Gastro-intestinal and Respiratory Parasites Infecting Psittacine Birds in Australia

Tanya McKeon*

Introduction

Many parasites are known to commonly infect psittacine birds in different states of Australia. However, there is a lack of written data of such infections making the diagnosis and identification of parasitism difficult for veterinary practitioners. The following is a summary of the available literature relating to confirmed reports of parasites infecting psittacine birds in Australia. Significant reviews include those by Steiner and Davis (1981), Keymer (1982), and Burr (1987).

Flagellated Protozoan Parasites

Trichomonads

Most species of trichomonads are not considered pathogenic to their hosts (Honigberg 1978) except *Trichomonas gallinae*, which infects the upper alimentary tract of a wide variety of avian species. This organism causes the disease known as trichomoniasis in infected birds (Honigberg 1978) and is a recognised pathogen.

Originally *T. gallinae* was isolated from pigeons (*Columbia livia)* and is currently considered a problem in commercial squab operations and racing pigeons. Natural infections also occur in dove, turkey and chicken species. The disease is known as "Frounce" in birds of prey and thought to be contracted by feeding these birds infected pigeons.

T. gallinae is a protozoan parasite which reproduces by longitudinal binary fission and exists in the form of a trophozoite. Sexual stages, cysts or vectors do not occur in the life-cycle (Kemp 1978). *T. gallinae* ranges in size from 2-9 x 5-19µm with four anterior flagella and a posterior flagellum (Kemp 1978). Whilst sharing these characteristics with *T. gallinarum*, a trichomonad that infects the caeca of gallinaceous birds, *T.gallinae* is distinguished by the fifth flagella being attached to an undulating membrane for three quarters of it's length. Special stains can also distinguish the two avian strains of *Trichomonas*.

Sporadic reference has been made to infections in psittacine birds, including an early report from Callender and Simmons (1937) on the experimental infection of a Tovi parakeet (*Brotogeris juglaris*) with *T. gallinae.* Blackmore (1965) also recognised the significance of budgerigars (*Melopsittacus undulatus)* being infected by pigeons and fowls under experimental conditions. In 1970, Ahmed *et al.* documented the first natural infection of *T. gallinae* in psittacine birds but did not specify the species involved, whilst more recent works have confirmed natural infections in budgerigars in which vomiting was seen as the only clinical sign (Baker 1986); in a blue-fronted amazonian parrot (*Amazona aestiva)* in 1990 by Garner and Strurtevant, and in cockatiels (*Nymphicus hollandicus*) (Murphy 1992). Other accounts of psittacine

* **Veterinary Biology, School of Veterinary Studies, Murdoch University, Perth, W.A. 6150**

bird infections are given by Murtaugh *et al.* (1984); Ramsay *et al.* (1990) and Emanuelson (1993).

In contrast to feral pigeon populations where up to 80-100% of a population may by asymptomatic carriers of *T. gallinae* (Kocan and Knisley 1970),psittacine bird populations reportedly suffer a high morality rote from *T. gallinae* infections with flock losses of up to 72% with an 80% morbidity rate (Murphy 1992).

In pigeons the characteristic caseous lesions associated with trichomoniasis can often be seen in the mouth. This is rarely the case in psittacine birds. In young psittacine birds the build up of caseous material may occlude the oesophagus and pharynx causing death by asphyxiation. Such birds can die within 2-3 days of infection whereas older birds may die within 2-3 weeks (Emanuelson 1983).

The physical signs of trichomoniasis in psittacine birds have been recorded as; loss of weight, diarrhoea, vomiting, and the appearance of a greenish yellow fluid in the mouth. Soiled head and breast feathers were also related to vomiting.

Diagnosis of *T. gallinae* infections can be made by the aspiration of crop contents and identifying the motile flagellated organisms, and by identifying the characteristic lesions in the upper digestive tract at postmortem examination. False negative results can occur when diagnosing from wet mount examinations, which Murphy (1992) attributes to very few organisms being present in the crop in the early stages of the infection. Previously, where direct examination of throat swabs were used to test for *T. gallinae,* 20% of negative samples were later proved positive by culturing (Kocan and Knisley 1970).

As *T. gallinae* does not utilise cysts or intermediate hosts in it's life-cycle it is not surprising that the organism is regarded as a very hardy parasite (Honigberg 1978). In culture, Honigberg maintained living organisms in tap water for two hours at 47°C with 100% recovery of the parasite and also maintained the organism for 5 days at 5-10 degrees with the recovery rate dropping to 25% after 2 days.

Given this information, and the fact that the organism is known to be transferred orally, water contamination in aviaries is the most likely cause of the spread of *T gallinae* within a flock. This is supported by Miessner and Hansen (1936), who isolated and cultured *T. gallinae* from a water container in an infected pigeon loft.

