Seabird Health Problems - Parasites on the Wing

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Summary

Information from the literature relevant to research in progress on *Contracaecum* spp. infection and a survey for haemoparasites, both in the Little Penguin (*Eudyptula minor*), is summarised. *Plasmodium* spp. infection is a significant cause of mortality in overseas captive collections of penguins. It has been recorded from a few wild populations of penguins, but its significance in the wild has not been examined. *Contracaecum* sp. infection of piscivorous birds can cause serious gastric ulceration, particularly in young birds. A list of species of *Contracaecum* from Australia and adjacent regions with host records is given.

Introduction

These notes are not intended as a review of parasitism in seabirds, but instead emphasis will be given to two aspects of marine bird parasitic disease which are relevant to the project "Parasitic Diseases of the Little Penguin" with which I am involved at Melbourne University. Where synonymies of avian species names could not be confirmed at the time of writing, the citation given in the paper referred to is used.

Parasitic diseases of birds which a veterinary surgeon practising in Australia is likely to encounter have been described in some detail by Harrigan (1978). This comprehensive paper includes information on diagnosis, treatment, pathology and life history of parasites taken from the literature of the day, the experience of the author and personal communications from other workers in the field.

Most recently a checklist of lice, fleas, hippoboscid flies and ticks parasitic on a range of birds, and particularly seabirds, from the Australian, New Zealand and Antarctic regions has been published (Murray, Palma, and Pilgrim, 1990). Reece and co-workers at the Veterinary Research Institute published their findings on neoplasms and a range of pathologies encountered in birds in Victoria over a ten year period (Reece, 1992; Reece, Scott, and Barr, 1992) and the joint paper provides some information on seabird parasites.

Johnston (1912) provided an early list of internal parasites of avian hosts in Australia. This author, subsequently in partnership with Mawson, was prolific in his descriptions of species

parasitic in a wide range of hosts including birds from Australia and the Sub-Antarctic region. Cleland (1922) reviewed the published records of internal parasites of Australian birds and added a large number of his own observations.

Mackerras and Mackerras (1960) published a checklist of haemoparasites of Australian birds. Keast (1977) explored some aspects of disease, including parasitism, of wild birds and noted the absence of detailed study at that time.

Seabirds have not been treated as a natural body in the literature on systematic or pathological parasitology, so it is necessary to take the relevant information from general works or to gather together the detailed papers to gain a perspective on this field.

I. **Avian Malaria.**

History

A wider definition of avian malaria embracing the three genera of the family Haemosporidae: *Plasmodium*, *Leucocytozoon*, and *Haemoproteus*, has been used sometimes in the study of this parasitism (Huff, 1963; Huff, 1968). Other authors writing on the topic of avian malaria have confined themselves to *Plasmodium* (Manwell, 1938; Garnham, 1979). A concise treatment of the development of knowledge about malaria is given by Seed and Manwell (1977), and a more detailed narrative by Schmidt and Roberts (1977). Meckel observed black pigment granules lying within protoplasmic masses in cells of the blood and spleen of a patient who died of malaria. Afanasiev suggested the granules were the cause of the disease in 1879. In 1880, Laveran observed gamogony and exflagellation (the formation of male gametes) in blood. In 1884 Danilevski observed a malarial parasite in the blood of a bird (Garnham, 1979). Ross, encouraged by Manson, demonstrated the role of the mosquito (*Anopheles*) in the life history of human malaria in 1897. Bignami and Grassi experimentally transmitted malaria from the mosquito to a human in 1898. Ross continued his work with *Plasmodium relictum* from birds (Daniels, 1900) and was awarded the Nobel Prize in Medicine in 1902 (Schmidt and Roberts, 1977). Avian malaria (particularly *Plasmodium relictum* and *Plasmodium cathemerium* in sparrows and canaries) proved an important experimental model for research in the pathology, life history and chemotherapy of the human disease (Seed and Manwell, 1977). The discovery of *Plasmodium gallinaceum* by Brumpt in 1935, which infects chickens, and *Plasmodium lophurae* by Coggeshall in 1938, which infects ducks allowed larger experimental hosts to be used in research. James and Tate observed *Plasmodium gallinaceum* infecting capillary endothelial cells of the brain of the chicken in 1937. Hewitt published an important monograph on avian malaria in 1940. The use of primate models (*Plasmodium malariae*, *Plasmodium knowlesi*, *Plasmodium coatneyi*, *Plasmodium cynomolgi* and *Plasmodium simium*) and rodent models (*Plasmodium berghei*) for recent research has reduced the proportion of work done on avian malarias.

