**Haemosporidia and Australian wild birds**

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**Introductory statement**

Haemosporidia of birds (*Leucocytozoon, Haemoproteus*, and *Plasmodium* species) are single-celled two-host parasites that cycle between birds and their insect vectors. Infection with these parasites is common in many species of birds in Australia and around the world. Historically, these parasites have been considered to be host-adapted and to cause little disease in the species that they infected. However, as a result of intentional and accidental introduction of avian hosts and insect vectors into ecosystems where these parasites have not previously existed, infection in native bird species has occurred, at times with devastating consequences. The advent of molecular tools has allowed a better understanding of the epidemiology of haemosporidia. These tools are now being used to measure and predict the impact of climate and other anthropogenic impacts on the distribution of haemosporidia and wild bird health in Australia and globally.

**Aetiology**

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<td>Achromatoida</td>
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**Natural hosts**

Haemosporidian parasites of birds are two host parasites. They undergo sexual replication in their insect vectors and undergo asexual replication in their avian hosts. *Plasmodium* is transmitted between birds by mosquitoes (*Culicidae*), *Haemoproteus* by biting midges (*Ceratopogonidae*) and louse flies (*Hippoboscidae*), and *Leucocytozoon* by blackflies (*Simuliidae*) (reviewed in Bennett *et al.* 1993, La Pointe *et al.* 2012).

More than 200 species of haemosporidia have been described among the 4000 bird species investigated worldwide (reviewed in Bennett *et al.* 1993, La Pointe *et al.* 2012). Most species of *Haemoproteus* and *Leucocytozoon* are relatively host-specific and restricted to closely related species (reviewed in Bennett *et al.* 2012).
Infection can also occur in poultry species. Disease has been documented in North American ducks and turkeys and in chickens in Africa and Asia (Sehgal et al. 2006, reviewed in Bernudez 2008).

**World distribution**

Worldwide.

**Occurrences in Australia**

There have been few comprehensive studies of haemosporidia in Australia birds, but the ones that have been done show that infection is widespread in many species of birds, at least in Queensland (Adlard et al. 2004, Beadell et al. 2004, Heather et al., 2011, Zamora-Vilchis et al. 2012). The prevalence of infection in other areas of Australia will likely depend on environmental conditions such as temperature, humidity and rainfall that will in turn impact the numbers and distribution of the parasites insect vectors (Zamora-Vilchis et al. 2012). Given that Australia’s climate varies significantly from year to year, it is likely that parasite prevalence will also change from year to year in some geographic areas.

Deaths attributed to haemosporidial infections have be reported in a peregrine falcon (*Falco perigrinis*), nankeen kestrels (*Falco cenchroides*) (Raidal and Jaensch 2000), little blue penguins (Cannell et al. 2013), and pied currawongs (*Strepera graculina*) (Lederer et al. 2002).

**Epidemiology**

Birds are infected by the parasites’ sporozoites when they are bitten by an infected insect vector. These sporozoites enter host cells where they undergo asexual replication (schizogony) ultimately resulting in the rupture of the host cell with the release of meronts. This process is repeated until meronts are released that then penetrate circulating red blood cells where they mature into microgametocytes (male) and macrogametocytes (female). Plasmodium species can also undergo schizogony in red blood cells whereas the other two genera cannot. When ingested in a blood meal by the insect vector, the microgametocytes and macrogametocytes enter the insect’s gut where they fuse, penetrate the gut epithelium and undergo replication. Infected cells rupture and release sporozoites that then move to the salivary glands of the insect where they can then be injected into the next bird upon which the insect vector feeds (reviewed in Bennet et al. 1993, La Pointe et al. 2012).

Infections with multiple species and genera of haemosporidia are common (Adlard et al. 2004, Beadell et al. 2004, Heather et al. 2011, van Rooyen et al. 2013). Infection can occur during the breeding season or throughout the year. Migratory birds can be infected on the breeding grounds, wintering grounds or in both locations (Dodge et al. 2013, Hellgren et al. 2013). Parasite infections may persist for many years, but there is some thought that a moderate percent of infected birds will cure themselves of infection (van Rooyen et al. 2013). Proving this is difficult, as gametocyte’s presence and concentrations in the blood change over the course of the year (Hellgren et al. 2013). Persistent infections in the avian host allow the parasites to survive in between the seasons when there are no insect vectors.