Opinion is divided as to the likelihood of faecal contamination with *T. gallinae*. Some authors claim that the organism is not found beyond the proventriculus in the bird and therefore is incapable of contaminating faeces (Kemp 1978), whilst others including Honigberg (1978) have found that virulent strains migrate to other organs via the blood including the lungs and heart. Under these circumstances the organisms may enter the lower digestive system and possibly contribute to faecal contamination, although no proof of this exists.

Treatment has been successful with metronidazole at a dose of 30mg/kg given orally, twice a day for 10 days (Murphy 1992). Applying treatment just before the breeding season has been suggested to prevent the organism being passed on to the young (Pass and Jakob-Hoff 1987). Current medications available in Australia include Ronivet® (Vetafarm) (Ronidazole), Emtryl® (Rhone Merieux) (Dimetridazole), Spartrix® (Janssen) (carnidazole) and Metrin®(Parnell Lab.Pty.Ltd.)(Metronidazole).

Diseases that could be confused with trichomoniasis include hypovitaminosis A, avian pox, tuberculosis, salmonellosis and candidiasis (Emanuelson 1983).

Giardia

In a study conducted in Japan by Tsai *et al*. (1992) *Giardia* was found to be one of the two most common parasites infecting aviary birds in that country. Infections have also been recorded in psittacine birds in

America and England(Panigrahy *et al.* 1981; Jones and Carrol 1977 and Schollens *et al.* 1982). Although not presently reported in psittacine birds in Australia, *Giardia* has been isolated from straw-necked ibis (*Threskiornis spinicollis)* in Western Australia. The ability of this species to infect psittacine species is still unknown (Forshaw *et al*. 1992).

The species of *Giardia* that have been isolated from psittacine birds include *Giardia intestinalis,G. duodenalie* and *G. lamblia,* the species infective to humans. Recently Erlandsen and Bemrick (1987) have also named a new species isolated from budgerigars as *G. psittaci*.

Giardia is a relatively small binucleate protozoan parasite measuring approximately 14 x 6µm and dividing by longitudinal fission similar to *T. gallinae*. The parasite exhibits distinct morphological characteristics when seen under the microscope as a pear shaped organism with two nuclei resembling “eyes”.

Infections are obtained by the ingestion of infective cysts that are passed out of the host in the faeces, where, in moist conditions they can remain viable for up to three weeks (Keymer 1982). It has been reported that as few as ten *Giardia* cysts are required to infect the host (Evans and Carey 1986).

When swallowed, trophozoites are liberated from the cysts into the small intestine where they migrate to the large intestine to complete the life-cycle.

The mortality rate in psittacine birds due to *Giardia* infections can range from 20-70% (Keymer 1982), following chronic and recurrent diarrhoea, lethargy, anorexia and weight loss. Panigrahy *et al*. (1981) attributed a much higher mortality rate of 90-100% in infected budgerigar nestlings.

Diagnosis of infection can be made by identification of thick-walled oval-shaped cysts in **fresh** faeces (Keymer 1982). At postmortem the organisms can be isolated from the intestine as motile flagellated protozoans associated with the epithelium.

Treatment of *Giardia* infections in psittacine birds has included the use of dimetridazole and adequate hygiene is recommended to prevent re-infection through faecal contamination (Panigrahy *et al.* 1981).

Hexamita

Hexamita meleagridis is a flagellated protozoan parasite which commonly occurs in poultry. Recently, infections have been reported in cockatiels and a splendid grass parrot *(Neophema splendida)* (Harper 1991) and in King Parrots *(Alisterus scapularis)* (Raidal pers.com; Macwhirter 1989). *Hexamita columbae* has also been recorded in pigeons in the United States.

Hexamita species are characterised as measuring 9x3 µm, being bilaterally symmetrical with eight flagellae, six anterior and two trailing, and showing rapid straight line movement when viewed under a microscope (Keymer 1982). *Hexamita* produces cysts that are passed in the faeces and may remain infective for several weeks in moist conditions (Madill 1987)

Hexamita infections are mainly confined to the upper digestive tract where they may be confused with *Trichomonas* or *Giardia* infections.

The clinical signs of *Hexamita* infections are emaciation and watery diarrhoea. Post-mortem diagnosis is difficult as the trophozoites rapidly degrade following host death

Treatment in pigeons and cockatiels has been given as a single dose of carnidazole (10mg) followed by ronidazole (10% soluble) at 5g/ 4.5L for 7 days (Harper 1991). Heavily infected cockatiels have also

initially been given 2mg of carnidazole directly into the crop (Harper 1991).

Coccidia

There are a large number of coccidian species known with varying degrees of host and site specificity. Despite this, information is currently limited concerning coccidian infections in caged-birds, particularly psittacine birds.