Summary of the Lifecycle of Plasmodium spp. in Birds.

There is some variation between species of *Plasmodium* in the process of development, but the essentials can be summarised with the following generalised outline of the human malariae (Mehlhorn and Walldorf, 1988). Elongate sporozoites in the salivary gland of a mosquito are injected into the host while the mosquito takes a blood meal. The sporozoites travel in the circulation to the liver within 2 to 30 minutes and enter hepatocytes where asexual multiplication (schizogony) leads to development of schizonts which contain many merozoites. Schizonts rupture, destroying the hepatocyte and releasing the merozoites, which may reinvade other hepatocytes or begin the erythrocytic cycle. In the erythrocytic cycle, merozoites enter erythrocytes and schizogony begins. A constant number of cycles of erythrocytic schizogony may be associated with a particular species of *Plasmodium*. After these stages of asexual multiplication, the final generation of merozoites will enter erythrocytes and each one will become either a macrogamont or a microgamont. If a mosquito ingests erythrocytes containing macro- and microgamonts, these are released into the gut of the insect as the erythrocytes rupture, and the microgamont divides to form a number of microgametes and the macrogamont develops into a single macrogamete. The macrogamete is fertilized by a microgamete, forming a zygote which develops into an ookinete. The ookinete encysts within a gut cell of the mosquito, forming an oocyst. This develops to a sporoblast containing thousands of sporozoites. The sporoblast ruptures into the haemocoel of the insect, liberating the sporozoites, which make their way to the salivary gland. The complex nomenclature, which has not been given in full here, should suggest the enormous multiplication during the stages between sexual reproduction. Incidentally it has been suggested that the mosquito could be regarded as the definitive host of *Plasmodium* spp. as it is in this host that sexual reproduction occurs (Schmidt and Roberts, 1977).

Species of avian malaria differ from this mammalian pattern at a number of points. *Plasmodium relictum* sporozoites injected by a mosquito are carried to the reticular cells of the splenic Malpighian body where the initial generation of exoerythrocytic schizogony occurs. Subsequent exoerythrocytic generations are distributed more generally, and lung, brain and spleen may be involved. *Plasmodium elongatum* will not parasitize erythrocytes exclusively, but may parasitize all blood cells including stem cells in the bone marrow (Seed and Manwell, 1977). *Plasmodium cathemerium* and *Plasmodium relictum* distort erythrocytes markedly and their gametocytes may displace the nucleus from the cell (Seed and Manwell, 1977). Avian malaria merozoites from erythrocytes may reinitiate exoerythrocytic schizogony (Seed and Manwell, 1977; Huff, 1957), so infections transmitted by inoculation of infected blood can lead to both the erythrocytic and exoerythrocytic cycles becoming established. This may not occur in mammalian malaria (Schmidt and Roberts, 1977; Seed and Manwell, 1977).

The most important vectors for human malaria are *Anopheles* spp. Development in the mosquito takes from 10 days to 2 weeks (Schmidt and Roberts, 1977), depending on temperature (Daniels, 1900). *Plasmodium gallinaceum* has frequently been studied in *Aedes aegypti*, while *Plasmodium cathemerium* has usually been studied in *Culex pipiens* (Huff, 1965). Huff's paper (1965) presents an exhaustive review to that day of experimental host, parasite

and vector associations. Several *Aedes*, *Culex*, and *Anopheles* spp. are listed as proven susceptible to *Plasmodium relictum*. Several *Culex* spp. and one *Aedes* sp. is listed as susceptible to *Plasmodium elongatum*.

Pathogenesis.

