Wildlife Health in Tasmania

DISEASE INFORMATION

- Devil Facial Tumour Disease
- Amphibian Chytridiomycosis
- Platypus Mucormycosis
- Toxoplasmosis
- Sarcoptic Mange
- Avian Influenza
- Seal Tuberculosis

Department of
Primary Industries and Water

Tasmania
Explore the possibilities

Acknowledgments

Introduction

The first part of the section provides wildlife health managers with concise reference material in relation to diseases of significance affecting or potentially affecting Tasmanian terrestrial and marine wildlife.

The second section should be used when presented with sick or injured Tasmanian wildlife. The 'First Response to Sick or Injured Wildlife' form outlines who veterinarians, wildlife health managers, rangers and others should contact and report to when presented with sick, injured or dead Tasmanian wildlife. The 'Wildlife Health Incidence Record / Laboratory Advice' form can be used or photocopied by the investigator. This form should accompany sick or dead wildlife to the Animal Health Laboratories, Prospect, Launceston. Results will be collated and used to monitor wildlife health in Tasmania.

Handling chemicals such as formalin and wild animals potentially affected by zoonotic disease can cause harm to humans. This section serves as a guide only and due care and personal responsibility should be taken.

Devil Facial Tumour Disease

Aetiology

Devil Facial Tumour Disease (DFTD) is a neoplastic disease affecting primarily the face of the Tasmanian Devil (*Sarcophilus harrisii*). The cause of DFTD is the subject of research. The most likely explanation for the observed epizootiology seen since its emergence in the middle 1990's is that this disease is a transmissible tumour (infective allograft cell line) with its most comparable analogue being Canine Transmissible Venereal Tumour.

Occurrence in Tasmania

The existence of dramatic facial tumours were first reported in 1996 (Hawkins et al. 2006). There were no records of similar pathology in over 2000 devils trapped or examined in previous surveys. By mid 2005 DFTD had been histologically confirmed from 41 separate sights, covering 32 930 km (51%) of the Tasmania landmass. At one long-term study site, surveillance commenced before the emergence of DFTD. From these observations it is suggested that DFTD has spread geographically from a nidal focus in a high-density devil population. Of 147 devils with DFTD-like signs, at least 140 were sexually mature. The proportion of animals displaying signs at any one site reached up to 83% (15/18) of trapped adults. Spotlight surveys and trapping indicated a significant local association between population decline and date of first report of DFTD (Hawkins et al. 2006). In the region where the disease was first reported mean spotlight counts declined by 80% from 1993-1995 to 2001-2003.

On the basis of the threat posed by DFTD, the devil has been listed as a threatened species (classed as *vulnerable*) under Tasmanian and Commonwealth endangered species legislation.

Epidemiology

Devil Facial Tumour Disease is a new and emerging disease that is now widespread in, and constitutes a serious threat to the survival of the devil in its only wild habitat, Tasmania. The disease appears to be consistently fatal in afflicted individuals. DFTD primarily affects wild devils and at the time of writing has been confirmed within one captive devil population due to a

security breach. Transmission may occur by the transferral of neoplastic cells in saliva during biting episodes, via open wounds during mating, via open oral wounds while feeding on a carcass where a diseased devil has left loose neoplastic cells, or via open oral wounds during cannibalism of dead DFTD devils. A recurring feature of all tumours examined, irrespective of their stage, has been the absence of any cell-mediated immune response in the form of lymphocyte or macrophage activity. This suggests that either growing tumours can evade immune recognition of their host, or devils are insufficiently genetically diverse to recognise the tumours as foreign. Very low devil genetic diversity has been demonstrated (Jones et al. 2004).

Clinical signs

Nodular soft tumour masses occur in the mouth, under the tongue, along the gum line, on the facial skin areas of the cheeks, lips, muzzle, head and neck. Tumours have also been seen occasionally on the rump and various other parts of the body. Tumour growth is rapid and affected animals usually die within 3-8 months of the lesions first appearing.

Tumour masses are quite soft and friable with many tumours having ulcerated surfaces, which are then prone to secondary infection, bleeding and necrosis. Facial disfigurement, cavitation, and fistula formation across the cheek tissues are regular findings. Tumour invasion into bone and other soft tissues can lead to tooth loss and secondary infections.

Metastases to draining lymph nodes (submandibular and cervical) are seen in devils with advanced primary tumours. Multiple nodular tumour masses of varying sizes can be found in various organs and tissues such as liver, kidney, lungs, heart, spleen and bone.

Diagnosis

Late-stage tumours (FIGURE 1) are pathognomonic for DFTD however a definitive diagnosis relies on microscopic examination of tumour tissue sections (histopathology). A diagnosis by visual examination of animals with early stage tumours is less accurate (FIGURE 2). The pathogenesis and aetiology are the subjects of ongoing research. Histopathology and immunohistochemistry defines the tumour type as an anaplastic sarcoma of probable neuroectodermal origin (Loh et al. 2006). The likelihood of a 'prodromal' stage of the disease in affected animals with no visible lesions is currently unclear.

Further work is underway to identify specific genomic or proteomic markers for this disease.

Differential diagnoses for DFTD include chronic proliferative granulation tissue that is common in fascial tissues of adult devils, follicular/epidermal cysts, facial abscesses and other cutaneous nodular neoplasms.

FIGURE 1. ADVANCED DFTD LESION ON THE FACE OF A TASMANIAN DEVIL


FIGURE 2. SUSPECTED EARLY STAGE DFTD LESIONS IN THE ORAL CAVITY

Pathology

The most characteristic gross feature of this disease is the localisation of the pathology to the face or oral cavity. Typical DFTD lesions can be raw ulcerated nodular masses or irregularly swollen masses disfiguring the cheek, lips and gum tissues. An exposed or ulcerated tumour mass may show cheek fistulae, necrotic cavitations, secondary infections, myiasis, traumatic bleeding and abrasions. Early stage tumours are thought to commence as sub-epithelial nests of transformed cells, perhaps associated with bite wounds inflicted by devil-to-devil contact. Capture-recapture observations of affected devils demonstrates that small superficial nodules emerge at an epithelial surface and as they grow in size they can become eroded on the surface and continue to invade adjacent tissues. Advanced stage tumours can grow to greater than 15cm diameter. In these cases secondary tumour metastases are common in the draining lymph nodes, and other organs and tissues of the body (heart, lung, adrenal gland, pituitary, spleen and kidney) (FIGURE 3).