In 1960, Farr described a coccidian parasite isolated from a budgerigar. Other authors have also described coccidian infections in an alexandrine parrot (*Psittacula eupatria hipalensis*) (Chakravarty 1947), a rainbow lory (*Trichoglossus haematodus)* (Varghese 1977), a yellow fronted amazon parrot (*Amazona ochrocephala)* (Hooimeijer not pub.) and a Princess Wales parrot (*Polytelis alexandrae)* (Hooimeijer et al.1993). Further cases have been recorded in a peach faced love-bird *(Agapornis roseicollis)(* Keymer 1967 not pub.), and recently Panigrahy *et al*. (1981) has reported three outbreaks of coccidiosis in budgerigars in Texas.

The life-cycles of coccidian species are similar, with birds becoming infected by ingesting oocysts,which on arrival in the intestines release sporozoites into the lumen that invade the epithelial cell lining (Keymer 1982). Once inside the epithelial cell the sporozoite becomes a trophozoite, further developing into a mature schizont containing numerous merozoites.
On rupturing the merozoites may further invade other epithelial cells until reaching the sexual phase that produces oocysts which are consequently passed in the faeces.

Characteristically coccidian oocysts are very resistant and easily identified when examining faecal material.

The clinical signs of coccidiosis include enteritis or watery, bloody faeces. Weight loss may also be associated with chronic diarrhoea (Burr 1987). Death may occur if the infection is heavy and sustained over several months. Sudden deaths seldom occur.

Hooimeijer *et al*. (1993) reported the successful treatment of coccidiosis in lorikeets with Toltrazuril (Bayer®) administering 7mg/kg body weight for 2-3 days as is recommended for poultry species.

The coccidian species directly attributed to causing coccidiosis in psittacine birds have been *Eimeria, Sarcocystis, Toxoplasma and Cryptosporidium. Isospora* infections have been found to be pathogenic in canaries and passerine birds (Gray 1936) although there are no references to clinical signs being found in psittacine birds.

Eimeria

Very little information is available concerning *Eimeria* infections in psittacine birds although Todd *et al*. (1977) isolated *Eimeria dunsingi* from naturally infected budgerigars and Vargese (1977) isolated *E.haematodi* from wild lorikeets in New Guinea. Recently, Panigrahy *et al.* (1981) also recorded a case of coccidiosis in budgerigars that presented with haemorrhagic enteritis characteristic of *Eimeria* species.

The sporulated oocysts of *Eimeria* are known to contain four sporocysts, each containing two sporozoites (Burr 1987), with the oocysts recorded as measuring 25-36µm x 22-28µm (Todd *et al.*1977). Todd *et al.* (1977) also commented that, in budgerigars, little or no inflammatory response could be seen around the parasites in the small intestine.

Treatment is with Sulfamethazine (0.1-0.2 % for five days is reportedly affective) (Panigrahy *et al.* 1981).

Sarcocystis

Sarcocystis falcatula has a wide intermediate host range including Psittaciforme, Passeriforme and Collumbiforme birds with the documented definitive host being the opossum. Specific infections with this parasite have been recorded in the crimson rosella, *(Platycercus elegans*) eastern rosella, *(P. eximus)* red-rumped parrot (*Psephotus haematonotus*) and the mulga parrot (*Psephotus varius) (*Munday *et al*.1979).

In an Australian study in 1979, *Sarcocystis* was found in 105 out of 832 avian muscle samples (Munday *et al.* 1979). Jacobson *et al.* (1984) have also described a case of encephalitis in cockatiels caused by *Sarcocystis.* Recently in Australia *Sarcocystis* has also been recorded in the budgerigar (Reece *et al.* 1992).

Sarcocystis produces sporulated oocysts containing four sporozoites. Further life-cycle characteristics of *Sarcocystis* in Australia are not known as the definitive host has not been identified.

Munday *et al.* (1979) suggested that *Sarcocystis* may exist in Australia in a predator-prey relationship with raptors and smaller prey birds. Ground eating psittacine birds may then become accidentally infected by feed contaminated by raptor faeces.

Toxoplasma

Toxoplasmosis has been recorded in several psittacine species with accounts of natural *Toxoplasma gondii* infections being widely recorded in poultry, ducks, canaries, sparrows and other avian species. Howerth *et al.* (1991) described the parasite in a red lory (*Eos bornea)* which had lesions in the lungs, liver and heart caused by the parasite.

Death occasionally results with focal necrosis in the liver, kidney, spleen, pancreas, eyes, brain, and intestine. Encephalitis and eye disorders may also be seen in chronic cases of toxoplasmosis (Burr 1987).

No treatment is known. Prevention includes hygiene and prevention of cannibalism.
Cats should not be permitted near aviaries to avoid oocyst contamination from faeces as the cat is the definitive host for *T. gondii* (Harrigan 1981).

