

Hygiene Protocols for the Prevention  
and Control of Diseases  
(Particularly Beak and Feather Disease)  
in Australian Birds

Chlamydophilosis



**Australian Government**

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**Department of the Environment and Heritage**

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# Chlamydophilosis

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Chlamydophilosis is also known as parrot fever and psittacosis (psittacine birds), ornithosis (non-psittacine birds) and psittacosis (humans). It is caused by *Chlamydophila psittaci*, formerly known as *Chlamydia psittaci*. Thus the disease that we currently call chlamydiosis and caused by *Chlamydia psittaci* is now chlamydophilosis and caused by *Chlamydophila psittaci* (Everett *et al.*, 1999).

Avian chlamydophilosis is a significant bacterial disease of wild, captive and intensively reared birds. The agent responsible, *Chlamydophila psittaci*, is an obligate intracellular parasite.

*C. psittaci* is a significant zoonosis and the term "psittacosis" has been used to describe the disease in humans to distinguish psittacosis from the venereal disease caused by *C. trachomatis*. Avian chlamydophilosis (AC) may range from a rapidly fatal peracute disease to a subclinical latent infection, depending on host factors and the strain of *C. psittaci* involved. The body systems primarily affected include the respiratory and gastrointestinal tracts (including the liver), the cardiovascular system, the spleen and the eyes. Significant mortality and morbidity may occur as a result of the disease, particularly in parrots and turkeys.

## **Structure and Life Cycle**

Chlamydiae are spherical intracytoplasmic organisms 0.2 µm to 1.5 µm in diameter (depending on their stage of development). They have a cell wall similar to Gram-negative bacteria and they parasitise energy (ATP from the host's mitochondria). The two major morphological stages in the life cycle of chlamydiae are termed the elementary body (EB) and the reticulate body (RB). The EB is the smaller infectious form (0.2-0.3 µm diameter) characterised by a thick, rigid cell wall and very dense cytoplasm. It may be found outside the host cell. The RB is larger (0.6-1.5 µm), has a thinner cell wall, less dense cytoplasm, and is the vegetative stage which reproduces by binary fission. Different strains of *C. psittaci* are recognised on the basis of antigenic and pathogenic differences. Avian strains of *C. psittaci* are distinct from mammalian strains (Moulder, 1985)

## **Epidemiology**

All bird species including domestic poultry are susceptible. Turkeys are very susceptible, chickens are relatively resistant. Surveys of avian chlamydophilosis in feral pigeons have revealed carrier rates of 50 - 90%. High rates of infection are commonly reported in psittacine birds and in some zoos the rate may reach 100%.

Chlamydophilosis is often introduced into an aviary by infected birds which die several days later (often with uncertain signs) or which infect other birds in the aviary, which die two to three weeks later. The incubation can be very short, birds may begin shedding chlamydia within a few days of being infected or it can be very long.

In natural hosts, chlamydial strains are thought to be "host adapted", i.e. the host-parasite relationship has had time to evolve towards an equilibrium state. Thus, in its "normal" host, a chlamydial strain may be relatively avirulent and remain latent unless the bird is stressed, whereas in an abnormal host the same strain may be highly virulent and cause epizootic disease. Overt disease in the normal host may be induced by stress factors such as poor nutrition and hygiene, overcrowding, bacterial or protozoal disease,

shipping, racing, migration, breeding, inclement weather or moulting. Prevalence of chlamydophilosis in native birds can rise from a normal value of less than 5% to 100% when they are trapped and crowded together.

Chlamydophilosis is common in wild Eastern Rosellas and Crimson Rosellas in NSW and Victoria during winter. Periodic outbreaks occur in aviary birds where the disease is endemic and the birds are undergoing their first moult (particularly the *Neophema* species). Often there is a history of stress such as moulting, surgical sexing and overcrowded transport.

Young birds are more susceptible to chlamydial infection because they are immunologically immature and are often exposed to the organism excreted by the parent birds due to the stresses associated with breeding.

In wild and captive flocks with endemic chlamydophilosis the majority of birds carry latent infections. Mortality and morbidity is highest amongst the young, however, losses generally don't exceed 20%. In contrast, where infective organisms are introduced to disease-free flocks, mortality may approach 90%. The latter scenario occurs most commonly where wild infected birds are able to mingle with naive domestic poultry flocks or where new birds are introduced to existing stock without adequate prophylaxis.

*C. psittaci* is excreted in the faeces and nasal discharges of infected birds. The organism is resistant to drying and can remain infectious for several months. Some infected birds can appear healthy and shed the organism intermittently. Shedding can be activated by stress factors, including relocation, shipping, crowding, chilling, and breeding.