FIGURE 3.
METASTASES OF DFTD TO THE SPLEEN
(TOP LEFT), LIVER AND DIAPHRAGM
(BELOW LEFT) AND
LUNG (BELOW RIGHT)
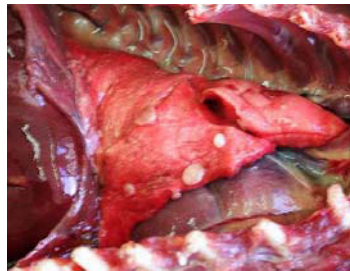
Handling, restraint and biosecurity

Tasmanian devils should be placed into a clean hessian sack to reduce stress to the animal and disease transmission. The handler should wear disposable latex gloves, washable apron, and mask.

By patting down gently over the sack, the head of the animal may be located and pressure over the neck region should adequately restrain the animal for gaseous anaesthesia, injection, or microchip reading / insertion.

Carcasses of devils, whether diseased or not, should either be left where the animal was first found / caught or disposed of in accordance with instructions in the second part of this section. Veterinarians should bag devil carcasses with in-contact gloves and sacks.
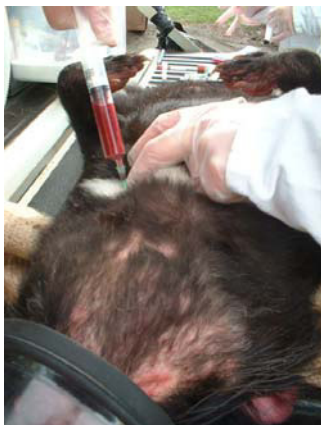
Anaesthesia

After being placed in a hessian sack, general anaesthesia is the most efficient method for thoroughly examining a devil. Gaseous anaesthesia is most commonly used with isoflurane the anaesthetic of choice. The gas may be delivered via a facemask through the hessian sack. Injectable anaesthetics may be used on devils however they often do not produce as steady a plane of anaesthesia as isoflurane. Injectable agents include Zoleteil® (10mg/kg) and Domitor® / ketamine combinations. Intubation may then be attempted, although the muscular larynx of the devil can make intubation difficult. Assessment of anaesthetic depth is usually achieved via measurement of jaw tone, reaction to pain, and assessing muscle fasciculations on the head, because palpebral reflex is often unreliable.

Physical examination

After the devil has been anaesthetised, a routine clinical examination may be performed. Lung and heart auscultation is greatly facilitated under anaesthetic as conscious devils become stressed during handled which greatly elevates heart rates and makes chest auscultation difficult. Rectal temperatures can also be taken; the normal temperature range is between 35.5^{o}C and 36.5^{o}C. Abdominal palpation is similar to that in domestic species, remembering that paired epipubic bones will be present in the caudoventral abdomen. Devils are particularly prone to developing neoplasms in general; therefore any masses detected should be investigated.

Blood sampling devils can prove to be challenging depending on the amount of blood required. The most accessible veins are the ear veins (<0.5ml), jugular veins (>10mls), and the saphenous vein (<4mls). When using jugular venipuncture, place the animal on its back and extend the neck. A roll under the neck also helps to extend the neck (FIGURE 4). A thumb is placed at the thoracic inlet and the needle is inserted approximately 1cm above the thumb at right angles to the skin.

FIGURE 4.
COLLECTING BLOOD
FROM THE JUGULAR VEIN OF AN
AN ANAESTHETISED DEVIL

Management and research

- The DFTD Disease Management Strategy (2005) comprises the following five key elements:

- Ongoing field mapping and monitoring of the disease, diagnostic research and investigations;

- The isolation of devils in quarantined insurance populations;

- Field trials to test the effectiveness of isolation and disease suppression techniques in maintaining and restoring devil populations and limiting the apparent spread of the disease;

- Ongoing work to maintain the biosecurity and genetic diversity of captive devils held by wildlife parks in Tasmania; and

- Improved management of captive devils held by interstate wildlife parks to maximise breeding success.

Human health

There is no research to date to indicate that humans could be affected by DFTD. It is considered prudent, however to wear a mask and gloves when handling devils.

Notes:

Amphibian Chytridiomycosis

Aetiology

Amphibian chytridiomycosis (*Batrachochytrium dendrobatidis)* is a chytridiomycete fungus. This fungus completes its life cycle in the skin of amphibians utilising keratin as a protein substrate (Powell 1993, Berger et al. 1998). The disease has become the agent of a global pandemic in amphibian populations and has been linked to the decline and extinction of several frog species.

Global distribution

Amphibian chytridiomycosis is currently found in two of the three amphibian orders, Anura and Caudata, and has been reported in 17 families, 41 genera and 137 species of frog (Speare and Berger 2000). Over the course of the last 40-50 years *B. dendrobatidis* has managed to enter, establish and spread in every continent where amphibians exist (Daszak et al. 1999, Daszak et al. 2003). Reports of infection have been recorded in free-living frogs in Australasia, Europe, Asia, Africa and the Americas.

Occurrences in Australia

Chytridiomycosis has been found in wild amphibian populations on the east - coast of Queensland, New South Wales, the Australian Capital Territory, Victoria, the south-west and Kimberley regions of Western Australia, and Tasmania.

Occurrence in Tasmania

- Amphibian *B. dendrobatidis* is established in free-living populations of Tasmanian frogs in suburban and peri-urban locations.

- Seven species of frog have shown visible lesions of chytridiomycosis during the tadpole stage of development (confirmed by PCR analysis and/or histology); the Brown Tree Frog (*Litoria ewingii*), Tasmanian Tree Frog (*Litoria burrowsae*), Eastern Banjo Frog (*Limnodynastes dumerilii*), Spotted Marsh Frog (*Limnodynastes tasmaniensis*), Tasmanian Froglet (*Crinia tasmaniensis*), Common Froglet (*Crinia signifera*), and the Green and Gold Frog (*Litoria raniformis*).