Cryptosporidium

Goodwin (1989) reports that at least three species of *Cryptosporidium* are thought to infect birds. Although known to be common in poultry, authors have also cited the parasite in a parakeet (Goodwin 1989), budgerigars and two parrot species *(Amazona aestiva aestiva*) and *(Psittacus erithacus erithacus)*(Tsai *et al.*1992). The parasite is not very host specific and is also found in Anseriforme and Passeriforme birds.

Cryptosporidium is an intracellular, extracytoplasmic coccidian that parasitises the brush border of epithelial surfaces, particularly the intestinal villi. The life-cycle is direct and the infection is obtained via ingestion of sporulated oocysts in contaminated food or water. The life-cycle is then maintained in the microvilli which causes villous atrophy and leads to diarrhoea.

Small round oocysts are passed in the faeces and are sources of infection. As the number of oocysts passed from one bird can be considerable, build up in the aviary is quick and strict hygiene is extremely important.

Nematodes

Spiroptera

Spiroptera incerta is reportedly an important nematode affecting caged-birds, especially Australian parakeets (Keymer 1982) although there are no definite reports of this parasite infecting psittacine birds in Australia.

S. incerta has an indirect life-cycle with arthropods acting as intermediate hosts. The infective larvae are ingested by the host in the egg stage and released into the intestine. From there the larvae migrate into the mucosa of the upper digestive tract where development into the adult form is completed.

The clinical signs of infection include acute death or wasting. *S. incerta* may interfere with the passage of food in the intestines by creating swellings or nodules on the mucosa of the proventriculus or ventriculus (Barnes 1986).

If the infection is severe degeneration of the gizzard lining may occur. The worms can burrow into the mucosa and immature worms are commonly found under the koilin lining of the gizzard. Occasionally they penetrate the wall of the proventriculus and move into the airsacs (Keymer 1982).

The infection is transmitted through the expulsion of thick-walled, embryonated eggs in the faeces (Barnes 1986)

Syngamus

Syngamus trachea, also commonly known as the gapeworm is a strongyle nematode that parasitises the trachea of infected birds. *S. trachea* commonly infects poultry and can also cause cross infections in aviary birds. Wild birds such as magpies and pigeons can also transmit the organism to aviary birds (Madill 1987; Harrigan and Arundel 1979; Reece *et al*.1992) with Perry (1983) recording the parasite in a white-backed magpie (*Gymnorhina tibicens*) in Australia. The parasite has also been diagnosed in a yellow-tailed black cockatoo (*Calyptorhynchus funereus*) from Melbourne Zoo.

At present *S. trachea* is not considered to be a common parasite in Australian aviary birds but the chance of infection is high if contact is possible with wild birds and as such it has the potential to become widespread.

S. trachea is a bright red worm with the male measuring 2-6mm long and the female 5-20mm long (Burr 1982). Both male and female worms inhabit the infected bird, the infection being obtained by direct or indirect (via a paratenic host) ingestion of the infective larvae (Keymer 1982). On ingestion, the larvae penetrate the intestinal wall and are carried in the bloodstream to the lungs. The young worms then further migrate via the bronchi to the trachea (Keymer 1982).

Once in the trachea both sexes attach themselves to the epithelium with the male permanently attached to the vulva of the significantly larger female (Keymer 1982).The adherence of both sexes to the epithelium causes a local inflammatory response in the trachea.

The minimum time taken to complete the life-cycle is three weeks (Madill 1987).

Young and small birds are most severely affected as worms and mucous can occlude the trachea (Steiner and Davis 1981). Such birds may eventually die from asphyxiation (Burr 1982) and present the clinical sign of 'gaping' for air. Other clinical signs may include; coughing, sneezing or head shaking in an effort to dislodge the worms (McDonald 1980). Loss of voice or voice changes may also be encountered (Barnes 1986).

S. trachea infections may be confused with trichomoniasis, bacterial, viral or air sac mite infections.

Treatments for this parasite include levamisole hydrochloride, disophenol or a benzimidazole. Care needs to be taken in birds with heavy infections because killing all the worms at once may cause tracheal blockage. Low doses are therefore recommended.

Acuaria

Acuaria spiralis previously known as *Dispharynx* has been isolated from the gizzard and proventriculus of budgerigars and other psittacine birds, passerine and game birds (Shanthikumar 1987).

The male worm is 7-8mm long and the female 9-10mm long. Embryonate eggs are passed in the faeces and arthropods may act as intermediate hosts, where further development of the parasite occurs.

Birds obtain the infection by ingesting infected grasshoppers, slaters, weevils and other arthropods. In the gizzard the larvae enter the lining and mature to their adult state (Madill 1987). The minimum time to complete the life-cycle is 57 days (Schnock and Cooper 1978).

