservation Biologists, Annual Conference Proceedings, Brisbane. Pp 107 - 114.

Fraser GC, Hooper PT, Lunt RA, et al. (1996) Encephalitis caused by a Lyssavirus in fruit bats in Australia. Emerging Infect. Dis. 2: 327 - 330.

Garell DM (1999) Toxoplasmosis in zoo animals. In: Fowler ME, Miller RE (eds) Zoo and wild animal medicine. Current therapy 4. W.B. Saunders Company, Philadelphia. Pp 131 - 135.

Griner LA (1983) Pathology of zoo animals. Zoological Society of San Diego, San Diego. Pp 279 - 280.

Hartley WJ, Smith JS, Bellamy T (In preparation) Some neurological diseases seen in the brushtail possum (*Trichosurus vulpecula*) in Australia.

Heard DJ (1999) Medical management of megachiropterans. In: Fowler ME, Miller RE (eds). Zoo and wild animal medicine. Current therapy 4. W.B. Saunders Company, Philadelphia. Pp. 344 - 353.

Hemsley S, Canfield PJ (1994) Dermatitis in free-living common brushtail possums (*Trichosurus vulpecula*). Aust. Vet. Practice. 24(3): 147 - 155.

Higgins DP, Carlisle-Nowak MS, Mackie J (1997) Neural angiostrongylosis in three captive rufous bettongs (*Aepyprymnus rufescens*). Aust. Vet. J. 64: 201 - 203.

Hooper PT, Gould A, Kattenbelt KA, Mitchell G (1996) The retrospective diagnosis of a second outbreak of equine morbillivirus infection. Aust. Vet. J. 74: 244 - 245.

Hum S, Barton NJ, Obendorf D, Barker IK (1991) Coccidiosis in Common Wombats (*Vombatus ursinus*) J. Wildlife Disease 27(4): 697 - 700.

Jenness R, Regehr EA, Sloan RE (1964) Comparative biochemical studies of milks. II Dialyzable carbohydrates. Comp. Biochem. Physiol. 13: 339.

Lynch MJ, Obendorf DL, Stratham P, et al. (1993) Serological responses of tammar wallabies (*Macropus eugenii*) to inoculation with an attenuated strain of *Toxoplasma gondii*. Proceedings of the American Association of Zoological Medicine. St. Louise. Pp 185 - 187.

Martin RW, Handasyke KA, Skerratt LF (1998) Current distribution of sarcoptic mange in wombats. Aust. Vet. J. 76(6): 411 - 414.

Muller GH, Kirk RW, Scott DW (1989) Small animal dermatology. 4th Ed. W.B. Saunders Company, Philadelphia. Pp. 774 - 776.

Munday BL (1988) Marsupial diseases. In: Australian Wildlife. Proc 104. The Post Graduate Committee in Veterinary Science. Pp. 299 - 365.

Murray PK, Selleck P, Hyatt A, et al. (1995) A morbillivirus that caused fatal disease in horses and humans. Science. 268: 94 - 97.

O'Keefe J S, Stanislawek W L, Heath DD (1997) Pathological studies of wobbly possum disease in New Zealand brushtail possums (Trichosurus vulpecula). Veterinary Record. 141: 9, 226-229. 19 ref.

Perrott MRF, Meers J, Cooke MM, Wilks CR (2000) A neurological syndrome in a free-living population of possums (Trichosurus vulpecula). NZ Vet. J. 48: 1, 9-15. 16 ref.

Prociv P (1986) Parasites of Australian flying-foxes. Aust. Mamm., 10: 107-110.

Prociv P (1999) Personal communication.

Reddacliff LA (1981) Dermatoses - zoo animals. In: Dermatology refresher course for veterinarians. No. 57. The Post Graduate Committee in Veterinary Science. Pp. 407.

Reddacliff LA, Bellamy TA, Hartley WJ (1999) *Angiostrongylus cantonensis* infection in grey-headed fruit bats (*Pteropus poliocephalus*). Aust. Vet. J. 77 (7) 466-468.

Reddacliff GL, Parker SJ, Dubey JP (1993) An attempt to prevent acute toxoplasmosis in macropods by vaccination with *Hammondia hammondi*. Aust. Vet. J. 70: 33 - 35.

Rupprecht CE (1999) Rabies: global problem, zoonotic threat and preventative management. In: Fowler ME, Miller RE (eds). Zoo and wild animal medicine. Current therapy 4. W.B. Saunders Company, Philadelphia. Pp 131 - 135.

Skerratt L (1998) Parasitology of *Sarcoptes scabiei var wombati* and the debilitating effects of severe infestation on common wombats (*Vombatus ursinus*). Wildlife Disease Association - Australaisian section, Annual Meeting, Calperum.

Skerratt LF (2003) Cellular response in the dermis of common wombats (Vombatus ursinus) infected with Sarcoptes scabiei var. wombati. J. Wildlife Diseases. 39: 1, 193-202. 34 ref.

Skerratt LF (2003b) Clinical response of captive common wombats (Vombatus ursinus) infected with Sarcoptes scabiei var. wombati. J. Wildlife Diseases. 39: 1, 179-192. 36 ref.

Skerratt LF, Beveridge I (1999) Human scabies of wombat origin. Aust. Vet. J. 77: 9, 607. 8 ref.

Skerratt LF, Martin RW, Handasyke KA (1998) Sarcoptic mange in wombats. Aust. Vet. J. 76(6): 408 - 410.

Smaller J (2004) Serological diagnosis of angiostrongyliasis in macropods and dogs. BSc (Vet) thesis, University of Sydney.

Strahan R (1988) The Australian museum complete book of Australian mammals. Angus and Robertson Publishers, North Ryde, Sydney.

Sutton RH, Hariano B (1987) Lead poisoning in flying foxes. Aust. Mamm. 10: 125 - 126.

Sutton RH, Wilson PD (1983) Lead poisoning in grey-headed fruit bats (*P. poliocephalus*). J. Wildlife Disease 19(3): 294 – 496.

Thompson EG, McLeod BJ, Gill JM (1999) The prevalence of Wobbly Possum disease in a bush/farmland environment. Proceedings of the New Zealand Society of Animal Production. 59: 233-235. 9 ref.

Whittington RJ (1992) Impact of disease on monotreme populations. Proceedings of the Wildlife Disease Association - Australasia Section, Warrumbungles.

Whittington RJ (1993) Diseases of monotremes. In: Fowler ME (ed). Zoo and wild animal medicine. Current Therapy 3. W.B. Saunders Company, Philadelphia. Pp. 269 - 275.

Whittington R, Middleton D, Spratt DM, et al. (1992) Sparganosis in the monotreme *Tachyglossus aculeatus* and *Ornithorhynchus anatinus* in Australia. J. Wildlife Disease 28(4): 636 - 640.

Wilson P (1988) Veterinary Treatment of bats. In: Australian Wildlife. Proceedings 104. The Post Graduate Committee in Veterinary Science. Pp. 517 - 529.