- Two widespread and abundant species, the Brown Tree Frog and Common Froglet may act as infected reservoirs or dispersal agents of the disease (Mathew Pauza pers. comm.).

- With recorded declines in the abundance and distribution of the Green and Gold Frog, surveillance to determine the chytriomycosis status of this species across its known habitat range is a priority. Species of frog with highly specialised ecologies and restricted distribution, such as the Moss Froglet (*Bryobatrachus nimbis*) and Tasmanian Tree Frog (*Litoria burrowsae*), require monitoring.

Epidemiology

- It is likely that the skin of all amphibia can be used for the replication of the keratolytic fungus *B. dendrobatidis*. Keratinised tissues (skin and claws in adults, jaw sheaths and tooth rows in tadpoles) are thought to be the only sites of infection. Zoospores migrate through the stratum corneum into the deeper layers of the skin and colonise cells above the stratum germinativum. Autoinfection then leads to severe clinical disease.

- Tadpoles can display signs of oral chytridiomycosis (jaw sheath depigmentation), however it is usually metamorphlings and juvenile frogs that show clinical signs. Death usually follows within 2-3 days. There is no sex-linked predisposition.

- Mortality rate depends on three factors: susceptibility of host species, environmental characteristics (particularly low-moderate temperatures) and possibly the strain of *B. dendrobatidis.* Mortality in adults and metamorphs can be up to 100% in susceptible species such as Green and Gold Frogs.

- Transmission is via motile zoospores that leave the host via discharge tubes projecting through the surface of epithelial cells. Zoospores require water to survive although a liquid film is adequate. Zoospores spread via water-borne transfer (frog to tadpole stages and tadpole to tadpole) or through skin contact during mating.

- There is a carrier state. Frogs with subclinical infection can act as long term carriers. There is no recognised resting stage in the *B. dendrobatidis* life cycle, although zoosporangia can remain viable for several months under favourable conditions. The fungus does not survive drying.

- Chytridiomycosis spread from a nidus of infection can occur across a landscape by natural water-borne or fomite transfer. Aquatic substrates, plants and organisms as well as water can carry stages of the *B. dendrobatidis*. Other non-amphibian sources of keratin such as feathers or exfoliated skin have been sources of *B. dendrobatidis* in laboratory analysis - implications for transmission in nature are currently unknown. The movement of wet soil contaminated with *B. dendrobatidis* could also spread infection.

- The greatest incursion threat to *B. dendrobatidis* -free environments is the introduction of carrier frogs. Accidental introduction of frogs can occur via imported fruit, nursery plants, bulk horticultural produce etc. Local spread by human activities occurs where frogs, tadpoles or infective fomites are transferred to other wetlands.

Clinical signs

- Tadpoles with serious oral chytridiomycosis display depigmentation of their jaw sheaths and loss of tooth rows in the oral disc.

- Abnormal behaviours and changes in normal skin colouration are early clinical signs in some frogs. Absence of clinical signs does not preclude chytridiomycosis. Frogs become slow and somewhat uncoordinated in their movements, assume abnormal sitting postures and show stiffness in their body and limbs. Affected frogs become inappetent and appear grossly dehydrated when the disease is terminal.

- Hyperkeratinosis and sloughing in chytridiomycosis are typically microscopic and not detectable at the clinical level with any degree of confidence.

Diagnosis

- Examining tadpoles for signs of segmental depigmentation and/or thinning of the paired jaw sheaths and tooth rows in the oral disc can be a useful field test for the presence of chytridiomycosis (Obendorf 2005, Fellers et al. 2001, Knapp and Morgan 2006) (FIGURE 1 and FIGURE 2). This is probably the best field indicator for the presence of *B. dendrobatidis* in frog habitats. Detection requires magnification by an experienced practitioner. Caution needs to be taken if using this technique as a definitive test for Chytridiomycosis as cold climatic conditions have been demonstrated to cause similar signs.
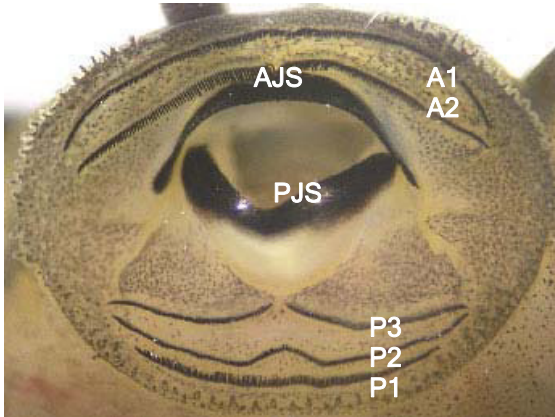
FIGURE 1.
NORMAL ORAL DISC FEATURES OF A BROWN TREE FROG (*LITORIA EWINGI*) TADPOLE. AJS - ANTERIOR JAW SHEATH; PJS - POSTERIOR JAW SHEATH; A1, A2 - ANTERIOR TOOTH ROWS 1 & 2; POSTERIOR TOOTH ROWS - P1, 2 & 3
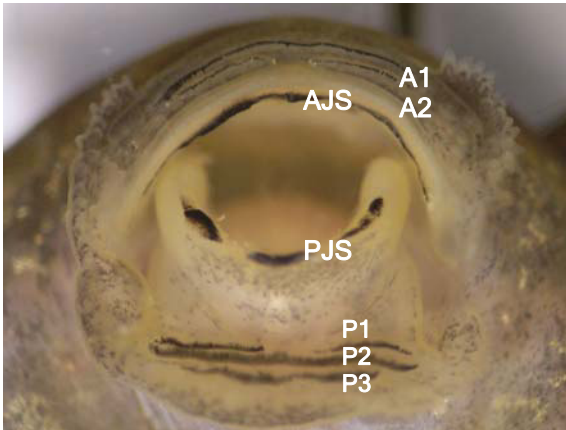
FIGURE 2.
ABNORMAL ORAL DISC FEATURES OF AN EASTERN BANJO FROG (*LIMNODYNASTES DUMERILII*) TADPOLE. NOTE THINNING & LOSS OF PIGMENTED JAW SHEATHS AND FINE TEETH WITH SEGMENTAL LOSS OF TEETH IN SEVERAL ROWS

- Chytridiomycosis can be diagnosed in tadpoles and adults by detecting *B. dendrobatidis* in the skin. This can be done by histopathology, culture, or by a gene probe Taqman PCR (Polymerase Chain Reaction) test. The real time Taqman PCR test is currently the standard test for chytridiomycosis surveillance and monitoring.