Disease occurs in birds that are exposed to haemosporidia with which they have not co-evolved (reviewed in Bennett et al. 1993). For example, the introduction of birds carrying *Plasmodium relictum* and its mosquito
vector to the Hawaiian Islands has, in part, resulted in the loss of half of the native Hawaiian forest bird species (van Ripper et al. 1986). Disease can also occur when a species is moved from an environment where the parasite and the vector are not present to one where they are. Examples of this include the gyrfalcon (Falco rusticolus), which is an Arctic species that is highly sought after for falconry in warmer climates. The gyrfalcon, if housed in warmer climates without protection from biting insects, will rapidly become infected and die (Remple 2004). Similarly, many species of penguins, if housed outside in temperate climates will succumb to Plasmodium infections (Graczyk et al. 1994). Although Leucocytozoon, Haemoproteus, and Plasmodium species have all been implicated in disease outbreaks, Leucocytozoon and Haemoproteus have a narrower host range than Plasmodium, and Plasmodium is more likely to infect unrelated species and is more often associated with disease (reviewed in Bennett et al. 1993, Remple 2004).

Anthropogenic factors may also impact the prevalence of infection in different species. It has been postulated that changing temperatures associated with global warming may impact upon prevalence. For example, Hawaiian forest species may be impacted as warming temperatures will allow mosquitoes to live at the higher elevations on the Hawaiian Islands that are the last remaining refuges for these species (Hobbelen et al. 2012). Closer to home, research in the wet tropics of Australia suggests that global warming will result in an increased prevalence of haemosporidial infection in birds (Zamora-Vilchis et al. 2012). Another example of changing prevalence associated with anthropogenic factors is that of the pied currawong. Plantings in urban areas now provide year round food for this species attracting it to coastal areas where it has increased exposure to insect vectors (Major and Parsons 2010). This may explain the high prevalence of Haemoproteus infection in this species (Adlard et al. 2004) and its associated disease (Lederer et al. 2002).

**Clinical signs**

The bulk of haemosporidian infections occur in adapted hosts and outward disease does not occur (Bennett et al. 1993). However, subtle impacts including the quality of the feathers, reproductive success (reviewed in LaPointe et al. 2012), survivability of the infected bird in the coming year (Merino et al. 2000) and behaviour (Dunn et al. 2011) may be impacted by the presence of the parasite infection.

The primary target of the haematospordian parasite can be one or more of the lung, liver, spleen or brain and spinal cord (reviewed in Bennett et al. 1993, reviewed in La Pointe et al. 2012). Damage to the capillaries in the brain and other cells can result in neurologic signs including mentation changes, problems with balance and blindness. Disease in the eye may result in intraocular haemorrhage and blindness (Raidal and Jaensch 2000). Damage to the capillaries in the lungs can cause difficulty breathing (Remple 2004). Birds may be lethargic and, if experiencing anaemia, will be pale (Graczyk et al. 2004, Remple 2004). Red blood cell destruction results in the release of biliverdin in the urine staining the urates lime green or sulfur yellow in some cases. Liver enlargement is common and the enlarged liver may be felt when examining the coelomic cavity. Often infected birds are found dead with no premonitory signs (Cannell et al. 2013).

**Diagnosis**

**Clinical signs**

Signs exhibited by infected birds are not specific and diagnosis often requires a post mortem examination.
Clinical pathological findings

Species identification in the blood smear: Morphology of the gametocyte has been used to identify the haemosporidia found in the blood to the genus and species level. *Leucocytozoon* gametocytes are large and distort the cell that they infect to the point that it is unrecognizable. *Plasmodium* is the only haemosporidia that will undergo schizogony in red-blood cells, therefore if schizonts are seen in a blood smear, a *Plasmodium* infection can be confirmed. Unfortunately, schizonts are rarely found so their absence does not rule out a *Plasmodium* infection. *Plasmodium* and *Haemoproteus* gametocytes are sausage shaped structures located in the cytoplasm of infected red blood cells. Based on the species infected and fine details of the gametocyte, the genus and species of the parasite can often be determined by experts (reviewed in Bennett *et al.* 1993). However, it is increasingly clear that there is considerable cryptic diversity of haemosporidia and sequencing of DNA amplified from the parasite is now considered the most definite means of species identification (Perkins and Schall 2002, Krizanauskiene *et al.* 2006, Sehgal *et al.* 2006, Bensch *et al.* 2009).

Detection of subclinically infected birds: Subclinically infected birds can be detected by examining blood smears for cells containing the gametocytes or by using PCR assays on DNA extracted from blood (Sehgal *et al.* 2006, Krams *et al.* 2012). Both PCR assays and examination of the blood smear are highly specific for infection. They are, however, lacking in sensitivity. This is because the presence and number of gametocytes in the blood is not constant and their presence in the blood will change with season, the bird’s age and how recently the bird was infected (Jarvi *et al.* 2002). As a result, many subclinically infected birds will not have gametocytes in the blood. In contrast, many other subclinically infected birds will have significant numbers of gametocytes in the blood and yet exhibit no signs of illness and the number of infected blood cells cannot consistently be correlated with the disease status of the bird. Both PCR assays and blood smear examination have similar sensitivities but often are not identical (Waldenström *et al.* 2004, Valkiunas *et al.* 2006, Valkiunas *et al.* 2008, Sehgal and Bensch 2008, Krams *et al.* 2012).