The physical signs of the disease are dullness, ruffled feathers, anorexia and an inability to thrive leading to emaciation and death in 2-3 weeks. Sudden death may also occur due to internal bleeding (Macwhirter 1989).

Treatment of the infection is with levamisole. Maintenance of dry conditions is also important to delay egg development in the environment.

Capillaria

Capillaria infections, also known as threadworm infections have been recorded in rosella parakeets (*Platycercus eximus*) (Keymer 1982), and the crop of a blue crowned hanging parrot (*Loriculus galgulus galgulus)* (Griner 1983).

There are two species that infect the crop and oesophagus respectively, *Capillaria contorta* and *C. columbae* and a species that infects the small intestine *C. obsignata* (Burr 1982).

C. contorta, the crop worm, lives in the mouth, oesophagus and crop of its major hosts, including Psittaciforme, Galliforme and Anseriforme birds. It has a direct life-cycle, completed in 60 days. Birds are infected by the ingestion of eggs in contaminated feed or water or by the ingestion of infected transport hosts such as earthworms (Shanthikumar 1987).

The male worm of this species is 6-45mm long and the female 15-46mm long.

Adult *Capillaria* burrow into the epithelium at the site of infection causing haemorrhaging and inflammation. In some cases the inflammation of the gut may be so severe that parts of the mucous lining separate, producing slimy, yellow or bloody diarrhoea. Birds subsequently become emaciated and die of anaemia (Arnall and Keymer 1975).

Visual signs of infection are listlessness, weight loss, reduced water intake, feather loss, anorexia and a pale mucosa due to anaemia (Pass and Jakob-Hoff 1987).

Treatment has included the use of Metyridine (Keymer 1982) which is no longer marketed in Australia (Harrigan 1981) at a dose of 1g/l (Altman 1977). Fenbendazole is currently used at a dose of 10-50 mg/kg once daily for five days (Clubb 1984,1986).Forty-eight hours after treatment the eggs of Capillaria are no longer shed in the faeces.

Ascarid Nematodes

Ascarid nematodes are common in psittacine birds, particularly in Australian parakeets that spend considerable time foraging on the bottom of cage's ie. the princess parrot (*Polytelis alexandrae)* and the scarlet chested parrot *(Neophema splendida)* (Shephard 1989). Other commonly infected parrots include rosellas, barrabands, many-coloured and crimson winged parrots (Perry 1991).

Reece and Scott (1992) recognised enteric ascarid infections commonly occurring in 11% of various psittacine birds surveyed.

Commonly encountered species in psittacine birds include *Ascaridia hermaphrodita*, *A.platycerci* and *A.galli*. *A. galli* has been recorded in an elegant grass parakeet (*Neophema elegans)* and a rosella parakeet (*Platycercus eximius cecilae),* although both were kept in close proximity with Galliforme birds so are not regarded as natural infections (Peirce and Bevan 1973). *A. columbae*, primarily encountered in pigeon's can experimentally infect budgerigars (Mines and Green 1983), and has also been found in aviary bred parrots by Mines (1979).

Ascarid species have a direct life-cycle although arthropods can act as transport hosts. The larvae are ingested in the egg and released into the small intestine where they mature into adults. As adults the worms are usually unattached and free-swimming but some species bury their heads a few millimetres into the mucosa (Steiner and Davis 1981).

Clinical signs of ascarid infections include poor lustre and poor general condition due to the parasite competing with the host for nutrients. Specific clinical signs also seen are diarrhoea, lethargy, exhaustion and anaemia. In parakeets it has also been recorded that leg paralysis occurs, however this symptom has yet be accounted for (Pass and Jakob-hoff 1987; Harrigan 1981).

Birds may die if heavily infected due to an obstruction of the duodenum with worms (Keymer 1982). Death can also occur due to peritonitis after rupture of the intestines (Burr 1982).

Eggs passed in the faeces require 2-3 weeks to become infective outside the host so extensive cleaning should be maintained within this period to aid the removal of the infection. Once infected however it is

hard to eradicate the parasite from the aviary as the eggs can survive for many years in a suitable environment.

An indication of the ease of transmission of the parasite has been recorded by Parsons *et al.* (1993 abst.) who documented a severe case of ascaridiasis in a situation where no access to the bottom of the cage occurred.

Crop treatment of infected birds for 2-3 consecutive days with fenbenzadole is used in Australia at a dose of 5mg/100gm body weight (Steiner and Davis 1981). Water medication is unreliable and not as effective in treating the whole flock, especially when Australian parrot species are infected as they can go several days without water and may miss treatment all together.

Paraffin oil should be given in addition to crop medication to prevent dead worms blocking the intestinal tract as they are passed (Pass and Jakob-Hoff 1987), and antibiotics should also be administered when treating ascarid infections to control bacterial infections secondary to larval migration (Harrigan 1981).