- While the organism has been cultured from soil and water samples in vitro (Johnson and Speare 2005), more extensive studies are needed to reliably detect *B. dendrobatidis* zoosporangia in the environment and in non-amphibian hosts.

Pathology

- In most cases there are few gross lesions. Occasional cases have increased sloughing of skin, but this is rarely visually detectable.

- Local hyperkeratosis of infected and adjacent cells occurs with presence of zoosporangia inside cells. Epithelial cells undergo dissolution, often leading to sloughing of the most superficial skin layer. Usually there is no associated inflammatory reaction in the dermis.

- The sites of predilection are the feet, limbs and drink patch however in heavy infections other sites on the body are also affected. There are no consistent lesions in other organs.

- For complete histological / microbiological description see Berger et al. (1999).

Laboratory diagnostic specimens

Samples needed:

- For Taqman PCR: Swab (MW100) taken from the skin of feet, limbs, mouth parts and ventral body. Swabs should be stored at 4°C.

- For histopathology: Skin of feet, fixed in 10% buffered neutral formalin.

Management

Infection of amphibians with *B. dendrobatidis* has been listed as *a Key Threatening Process* (Environment Protection and Biodiversity Conservation Act 1999 (EPBC Act) 2005) and a *Threat Abatement Plan* has been prepared (Department of Environment and Heritage 2006).

This threat abatement plan has two broad goals:

- to prevent amphibian populations or regions that are currently chytridiomycosis-free from becoming infected by preventing further spread of *B. dendrobatidis* within Australia

- to decrease the impact of infection with *B. dendrobatidis* on populations that are currently infected

Amphibian chytridiomycosis is not included in AUSVETPLAN.

Human health implications

B. dendrobatidis will not grow above 28°C and dies if held at 37°C for 4 hours. Homeotherms are therefore considered unsuitable hosts.

Notes:

Mucormycosis in the Platypus

Aetiology

Mucor amphibiorum is the causative agent of mucormycosis in the platypus (Obendorfet et al. 1993). *M. amphibiorum* is a dimorphic fungus occurring in a yeast form in infected tissues or in a hyphal form in the environment or culture medium (Frank et al. 1974).

Natural hosts

Naturally acquired infection has been reported in anuran amphibians (frogs and toads), and the platypus (Frank 1976, Munday and Peel 1983, Obendorf et al. 1993, Speare et al. 1994, Connolly et al. 1998). Infection has also been reported in salamanders exposed to infected captive anurans (Frank 1976).

World distribution

M. amphibiorum is considered to be an endemic Australian fungus.

Australian distribution

Mucormycosis is known to occur in free-ranging platypuses in Tasmania and in amphibians in Queensland, the Northern Territory and New South Wales. Infection in Tasmanian frogs or mainland platypuses has not been detected to date.

The distribution of mucormycosis has expanded since it was first detected at Campbell Town in 1982 and now occurs in 11 catchments in northern Tasmania (South Esk River, North Esk River, Supply River, Piper River, Mersey River, Upper Derwent River, Inglis River, Emu River, Hatfield River, Lower Derwent and Wilmot).

Epidemiology

The prevalence of disease can be high at affected sites (Connolly et al. 1998). In a 12 month study the proportion of platypuses captured at Brumby's Creek, Cressy with disease was 12/36 platypuses captured, and from Liffey River, Carrick was 2/3 platypuses captured. Mortality due to mucormycosis is also

thought to be high in populations at infected sites (Connolly et al. 1998) however uncertainty exists, as dead platypuses are seldom recovered in a condition that allows the cause of death to be determined with confidence.

Transmission of *M. amphibiorum* has been proposed to occur via cutaneous and/or respiratory routes (Munday and Peel, 1983, Obendorf et al., 1993, Connolly et al., 1998, Mundayet al., 1998). Superficial skin wounds such as those caused by tick attachment are thought to be sites at high risk for infection.

Potential risk factors for disease that have been considered include water quality and agent pathogenicity. Water quality was not found to increase disease risk in one study (Connolly et al. 1998). A second study by Stewart and Munday (2004) found that Tasmanian *M. amphibiorum* may be more pathogenic than mainland isolates.

Clinical signs

Infection with *M. amphibiorum* causes a severe granulomatous and often ulcerative dermatitis in the platypus, which may progress to involve underlying muscle and occasionally disseminate to internal organs, particularly the lungs (Obendorf et al. 1993). The gross appearance of the skin lesions varies from non-ulcerated, hairless nodules and abscesses, to ulcers with underrun or thickened margins, sinuses exuding pus, or exuberant granulation tissue attempting wound repair (FIGURE 1) (Connolly et al. 1998). Some lesions appear as discrete entities, whereas others coalesce to form plaques. Lesions have been found on haired regions including the hind limbs (38%), fore limbs (6%), tail (19%), trunk (6%) and head (6%), and unhaired regions - webbing of the fore limbs (13%) and bill (6%). Some affected animals have lesions at more than one site.

Platypuses with mucormycosis are alert and display normal responses to capture and handling (Connolly et al. 1998). However, morbidity and death is suspected to follow from secondary bacterial infections or impaired thermoregulation and mobility.

FIGURE 1. THE GROSS APPEARANCE OF MUCORMYCOSIS IN THE TASMANIAN PLATYPUS
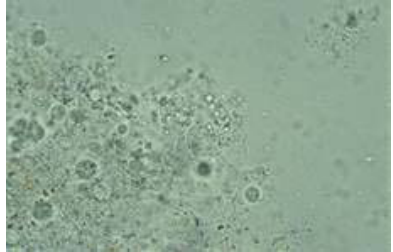
Diagnosis

Diagnostic criteria include the presence of skin ulcers, identification of fungal structures cytologically or histologically, and a positive culture of *M. amphibiorum*.