### ***Pathogenesis***

Transmission of *C. psittaci* occurs most commonly by inhalation (less often by ingestion) of infective EBs shed in faeces, lacrimal and nasal secretions and respiratory exudates. Infected birds may transmit chlamydia by regurgitative feeding of their young. Transmission may occur via consumption of infected carcasses by predatory birds. Arthropod vectors such as lice, mites and simuliid flies can transmit chlamydia (Eddie *et al.*, 1962).

Dissemination is favoured during periods of stress (e.g. poor nutrition, overcrowding, concurrent disease, shipping, racing and migration) due to activation of latent infection followed by excretion of large numbers of infective organisms - with or without the development of disease.

Behavioural traits which favour or enhance transmission include:

- colonial nesting, e.g. amongst herons, egrets, cormorants, pigeons and sparrows.
- regurgitation feeding of young by parents
- certain natural feeding strategies which promote aggregations of birds in a potentially contaminated environment.

*Chlamydophila psittaci* has a predilection for cells of the respiratory tract, serous cavities and reticuloendothelial system, especially mononuclear phagocytes. Multiplication of chlamydiae results in cell lysis and this combined with the host's inflammatory response causes the clinical manifestations of avian chlamydophilosis - conjunctivitis, rhinitis, airsacculitis, pericarditis, hepatic and splenic necrosis and arteritis. Enteric infections are common in most avian species. Excretion of infective EBs occurs in faeces and diarrhoea is a common clinical sign.

Chlamydial infections elicit both humoral and cell mediated immune responses. Acute fatal disease and recurrent subacute attacks occur, but there is a tendency toward chronic latent infections. The acute inflammatory reaction evoked by chlamydial invasion probably contributes to the pathogenesis of disease

by producing secondary injury to tissues.

Latency is an equilibrium between the host's immune defences and the pathogen's intracellular persistence. The pathogen is quiescent - present but not multiplying, while the host remains infected and susceptible to future episodes of disease should its immune defences be impaired.

Differences in pathogenicity of certain strains for certain species of bird may be related to the degree and mode of exposure, the route of infection and the hosts innate resistance which in itself is subject to physiological and environmental influences. After aerosol inoculation the organism multiplies in the lung, air sacs and pericardial membrane. By 48 hours organisms can be detected in blood, liver, spleen and kidney cells and after 72 hours there is excretion of organisms in the faeces and nasal secretions.

### ***Clinical Signs***

The clinical signs of chlamydophilosis may be indistinguishable from several other febrile septicaemic diseases of birds and differential diagnoses should include salmonellosis, tuberculosis, erysipelas, mycoplasmosis, pasteurellosis, *E. coli*, and aspergillosis. The usual time between exposure to *C. psittaci* and onset of illness ranges from 3 days to several weeks. However, active disease can appear years after exposure. Affected psittacine birds often have distended sinuses, blepharitis, sneezing, serous oculonasal discharge, dyspnoea, tail-bobbing, green diarrhoea, depression. Sudden death may be the only history (Vanrompay, 1995; Johnston *et al.*, 1999)

In affected turkey flocks, birds may be thin and anorexic, pyrexia and have yellow-green diarrhoea. There may be a rapid drop in egg production (up to 40%).

Young ducks with acute chlamydophilosis are depressed, anorexic, ataxic, and have diarrhoea and a serous to purulent oculonasal discharge. There may be terminal convulsions. Ducks with chronic chlamydophilosis may just appear emaciated and in poor health.

Acute cases in chickens involve mainly cardiovascular and gastrointestinal signs, with low mortalities and only in young.

Acute disease in pigeons presents as anorexia, ill-thrift, diarrhoea, weakness, conjunctivitis, blepharitis, rhinitis, creaking and rattling respiratory sounds. Chronic disease - weak, thin, emaciated. May have transient diarrhoea with mild infections.

### **Lesions**

Most birds which die of acute chlamydophilosis have marked splenomegaly and hepatomegaly but the presence of fibrinous or fibrino-purulent exudates on serosal surfaces, as well as congested organs such as liver and spleen is sufficient to initiate confirmatory diagnostic tests. These include cytological examination of smears; culture; and immunological tests. Histopathology is unreliable for confirming a diagnosis. Cytology is often more reliable. Elementary bodies may be visualised in liver and spleen smears with appropriate stains (Macchiavello, Giemsa, Gimenez, Castenada) within macrophages in affected tissues. Fluorescent antibody stains are a rapid diagnostic test (the smear should be dried and then fixed in acetone).