Cestodes

Although very rarely described, it is probable that many species of cestodes are capable of infecting psittacine birds. The most commonly recorded species in psittacine birds is *Raillietina* although Reece *et al.* (1992) also isolated an unnamed cestode from the family Davaineidae from the small intestine of a sulphur crested cockatoo.

Other species that have been isolated from Australian psittacine birds include; *Paronia trichoglossi, Hemiparonia cacatuae* and *H. bancrofti* (Schmidt 1972), although no further references are found to verify these results.

All cestodes require vectors such as beetles, ants and earthworms and they all produce characteristic eggs containing a hexacanth embryo that is easily identified.

Raillietina

Raillietina is a common cestode found in both seed and fruit eating birds and has recently been recorded in two lories (*Trichoglossus haematodus*), a lovebird (*Agapornis roseicollis*) and a parakeet (*Psittacula krameri manillensis*) (Tsai *et al*. 1992). *R. cacatuina* was originally described in 1913 by Johnston in a sulphur crested cockatoo and the species *R. polychalix* was described by Kotlan in 1921 in a rock parrot *(Neophema petrophila).*

The clinical signs of infection include general debility, diarrhoea, loss of appetite and weight.

The degree of pathogenicity is not well correlated with the numbers of parasites present.
The cestode is sometimes associated with small granulomatous nodules in the intestinal wall (Harrigan 1981).

Treatment is reportedly unrewarding although Yomesan ®(Niclosamide), given as a single dose into the crop has been used successfully in a sulphur-crested cockatoo (Perry 1983).
Control of the intermediate hosts (arthropods) is the best prevention of infection**.**

Miscellaneous parasites

Along with the commonly encountered and potentially significant parasites infecting psittacine birds in Australia, there are also those to which no cases have been documented in Australia but may be problematic when introduced birds are imported into the country.
Presently no conclusive reports are available identifying parasites of the genus *Cyathostoma*, *Baylisascaris* or *Heterakis* as causing infections in Australian psittacine birds.

Cyathostoma cacatua, a nematode parasite 9.5-13.6 mm long has been isolated by Bowie (1985) from the air sacs of a sulphur-crested cockatoo in New Zealand. Numerous eggs were also isolated from the lungs of the bird.

Balisascaris progonis is an ascarid nematode recognised in South America as causing CNS disorders in psittacine hosts due to the presence of larva migrans(Myers et al. 1983; Armstrong et al. 1989). This parasite is unlikely to be encountered in Australia given that it's definitive hosts are Racoons and Skunks.

Heterakis gallinarum, whilst common in poultry breeds reportedly also infects budgerigars (Shanthikumar 1987; Schnock and Cooper (1978). No conclusive reports are available linking this parasite to cases of parasitism in psittacine birds in Australia.

References

AHMED, Z., PANDURANG, A., ACHARYA, R. S., ALI, G. (1970). Some observations on Trichomoniasis of the upper digestive tract of Love Birds (Budgerigars) in Hyderabad, Andhra Pradesh. *Indian Vet. Journal*. 47: 210-212.

ALTMAN, R.B. (1977). Parasitic diseases of caged birds. In: Current Vet Therapy VI- Small Animal Practice.Ed: Kirk, R.W. p682-687.WB Saunders and Co. Philadelphia.

ARNALL, L. & KEYMER, I.F. (1975). Bird Diseases. pp. 141-152. Bailliere Tindell, London.

ARMSTRONG, D. L., RICHARD, J. M., DOSTER, A., KAZACOS, K. (1989). Cerebrospinal Nematodiasis in Macaws due to *Bayliascaris procyonis*. *J. Zoo. Wild. Med.* 20(3):354-359.

BAKER, J.R. (1986). *Trichomonas*, a major cause of vomiting in Budgerigars *(Melopsittacus undulatus*). *Vet. Record.* 118:447-449.

BARNES, H.J. (1986). Parasites. In: Clinical Avian Medicine and Surgery. Ed: Harrison. G.J, Harrison. L.R. W.B. Saunders and Co. Philadelphia.

BLACKMORE, D.K. (1965). The Budgerigar and the Vet profession. *J. Small. An. Prac.* 6:1-6.

BOWIE, J.Y. (1985). *Cyathostoma cacatua* n sp.(Nematoda: Syngamidae) from a Sulphur-crested Cockatoo(*Cacatua galerita*) (aves: Psittacaformes), with a key to the species of *Cyathostoma. New Zealand. J .Zool.* 12:425-429.

BURR, E. (1982). Diseases of Cage Birds. Iowa State Uni Press, Iowa.

BURR, E. (1987). Digestive Tract Protozoa in: Companion Bird Medicine E. Burr ED. Iowa State Uni Press, Iowa.

CALLENDER, C.R. & SIMMONS, J.S. (1937). Trichomoniasis (*T.columbae*), in the Java sparrow,Tovi Parakeet and Verraux's Dove. *Am. Journal. Trop. Med*. 17:579-585.