Samples required for culture and cytology include swabs, fine needle aspirates or a biopsy of lesions from live platypuses. Tissue samples of lesions from dead platypuses are suitable for cytology and culture and fixed tissue samples (in 10% formalin) are suitable for histopathology.

Cytologically, spherules typical of *M. amphibiorum* can be observed in unstained wet preparations, Diff Quik smears made from swabs, or fine needle aspirates (FIGURE 2).
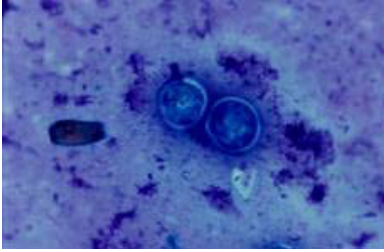
FIGURE 2. *MUCOR AMPHIBIORUM* (YEAST FORM) IN A DIFF QUIK SMEAR

MUCOR AMPHIBIORUM (YEAST FORM) IN AN UNSTAINED WET PREPARATION

MUCOR AMPHIBIORUM (HYPHAL FORM) CULTURE ON SABOURAUDS DEXTROSE AGAR

M. AMPHIBIORUM (HYPHAL FORM) CULTURE SMEAR STAINED WITH LACTOPHENOL COTTON BLUE

Differential diagnoses

1. Traumatic injury and inflammation/infection associated with fishing line entanglement.

2. Tail wear associated with breeding behaviour.

3. Ringworm infection.

4. Bite wound infection (eg. dog attack)

Treatment

Treatment of mucormycosis in the free-living platypus has not been attempted. The disease has not been detected in captive platypuses.

Human health implications

Humans are considered unsuitable hosts for *M. amphibiorum,* as growth does not occur at temperatures above 36°C.

Management and research

Further research is needed to describe the impact of infection on affected populations, identify the ecological niche and host range of *M. amphibiorum* and the risk factors for infection. An efficient surveillance method is also needed for monitoring disease spread and assessing the risk to platypuses in apparently disease free areas such as the World Heritage Area.

This information will improve our understanding of the epidemiology of this disease and enable potential management options to be identified.

Report all sightings of affected platypuses to the Wildlife Health Officer, Biodiversity Conservation Branch, DPIW on 03 6233 6556. Follow the procedure outlined in the second half of this section if presented with a diseased platypus.

Notes:

Toxoplasmosis

Aetiology

Toxoplasma gondii, a protozoan parasite, is the causative agent of the zoonotic disease toxoplasmosis.

Natural hosts

Cats (*Felis spp.*) are pivotal in the transmission of *T. gondii*. Several species of the Family Felidae are definitive hosts for this coccidian parasite. The intermediate host range includes warm-blooded vertebrates - birds, placental mammals and marsupials.

World distribution

Toxoplasmosis occurs throughout the world in locations where introduced or naturally occurring members of the Family Felidae exist. The domestic cat is the most effective maintenance host of *T. gondii* globally. When cats entered Australia, *T. gondii* became established within urban/domestic, rural and wildlife populations.

Australian distribution

Toxoplasmosis is widespread throughout Australia and is recognised as a significant cause of mortality in captive marsupials. Infection has been reported in free-living herbivorous, omnivorous and carnivorous marsupials. Introduced animals affected include sheep, goats, to a lesser degree cattle, pigs and deer species.

Epidemiology

Unsporulated oocysts (~10 um in diameter) are passed in very large numbers by cats intermittently for up to 3 weeks at a time depending on their immune status (FIGURE 1). Oocysts become infective following sporulation, which occurs 24-96 hours after leaving the host.

Ingestion of vegetation contaminated with *T. gondii* sporulated oocysts is the most likely source of infection for herbivorous marsupials (kangaroos and wallabies). Infection in carnivorous marsupials results from eating cysts in the

body tissues of predated or scavenged mammals or birds. The most likely source of infection for omnivorous and insectivorous marsupials is paratenic hosts such as earthworms, coprophagic insects and soil arthropods.

The incubation period after exposure to a concentrated source of infective *T. gondii* can be in 10 -14 days, but some clinical onset of disease appears to be associated with recrudescence of pre-existing infection. These instances may be triggered by immunological, nutritional or weather stress factors. Mortalities in free-living wildlife populations are commonly seen during the autumn and winter period, and in captive wildlife disease outbreaks can occur at any time. Orphaned juvenile animals undergoing transition to solid foods and starting to graze are particularly susceptible to this disease. Mass mortalities or morbidities due to toxoplasmosis occur as a result of multiple exposure factors (nutrition, ambient conditions, age structure, immunology) and not from transmission between animals within the population.
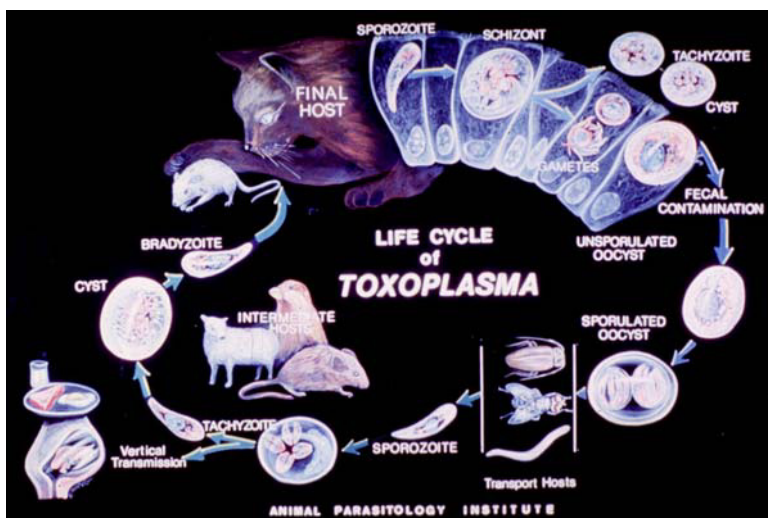

FIGURE 1. LIFECYCLE OF TOXOPLASMOSIS (DUBEY 1994)

Clinical signs

Toxoplasmosis infection is often unapparent. Clinical illness associated with infection 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. Signs include incoordination, erratic staggering movements, body weakness (paresis), apparent blindness, and respiratory distress. Other signs include inappetence, docility and a tendency for nocturnal animals to be unnaturally observed during daylight hours. Fever is not a feature.