Tentative diagnoses can be made on the basis of seeing EBs in stained tissues but because of their similarity to some other bacteria e.g. mycoplasmas, diagnosis should be confirmed by demonstration of chlamydial growth in experimental hosts after inoculation, demonstration of chlamydial antigen or significantly rising titres of antibody.

There is no single test or combination of tests which will determine that a bird is free of chlamydiae.

### ***Methods for diagnosing chlamydophilosis***

Diagnosis is by clinical signs supported by tests such as the Clearview<sup>®</sup> test (Oxoid) which detect chlamydial group specific antigen (designed for *C. trachoma*). The Clearview<sup>®</sup> test has limitations for antemortem use. These tests have a high sensitivity and lower specificity particularly if used on faecal or cloacal swabs due to cross-reactions with other antigens (Fudge, 1997). Other methods of diagnosis include histochemical staining, tissue culture and serology such as the Immunocomb<sup>®</sup> test (Flammer, 1997).

Combining a PCR test with a serologic titre offers the most thorough diagnostic plan (Tully, 2001).

### ***Treatment***

*Doxycycline*, is highly recommended for the treatment of acutely ill birds and has produced the most consistent therapeutic results in most birds. It is lipophilic which results in tissue concentrations higher than other tetracyclines. Its half life is 22 hours as opposed to 8 hours for tetracycline, and absorption from the gut is rapid, almost complete (95% vs 25% -80% for other tetracyclines) and subject to less interference from calcium. A major advantage of this drug is that it has less of an adverse effect on normal gut flora than other tetracyclines due to its rapid absorption and excretion as an inactive conjugate in the faeces. The incidence of secondary infections while on medication is reduced. Intravenous dose route of 10-100 mg/kg body weight for 1 to 2 doses, followed by oral administration (5-25 mg/kg, per os BID) for 45 days.

*Psittavet (Vetafarm)* - doxycycline hydrochloride 40 mg/g green powder). Dose: for parrots 10 g/l drinking water.; for pigeons 5 g/l drinking water for 45 days. Add citric acid 125 ppm for potentiation.

*Doxyvet* - The Australian Pigeon Company. Doxycycline hydrochloride 120mg/g. Dose 1.5 g/L in DW for birds.

### ***Supportive and adjunctive therapy***

- Elimination of concurrent parasitic, bacterial and fungal infections. Candidiasis can be treated with nystatin. Supplementary lactobacillus can be fed (psittacine isolate).
- Fluid and electrolyte therapy is advised for depressed and dehydrated birds. Warmed lactated Ringers administered subcutaneously at 5 - 10 mls/100 g BW is effective.
- Hypoglycaemic birds should be supported with intramuscular dextrose.
- Anorectic birds will require tube feeding.
- New birds should not be introduced to the aviary/flock without first being isolated and put on a 45 day course of prophylactic medication
- Sick birds should be isolated in a small enough cage to prevent excessive flying, kept warm and uncrowded (preferably 1 or 2 birds to a cage) in surroundings that are cleaned and disinfected daily.
- All surfaces with which infective organisms may come in contact should be cleaned and disinfected with 2% Virkon S solution.

### ***Prevention and Control***

Prevention and control of avian chlamydophilosis is reliant on isolation and treatment of affected birds, quarantine and prophylactic treatment of potentially infected birds and detection of carriers of the disease.

The source of infection of a flock should be identified where possible and further contact with infective organisms prevented. Contact between potentially infected wild birds and their droppings and poultry should be prevented.

Affected birds should be isolated and treated with doxycycline under sanitary conditions with minimum stress to the birds. The rest of the flock should be periodically monitored for several months after an outbreak to determine if infection has spread. It is wise to treat the rest of the flock prophylactically, as diagnosis of subclinical carriers is not very efficient.

Quarantine and treatment of new birds prior to introduction to a flock should last at least 45 days, depending on the treatment regime for the species involved. Provided there is no direct or indirect contact with wild birds, aviculturists should be able to maintain chlamydia free premises by instituting a single yearly 45 day course of CTC in the feed for all resident birds. Stress associated with handling, transport, housing and nutrition should be avoided and bird owners are advised to buy birds directly from breeders with known healthy stock. Anti-chlamydial prophylaxis pre- and post-shipment should be mandatory.

Doxycycline doses of 50 mg/kg may cause regurgitation in some psittacine birds (Carpenter, 2001).

Owners should be counselled that introduction of new birds can reinfect the original stock and they should be advised on appropriate preventative measures. Owners that present sick birds should be advised to seek referral to an infectious disease expert. Humans should not be treated prophylactically. Breeders and dealers should be acquainted with the clinical signs.

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