CLUBB, S.L. (1984). Therapeutics. Individual and flock treatment regimens.In Clinical Avian Medicine and Surgery. Ed: Harrison, C.J., Harrison, L.R. p327-355. WB Saunders and Co. Philadelphia.

CLUBB, S.L. (1986). Therapeutics in avian medicine: flock vs individual: bird treatment regimens. In Symposium on caged bird medicine. *Vet. Clin. N. Amer.*14:345-361.

CHAKRAVARTY & KARR. (1947). Cited in Hooimeijer et al. (1993).

EMANUELSON, S. (1983). Avian Trichomoniasis in: Current Vet Therapy, Ed: Kirk R. p619-621. W.D.Saunders and Co. Philadelphia.

ERLANDSON, S.L. & BEMRICK, W.J. (1987). SEM evidence for a new species*, G. psittaci*. *J. Parasitol.*173:623-629.

EVANS, R.H., CAREY, D.P. (1986). Zoonotic Diseases in: Clinical Avian Medicine and Surgery Ed; Harrison, C.J., Harrison, L.R. 537-540, W.B. Saunders and co. Philadelphia.

FARR, M.M. (1960). *Eimeria dunsingi* n sp.(Protozoa:Eimeriidae) from the intestine of a parakeet, *M. undulatus.* Libro Homenaje als Dr. Edwardo Caballero Y Caballero.31-35 (Cited in Keymer 1982).

FORSHAW, D., PALMER, D.G., HALSE, S.A., HOPKINS, R.M., THOMPSON, R.C.A. (1992). *Giardia* in straw-necked ibis (Threskiornis spinicollis) in Western Australia. *Vet. Record.* 131:267-268.

GARNER, M.M. & STRURTEVANT, F.C. (1992). Trichomoniasis in a blue-fronted Amazon parrot. *J.Assoc. Av. Vet.* 6:17-19.

GOODWIN, M.A. (1989). Cryptosporidiosis in Birds, A review. *Avian pathology* 18:365-384.

GRAY, H. (1936). The diseases of cage and aviary birds,with some reference of furred and feathered game. *Vet. Record.*16:343-362. (Part 1), 377-386 (Part 2), 417-427 (Part 3).

GRINER, L.A. (1983). Pathology of Zoo Animals. Zoological Society of San Diego.

HARPER, F.D.W. (1991). *Hexamita* species present in some avian species in South Wales. *Vet. Record.* 128:130.

HARRIGAN, K.E. (1981). Bird Parasitism. Refresher course on aviary and caged birds Feb 16-20, 1981. 55:338-394 Sydney University Press.

HARRIGAN, K.E. & ARUNDEL, J.H. (1979). *Syngamus trachea* infections in the White-backed Magpie (*Gymnorhina hypoleuca*). *Aust. Vet. Rec*. 54:360-361.

HONIGBERG, B.M. (1978). Trichomonads of Veterinary Importance. In: Parasitic Protozoa. Ed: Kreier, J.P.p164-273. Academic Press N.Y.

HOOIMEIJER, J., PEEK, H.W., VERTOMMEN, M.H. (1993). Coccidiosis in Lorikeets infectious for Budgerigars. 59-61. 1993 Proc. Ass. Av. Vets.

HOWERTH, E.W. (1991). Fatal Toxoplasmosis in a Red Lory(*Eos borneo*). *Avian Dis.* 35:642-646.

JACOBSON, E.R., GARDINER, G.H., NICHOLSON, A., PAGE, C.D. (1984). *Sarcocystis* Encephalitis in a cockatiel. *J. Am. Vet. Med. Assoc.* 185:904-906.

JONES, D.M., CARROL, M.M. (1977). Debilitating syndrome in budgerigars (Melopsittacus undulatus). *Vet Rec.* 101:188.

JOHNSTON, T.H. (1913). Cestoda and Acanthocephala. *Rep. Austral. Inst. Trop. Med.* (1911) 75-96. (Cited in Schmidt 1972).

KEMP, R.L. (1978). Trichomonads, other flagellates and protozoa. In; Diseases of Poultry 7th Ed. Ed; Hoffstad,M.S., Calnek, B.W., Helmobolt, C.P., Reid, W.M. and Yoder, H.W. Iowa State Uni Press, Iowa.

KEYMER, I.F. (1982). Parasitic Diseases.In: Diseases of cage and aviary birds. Ed: Petrak, M.L. Lea and Febiger Philadelphia.

KOCAN, R.M. & KNISLEY, J.O. (1970). Challenge infection as a means of determining the rate of disease resistant *Trichomonas gallinae*-free birds in a population. *J. Wild. Dis*. 6:13-15.