Diagnosis

A two-stage direct and modified agglutination test to detect the presence of IgG and IgM antibodies specific to *Toxoplasma gondii* is the most diagnostic test available for the disease currently. The advantage of this test is its application to virtually any host species and only small volumes of serum or plasma are required. The test is capable of differentiating between acute and subacute disease, and carrier states.

Clinical pathology

In clinical cases blood parameters are consistent with general tissue necrosis, acute inflammation and relative lymphopenia.

Laboratory diagnostic specimens

Samples to collect from live animals suspected of toxoplasmosis include whole blood for serology and clinical pathology. Post-mortem samples include heart blood for serology, formalin-fixed heart, lung, brain and any reactive lymph nodes (FIGURE 2 and FIGURE 3).

FIGURE 2. SECTION THROUGH THE MID-BRAIN OF A COMMON WOMBAT (VOMBATUS URSINUS) SHOWING AREAS OF MALACIA, CEREBRAL OEDEMA AND HAEMORRHAGE ASSOCIATED WITH PROLIFERATING TOXOPLASMA CYSTS
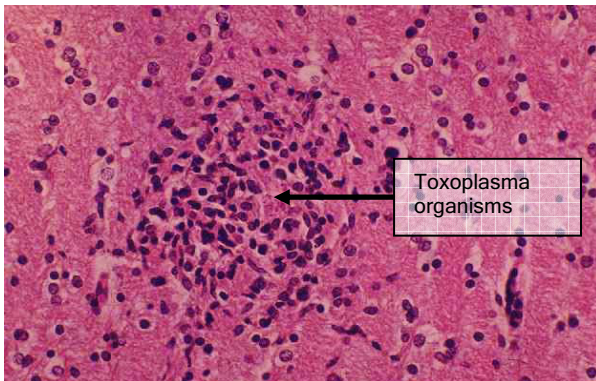
FIGURE 3. HIGH-POWER HISTOPATHOLOGY SECTION SHOWING A NEURONE GRANULOMA (HOST CELL INFLAMMATION) AROUND TOXOPLASMA ORGANISMS RELEASED FROM A TISSUE CYST (DUBEY 1994)

Treatment

Treating or nursing clinically affected animals is not recommended and is invariably unsuccessful. In case of multiple mortality or morbidity events in captive and endangered species where toxoplasmosis has been confirmed, prophylactic treatment of in-contact animals using oral anti-coccidial drugs may limit losses.

Prevention and control

Reducing the opportunity for highly susceptible wildlife species and juvenile herbivorous marsupials to be exposed to potentially infective environments or foods is the only intervention control currently available. As cats are pivotal to the maintenance and transmission of *T. gondii*, reducing the density and activity of feral or stray cats at an affected location is recommended. Control measures are particularly relevant for species that are endangered and where toxoplasmosis is identified as a key threatening process. A vaccination is currently not available for marsupials.

Surveillance

Surveillance opportunities include opportunistic sampling through wildlife monitoring programs. Targeted surveillance is appropriate in cases where *T. gondii* is identified as a key threatening process for an endangered species.

Research needed

- Impact on particular populations or species.

- Association of feral cat density, mammal biodiversity and disease expression.

- Monitoring wildlife health in association with feral cat control.

Human health implications

Humans who have not developed appropriate immunity, particularly the immunosuppressed, are potentially at risk from ingestion of oocysts in cat faeces. Infection is also possible (though less likely) from intermediate hosts via ingestion of tissue cysts (eating under cooked or infected meat), or via skin and mucous membrane contamination (contact with highly infected carcasses as in the case of butchers). Exposed non-immune pregnant women may place their foetuses at risk.

Sarcoptic Mange

Aetiology

The causative agent, *Sarcoptes scabeii* is an obligate parasitic mite, 200-500 μm in length.

Natural hosts

Sarcoptes scabeii was first described in humans. *S.scabeii* is an emerging disease affecting an increasing number of mammalian species within 8 orders: the Artiodactyla, Perissodactyla, Carnivora, Primates, Insectivora, Lagomorpha, Rodentia and Diprotodonta.

The old, young and immunocompromised have higher densities of mite infestation.

World distribution

Sarcoptic Mange occurs throughout the world.

Occurrences in Australia

S. scapeii is known to be widespread throughout Australia. It occurs in a wide range of Australian native and introduced mammals. Humans are also affected.

Sarcoptic mange is endemic in common wombat populations throughout their range. The disease is relatively common in red fox populations.

Epidemiology

Most information on *S. scabeii* is derived from opportunistic wildlife observations and one systematic study on common wombats in Victoria.

Morbidity and mortality rates are high in naïve populations of common wombats and possibly red foxes. Population declines may be large and subsequent endemic disease may slow, limit or prevent recovery from *S. scabeii*. Much lower rates of morbidity and mortality appear to occur in other species in Australia.

The incubation period for clinical signs to develop is around 14 days but reinfestation can result in a 24 hour incubation period.

Transmission may occur at any time but is most likely to occur when mite densities are high. High densities of mites (>1000 mites/cm^2) may occur within 2-3 months of transmission.

Most transmission between wombats is thought to occur due to sharing of burrows. This obligate parasite can live for around 3 weeks in the environment. Close contact is more important in other species for transmission.

Clinical signs

Evidence of infestation in wombats may begin with erythema followed by adherent parakeratotic scale and then alopecia (FIGURE 1). Other species in Australia tend to have less adherent parakeratotic scale than wombats. The parakeratotic scale initially appears as confluent sheets of dandruff. This may build up over time into an adherent crust up to 1 cm thick. Fissures develop in the crust and underlying epidermis resulting in exposure of the dermis, haemorrhage, bacterial infection and sometimes flystrike.