Diagnosis of haemosporidial disease (Haematology): Disease caused by these parasites is predominately the result of damage done to internal organ systems in the early phase of infection. At this time, gametocytes may or may not be present in the blood, so their absence cannot be used to rule out infection (Raidal and Jaensch 2000, reviewed in LaPointe *et al.* 2012). Anaemia is a common finding in birds infected with *Plasmodium* and a less frequent finding in birds infected with *Leucocytozoon* and *Haemoproteus* (reviewed in Remple, reviewed in LaPointe *et al.* 2012). Elevation of the white blood cell count has been observed in some cases but not in others. In penguins a relative lymphocytosis has been observed in birds that survived infection, but not in those that died as the result of infection (Gracz *et al.* 2004).

Diagnosis of haemosporidal diseases (Serum/plasma biochemical changes): In penguins, significant increases in gamma glutamyl tranferase, alanine aminotransferase and creatinine were seen in birds infected with *Plasmodium* as compared to those that were not. These changes, however, did not occur in all infected birds, are not specific and were not seen in birds that died quickly as the result of severe infection (Graczyk *et al.* 1995).

Pathology

Gross postmortem changes: The most common gross post mortem changes are enlargement of the liver and spleen. Multiple pale tan foci may be found on the serosal and cut surfaces of these organs. In cases where there is extensive haemolysis, the liver will be very dark brown to black. If parasite replication is occurring in
the lungs, then they may be moist and have reduced buoyancy (reviewed in Bennett et al. 1993, Raidal and Jaensch 2000, reviewed in LaPointe et al. 2012).

Microscopic changes: Impression smears of diseased organs stained with quick stains will often demonstrate cells containing schizonts. Schizonts can be present in one or more organs including kidney, liver, spleen, lung, heart, skeletal muscle, brain, spinal cord and eye. They are found within the cytoplasm of the infected cell, are multinucleate, range in size from 20 µm to 2.5 cms and severely distort the cells that they infect. Infected cells include vascular endothelial cells, macrophages, histiocytes, hepatocytes, renal tubular epithelial cells and myocytes (Earle et al. 1993, reviewed in Bennett et al. 1993, Atkinson et al. 2000, Raidal and Jaensch 2000, reviewed in LaPointe et al. 2012). Infected vascular endothelial cells can block capillaries and result in thrombus and embolus formation (Peirce et al. 2004). Damage to capillaries may also result in haemorrhage (Raidal and Jaensch 2000, Donovan et al. 2008).

Host reaction to infection can include multifocal necrosis of the liver, spleen, and kidney that may range from mild to severe. Degeneration of the heart muscle and skeletal muscle may occur when these organs are targeted. Capillary leakage in the lungs can result in pulmonary oedema and release of fibrin into the airways. Interstitial pneumonia may develop in severe cases (Earle et al. 1993, reviewed in Bennett et al. 1993, Atkinson et al. 2000, Raidal and Jaensch 2000, reviewed in LaPointe et al. 2012). A proliferative arteriopathy of the brain stem, optic nerve and the pectin were observed in a haemosporidial infection of a peregrine falcon and nankeen kestrel (Raidal and Jaensch 2000). Inflammatory responses range from none to severe. When inflammation occurs it is predominately monocytic and lymphocytic, with a lesser degree of a heterophilic response (Raidal and Jaensch 2000, reviewed in LaPointe et al. 2012, Cannell et al. 2013). A pronounced haemosiderosis of the spleen and liver may occur in birds that were experiencing haemolysis (reviewed in LaPointe et al. 2012).

**Differential diagnosis**

Signs, in most birds, are not specific and can be caused by many different diseases. The primary differential diagnosis for anaemia would be blood loss due to trauma, external or internal parasitism or bleeding due to damage to the intestine by a diffuse coccidial disease. Birds with chronic wasting diseases are often anaemic as well due to a failure of red blood cell production. Neurological signs can also be caused by trauma, many intoxicants including heavy metals, nutritional deficiencies and bacterial and viral infections of the brain. Respiratory signs can also be caused by trauma to the lungs or heart, inhaled toxins, vitamin A deficiency, and bacterial, viral and fungal infections of the respiratory system.