KOTLAN, A. (1921). Uj-Guineai madar-cestodak .I. Papagaly- cestodak.Ann. Mus. Natt. Hungerici18:1-27(In Hungarian) (Cited in Schmidt 1972).

MACWHIRTER, P. (1989). Avian Practice in Australia. p46-57. 1989 Proc. Assoc. Av. Vet.

MADILL, D. (1987). Parasitic Diseases. In: Everybird Ed: Macwhirter.P. 45-59, Incarta Press Pub. Melbourne.

McDONALD, S.E. (1980). Respiratory disease in psittacine birds. In: Current Vet Therapy. VII Ed: Kirk. R.W. W.B. Saunders and Co. Philadelphia.

MIESSNER, H. & HANSEN, K. (1936). Trichomoniasis der Tauben. Duet. Tieratz. Wchnschr. 44:323-330. (Cited by Stabler, R. (1947). *Trichomonas gallinae*, Pathogenic Trichomonad of Birds. *J. Parasitol.* 207-213.)

MINES, J.J. (1979). *A. sprenti*- a new apecies of nematode in Australian Parrots. *Int. J. Parasitol*. 9:371-379

MINES, J.J. & GREEN, P.E. (1983). Experimental *A. columbae* infections in Budgerigars (*M. undulatus*). *Aust. Vet. J*. 60:279-280.

MUNDAY, B.L., HARTLEY, W.J., HARRIGAN, K.E. (1979). *Sarcocystis* and related organisms in Australian Wildlife: II survey findings in birds, reptles, and fish. *J. Wild. Dis.* 15:57-73.

MURPHY, J. (1992). Psittacine Tichomoniasis. p21-24. 1992 Proc. Assoc. Av. Vet.

MURTAUGH, R.S., JACOBS, R.M. (1984). Trichomoniasis of the crop in a Cockatiel. *J. Am Vet. Med. Assoc.* 185:441.

MYERS, R.K., MONROE, W.E., GREVE, J. H. (1983). Cerebrospinal nematodiasis in a Cockatiel. *J. Am. Vet. Med. Assoc*. 183:1089-1090.

PANIGHRAHY, B., MATHEWSON, J.J., HALL, C.F., GRUMBLES, L.C. (1981). Unusual disease conditions in pet and aviary birds. *J. Am. Vet. Med. Assoc*. 178: 394-395.

PARSONS, B., WISSMAN, M.A. (1993 abstract). Severe ascaridiasis in aviary birds. *J. Assoc. Avian Vets*.7:100.

PASS, D.A, JAKOB-HOFF, R. (1987). Some common diseases of birds. In: Seminar for aviculturists on Diseases of cage and aviary birds. School for Veterinary Studies, Murdoch.

PERRY, R.A. (1983). VADE MECUM. Diseases of birds.Uni. of Sydney, Post graduate foundation in Vet Science. Sydney, Australia.

PIERCE, M.A. & BEVAN, B.J. (1973). *A. galli* (Shrank,1788) in Psittacine birds. *Vet. Record.* 92:261.

RAMSAY, E.C., DREW, M.L., JOHNSON, B. (1990) Trichomoniasis in a flock of Budgerigars. 309-311. Proc. Assoc. Av. Vets.

REECE, R.L. & SCOTT, P.C. (1992). Some unusual disease in the birds of Victoria, Australia. *Vet. Record.* 130:178-185.

SCHMIDT, G.D. (1972). Cyclophillidean Cestodes of Australian birds, with three new species. *J. Parasitol*. 58:1085-1094.

SCHNOCK, R.C. & COOPER, R. (1978). Internal parasitisms in captive birds. *Modern Vet. Practice.* 59:439-443.

SHANTHIKUMAR, R. (1987). Helminthology. In: Companion Bird Medicine. 135-146. Ed: Burr, E . Iowa State Uni Press, Iowa.

SHEPHARD,M. (1989). Aviculture in Australia, keeping and breeding aviary birds. Black Cockatoo Press Melbourne.

STEINER, C.V., DAVIS, R.B. (1981). Caged Bird Medicine-Selected Topics. Iowa State Uni Press , Iowa.

TODD. K.S Jn., SCHMIDT, J.M., GALLINA, A.M. (1977) *Eimeria dunsingi* (Farr,1960): A Coccidium of the Parrakeet *(M. undulatus*). *Proceed. Helm. Soc*.44.

TSAI, S.S., HIRAI, K., HAKURA, C. (1992). Histopathological survey of Protozoa, Helminths and Ascarids of imported and local psittacines and passerine birds of Japan. *Jap. J. Vet. Res*. 40:161-174.

VARGHESE, T. (1977). *Eimeria haematodi*. sp. n (Protozoa:Eimeriidae) from the rainbow lorikeet, *Trichoglossus haematodus*, in Papua New Guinea. *J. Parasitology*. 63:210-211.