Diagnosis

Definite diagnosis relies on examination of a skin scraping (10 cm^2) for *Sarcoptes scabeii* with a dissecting microscope. Parakeratotic crust in 70% alcohol may be sent to a laboratory for diagnosis.

Pathology

Pathological changes associated with *S. scabeii* infestation include epidermal inflammation, immediate and delayed type hypersensitivity dermal responses, secondary bacterial infections of the dermis, and emaciation.
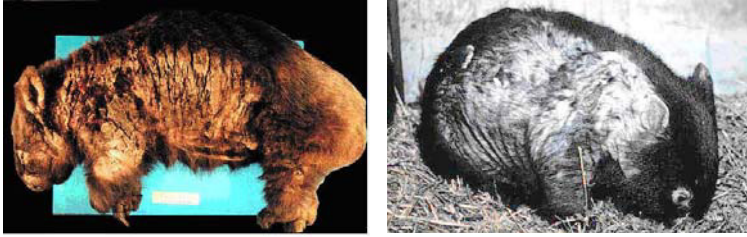
FIGURE 1. WOMBAT WITH SARCOPTIC MANGE

WOMBAT AFTER TREATMENT

Treatment

Treatment should be initiated early where possible. Treatment should include repeated injections of long-acting acaricides, removal of parakeratotic scale crust (bathing using keratolytic agents) and antibiotics for secondary bacterial infection. Topical acaricides may not reach mites due to a failure to penetrate parakeratotic scale crust or be adequately absorbed systemically due to a thickened epidermis. Euthanasia may be appropriate for severely emaciated and debilitated individuals.

Prevention and control

Sarcoptes scabeii is susceptible to heat and dessication. Mites survive for approximately three weeks off the host under favourable conditions of high humidity (100%) and low temperatures (10°C).

Surveillance and management

Passive surveillance is advisable by wildlife managers, veterinarians, wildlife carers, farmers, naturalists and biologists. Cases should be reported using the 'Wildlife Health Incidence Record / Laboratory Advice' form provided at the back of this chapter.

Research needed

Research is needed in the following areas:

- Modes and degree of *S. scabeii* transmission between and within species.

- Impact of *S. scabeii* on affected wildlife populations.

- Effective coordinated surveillance and management techniques.

Human health implications

There are reports of transmission of sarcoptic mange form wombats to humans (Skerratt and Beveridge 1999). Generally Sarcoptic Mange associated with wildlife is a mild self-limiting disease.

Notes:

Avian Influenza

Aetiology

Avian influenza (AI) is caused by viruses of the Orthomyxoviridae family. There are three types of influenza virus (A,B and C). Only Type A influenza virus has been isolated from cases of AI. There are two strains of AI depending on their effect on poultry - low pathogenic (LPAI) and high pathogenic (HPAI). LPAI causes mild disease while HPAI causes severe clinical signs and high mortalities. Within strains, subtypes are categorised on haemagglutinin (H) and neurominidase (N) antigens. The most recent epidemic of HPAI has been due to the H5N1 subtype that has also been shown to be a zoonosis.

HPAI is a notifiable disease throughout Australia.

Natural hosts

Wild waterfowl (ducks, swans and geese) are considered the natural hosts of AI. They are also considered the major reservoir host for poultry. Waterfowl generally do not show clinical signs unless a very virulent virus is involved (eg H5N1).

Susceptible species

Almost all bird species including psittacines are susceptible to AI infection although disease outbreaks have been reported most frequently in domestic chickens and turkeys. Mammals, both terrestrial and marine, including humans may be susceptible – susceptibility and clinical severity is determined by virus subtype and host response.

World distribution

The distribution of AI waxes and wanes according to the distribution of bird species, natural and human-related movement patterns and the virus strains active at the time. The recent AI epidemic which started in 2003 has involved south-east and central Asia, Europe and central and northern Africa.

Occurrences in Australia

Mainland Australia has had five outbreaks of HPAI in commercial poultry. All have been linked to wild bird contact. The outbreaks were in Victoria (1976, 1985 and 1992), Queensland (1994) and New South Wales (1997). The disease was eradicated in each instance. There has never been an outbreak of AI in Tasmanian poultry.

Evidence of exposure to various subtypes of LPAI has been detected in wild birds in Australia.

Epidemiology

Influenza viruses are capable of rapid changes in host specificity and clinical picture. Morbidity and mortality rates vary greatly.

The virus survives best in the environment in moist, cool conditions. It survives in faeces for up to 35 days at $4^{o}C$ and in lake water for 4 days at $22^{o}C$. The virus is stable over a pH range of 5.5-8.

AI viruses are highly susceptible to disinfectants including detergents if items are thoroughly scrubbed before disinfected.

Incubation periods varying widely from a few hours to 16 days but usually is less than 3 days.

Innate, passive and active immunity varies according to the avian species and AI sub-type, and is thus difficult to predict. Vaccination of poultry is an ongoing issue in terms of delivery systems and relative effectiveness.

AI is highly contagious and most commonly spread by oral contact with faeces or secretions of diseased birds, usually by drinking or eating contaminated food or water. Other common means of spread between flocks are bird movements, infected eggs, contaminated eggshells, clothing, footwear and equipment.

Wild bird migration and domestic bird movements have been implicated in the recent AI epidemics overseas.

Waterfowl hosts of HPAI in Asia and Europe do not migrate to Australia in significant numbers. Wading birds do migrate to Australia however these species are considered a low risk for spreading the disease.

Clinical signs

The clinical signs of AI look similar to some other avian diseases such as Newcastle Disease. Sudden onset of significant (>10%) morbidity or mortality with one or more of the following signs must be investigated as a suspect case; depression (FIGURE 1), weak and staggering, noticeable respiratory movements, watery eye and nasal discharge, discolouration of legs and keels, diarrhoea and nervous signs (eg head tilt, paralysis, muscle weakness).

Unfortunately the initial clinical picture can vary enormously from subtle reductions in food and water intake and poor egg production to mass mortality without other signs.

Death from HPAI typically occurs within 48 hours of infection. Mass mortalities over a 24 hour period in domestic poultry is common – often without other signs.