**Laboratory diagnosis specimens**

In the live animal, blood smears should be made from fresh blood to which anticoagulants have not been added. Samples that can be used for PCR assays included dried blood smears and blood for which the anticoagulant EDTA has been used. Schizonts can be found in impression smears made from liver, kidney, spleen, lung and brain and PCR can be done on any of these tissues if they are fresh or have been frozen. A complete set of tissues including brain, heart, skeletal muscle, liver, kidney, spleen, and lung, should be fixed in formalin and submitted for histopathology.
Laboratory procedures

Blood smears and tissue impression smears are alcohol fixed and can be stained with quick stains or Giemsa’s stain for direct microscopic observation of gametocytes in the blood and schizonts in impression smears. Schizonts are readily observed in haematoxylin and eosin stained sections of tissues.

Multiple PCR protocols have been developed to detect haemosporidia DNA in blood and tissues (reviewed in Bensch et al. 2009). Depending on the primer sets used, these assays can be used to amplify DNA from one or more genera. Identification down to the species level requires sequencing of the amplified DNA. Sequences of the mitochondrial cytochrome B gene have been used in most cases for species identification (Bensch et al., 2009).

Treatment

Most work on treatment has been done in birds of prey. Currently, a drug regimen using both chloroquine (Aralen, Sanofi-Synthelabo) and primaquine is advocated and has been found to be effective in curing infection with *Plasmodium* and *Haemoproteus*. Mefloquine hydrochloride (Larium, Hoffmann-LaRoche) has also been used to treat *Plasmodium* in falcons and may only require a single treatment to be effective (reviewed in Remple 2004). Successful treatment of *Leucocytozoon* has not been reported.

Prevention and control

Prevention of infection in wild birds is virtually impossible (Hobbelen et al. 2012). Preventing infection in captive birds is best done by building flying insect proof enclosures. In birds of prey weekly treatment with chloroquine and primaquine will also prevent the establishment of infection (Remple 2004).

Surveillance and management

Wildlife disease surveillance in Australia is coordinated by Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia’s States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au.

There is no targeted surveillance program for haemosporidia in Australian wild birds. However, cases may be logged in the National Wildlife Health Information System (eWHIS) as part of Australia’s general wildlife surveillance activities.

Statistics

There are two surveys where significant numbers of apparently healthy birds have been surveyed for blood parasites in Australia. In the first, 219 birds, most of which were passerine species, were sampled. More than 5 birds were sampled from each of 19 species and of these species 17 (89%) were infected with one or more haemosporidia. Of the total number of birds samples 28% were infected with *Haemoproteus* and 14% *Plasmodium* (Beadell et al. 2004). In the second study, 403 birds were sampled and 130 (32.3%) were infected with a haemosporidia (Zamora-Vilchis et al. 2012) with a 19.9% prevalence of *Haemoproteus*. 
infection, 6.2% prevalence of *Leucocytozoon* infection and 1.74% prevalence of *Plasmodium* infection. In the remainder of infected birds the genera of the species could not be determined.

A third study examined samples from birds from southeast Queensland that were submitted to a wildlife hospital for various causes (Adlard et al. 2004). The number and variety of bird species in this study was higher than the other Australian studies. Of a total of 3059 individual birds samples 11.4% were infected with a haemosporidia. *Leucocytozoon* were found in 51.1% of infected birds and *Haemoproteus* in 31.4% of infected birds. *Plasmodium* was found in 10.9% of infected birds. Species that were commonly infected with haemosporidia (prevalence > 25%) included the boobook owl (*Ninox boobook*), tawny frogmouth (*Podargus strigoides*), Torresion crow (*Corvus orru*), Australian magpie (*Gymnorhina tibicen*), pied currawong, grey butcher bird (*Cracticus torquatus*), pigeon (*Columbia livia*), Australasian figbird (*Sphecotheres viridis*), noisy miner (*Manorina melanopygia*), and bell miner (*Manorina melanophrys*).

**Research**

Relatively little is known about the prevalence and distribution of haemosporidia temporally and geographically in Australian birds outside of Queensland. Coordinated studies of birds caught for banding and those submitted to wildlife carers could be used to expand this knowledge base. There is also a need to characterize these parasites genetically, particularly the parasites from diseased birds to determine if they represent parasites that have evolved in Australia with Australian birds or are introduced species. Preliminary data suggests that some Australian parasites were introduced by invasive species (Yim 2009).

**Human health implications**

There are no human health implications.

**Conclusion**

Haemosporidial infections are common in many species of Australian birds. Most infections do not cause disease or the disease that they cause is minimal. Disease, however, has been infrequently documented in a few native Australian birds. Factors such as climate change, expansion of the range of some species, and possibly the introduction of new haemosporidial species into Australia may have contributed to these outbreaks, may predispose to an increased prevalence of infection and additional outbreaks of disease in the future.

**References and other information**


Lederer, R., Adlard, R.D., O’Donoghue, P.J., (2002), ‘Severe pathology associated with protozoal schizonts in two Pied Currawongs (Strepera graculina) from Queensland,’ Veterinary Record, 150, 520-522.


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**To provide feedback on this fact sheet**

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

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