Photo courtesy of CSIRO

FIGURE 1. CHICKENS WITH AVIAN INFLUENZA

Diagnosis

Suspect cases of AI in birds must be immediately reported to DPIW via the Emergency Disease Hotline 1800 675 888. Subsequent action will be advised. **Non-veterinarians must not autopsy suspect birds.**

The diagnosis of AI is a multi-stage process. Detection of antibody or antigen is followed by procedures to confirm taxonomy and pathogenicity.

Pathology

Acute avian influenza produces pathology consistent with generalised haemorrhagic, necrotic, congestive and transudative changes. Haemorrhages (petechial and ecchymotic) may be found to a varying degree in most organs and at points of external contact such as the keel and ventral surfaces of the legs.

Veterinarians have been issued with specific protocols for processing suspect cases. A full set of fixed and fresh organs, tracheal and cloacal swabs and whole blood is required.

Prevention, treatment and control

Prevention in commercial poultry flocks is by limiting contact with wild birds, sanitising water supplies and preventing feed contamination. Similar precautions should be taken in all captive avian colonies.

Vaccination of birds is not a current option in Australia.

There is no treatment for AI infected birds.

In Australia, management of an HPAI outbreak is in accordance with the current Australian Veterinary Emergency Plan (AUSVETPLAN), Disease Strategy for Avian Influenza. Responses to outbreaks in wild birds are primarily concerned with enhancing public awareness and biosecurity and surveillance in commercial flocks.

Human health implications

Generally, AI represents a small risk to human health. There are notable exceptions. There is evidence that the 1918 human influenza pandemic was derived from an avian influenza virus (Taubenberger et al. 2005).

The risk of exposure to AI viruses is reduced by using appropriate protective equipment such as gloves and face masks and maintaining strict hygiene when handling birds.

Further information and references

Incidences of sick or dead wild birds must be reported according to current protocols. If in doubt report via the Emergency Disease Hotline 1800 675 888.

The Department of Primary Industries and Water has provided a simple checklist of measures that bird owners can take to reduce the risk of AI. Follow the links to Avian Influenza from www.dpiw.tas.gov.au

Latest information on the H5N1 outbreaks can be found on the World Organisation for Animal health (OIE) website at www.oie.int/eng/eng index.htm

or the Food and Agricultural Organisation's website at www.fao.org/AG/AGAInfo/subject/en/health/diseases-cards/special avian.html

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Notes:

Tuberculosis in Seals

Aetiology

The first confirmed cases of tuberculosis in seals were initially thought to be related to the bacteria *Mycobacterium bovis* (Forshaw and Phelps 1991). It was later shown that these seal isolates, now known as *Mycobacterium pinnipedii* are related to the *Mycobacterium tuberculosis* complex (Cousins et al. 2003).

Natural hosts

Pinnipeds appear to be the natural host for seal isolates of *M. pinnipedii* though the organism is also pathogenic in guinea pigs, rabbits, humans, Brazilian tapir (*Tapirus terrestris*) and possibly cattle (Cousins et al. 2003).

World distribution

M. pinnipedii has been identified from pinnipeds in Australia, Argentina, Uruguay, Great Britian, and New Zealand (Cousins et al. 2003).

Australian distribution

In 1986 *M. pinnipedii* was first confirmed in pinnipeds in a marine park in Western Australia. The organism has been isolated from captive and wild Australian sea lions (*Neophoca cinerea*), New Zealand fur seals (*Arctocephalus foresteri*), and an Australian fur seal (*Arctocephalus pusillus*), (Forshaw and Phelps 1991, Cousins et al. 2003). Disease in the captive colony was most likely introduced with wild pinnipeds. Woods et al. (1995) confirmed the organism in a wild Australian fur seal in Tasmania.

Epidemiology

Pinniped tuberculosis is most likely transmitted by aerosol as most seals develop pulmonary lesions. Liver lesions in one case suggest that alimentary infection may also be possible (Forshaw and Phelps 1991).

Little is known about the prevalence of tuberculosis in wild free-ranging seals. Opportunistic screening yielded nine animals sero-reacting out of 59 wild Australian fur seals from waters near Hobart using enzme-linked immunosorbent assay (ELISA) testing (Barrie Wells pers. comm.)

Clinical signs

Tuberculosis seals may exhibit clinical signs of respiratory embarassment (coughing and/or dyspnoea), lethargy and weakness (Woods et al. 1995). Some infected animals may exhibit no clinical signs however (Forshaw and Phelps 1991).

Diagnosis

In live, wild marine mammals an ELISA test using serum (Cousins 1987) and a polymerase chain reaction (PCR) test on nasal sputum may be the best screening and diagnostic tests (Woods et al. 1995). Intradermal tuberculin tests require two tests 72 hours apart and results may be misleading. Histopathology and culture can be definitive on post-mortem samples.

Pathology

Infection caused by *M. pinnipedii* is associated with granulomatous lesions in the lungs, pleura, spleen, peritoneum and peripheral lymph nodes (Cousins et al. 2003). Lesions have also been identified in the liver and a case of tuberculous meningitis has also been found (Forshaw and Phelps 1991).

Prevention, treatment and control

Disease prevalence surveys should be undertaken on marine mammals in captive populations, particularly new admissions from the wild. In addition opportunistic testing should be undertaken from stranded or captured seals. Monitoring is important because treatment and control are difficult and because of the zoonotic potential.

Human health implications

In 1988 a seal trainer who worked with the affected Western Australian colony for several years developed pulmonary tuberculosis (Forshaw and Phelps 1991). Though tuberculosis is a zoonotic disease, the risk of most humans (or other species) becoming infected is probably low due to rare and/or brief contact with marine mammals. Individuals at potentially higher risk include marine park staff, marine aquaculture industry workers (in regions where seals interact with aquaculture operations), marine / wildlife management and research staff, and associated veterinarians. Appropriate precautions such as

a disposable face mask and effective personal hygiene procedures should be taken by in-contact individuals.

Research needed

Further studies are needed into the prevalence and incidence of tuberculosis in wild pinnipeds.

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