The parasitaemia of *Plasmodium* infection leads to increased destruction of erythrocytes, causing an anaemia which may be fatal (Seed and Manwell, 1977). Erythrocyte destruction may be by intravascular haemolysis of both parasitized and non-parasitized cells. Both become fragile, the former because of the growing intracellular parasite, and the latter due to alterations of the plasma (Seed and Manwell, 1977). The destruction of erythrocytes during erythrocytic schizogony releases the metabolic waste of the intracellular parasite (Schmidt and Roberts, 1977). Enlargement of the spleen may be extreme, as a result of both oedema and a hypercellularity contributed to by increased phagocytosis of erythrocytes and lymphoid hyperplasia (Seed and Manwell, 1977). Thrombosis and necrosis may occur. Similar changes may be evident in the liver. Glomerulonephritis occurs in *Plasmodium gallinaceum* infection in chickens probably due to circulating immune complexes, which also contribute to the anaemia (Soni and Cox, 1974). Exoerythrocytic stages parasitizing endothelia of blood vessels can lead to occlusion of cerebral blood flow and stroke (Seed and Manwell, 1977).

The pathogenicity of infection by different species of *Plasmodium* varies with the host species. The interaction of host and parasite will dictate the pattern of occurrence and the distribution of exoerythrocytic stages (Huff, 1957).

Diagnosis.

The most important method of diagnosis used for the human malarias is examination of stained blood smears. Several techniques are available. Thick or thin smears may be stained with Romanowski's stain, Giemsa, Wright's or Leishman's stains (Seed and Manwell, 1977). More sophisticated techniques using fluorescent dyes such as acridine orange have enhanced the sensitivity of screening blood smears (Kawamoto and Billingsley, 1992; Kawamoto, 1991; Spielman, Perrone, Teklehaimanot, Balcha, Wardlaw, and Levine, 1988). The use of a modified QBC microhaematocrit tube coated with acridine orange dye can increase the sensitivity of screening peripheral blood samples because the parasitized erythrocytes are concentrated into a narrow zone of the tube (Levine, Wardlaw, and Patton, 1989). These fluorescent dye techniques require the use of fluorescence microscopes, although a simpler and much cheaper arrangement using interference filters has been described (Makler, 1991). There is the potential for the molecular techniques of the polymerase chain reaction and ribosomal RNA detection to be used in malaria diagnosis, but this has not yet been realised (Waters and McCutchan, 1990).

The geographical location and clinical picture will assist the identification of the species involved in human infections (Schmidt and Roberts, 1977).

Seabirds in Australia and Penguins.

The Australian literature of the first decades of this century indicates the enthusiasm with which avian haemoparasites were sought (Cleland and Johnston, 1910; Gilruth, Sweet, and Dodd, 1910; Cleland and Johnston, 1911; Cleland, 1915; Cleland, 1922). Both positive and negative findings are reported in these papers, although frequently only one or a few birds of a species were examined. With such small samples, little can be concluded from a negative result but amongst these a *Eudyptula minor* from Encounter Bay had no haemoparasites (Cleland, 1922) and neither did one from Broughton Island (Cleland and Johnston, 1911). The first seabird host in Australia recorded infected with a *Plasmodium* sp. was a Silver Gull (*Larus novaehollandiae*), observed by Bearup (Mackerras and Mackerras, 1960).

Scott, the pathologist working at London Zoo in 1926, recorded the death of a King Penguin (*Aptenodytes patagonica*) with air-sac mycosis, acute enteritis and a very high *Plasmodium* parasitaemia (Scott, 1927). In 1937, Rhodain found large numbers of erythrocytes in the heart blood of an African penguin (*Spheniscus demersus*) to be parasitised by a *Plasmodium*. The penguin had died at the Antwerp Zoo, and it was noted that the infection may have been acquired after the bird arrived at the zoo, perhaps with a *Culex* sp. acting as a vector. He passaged the parasite through canaries and a number of other avian hosts, and his paper describes the morphology and affinities of the species. The role of the infection in the death of the host was considered. Fantham and Porter (1944) described *Plasmodium relictum* var. *spheniscidae* in the peripheral circulation of wild *Spheniscus demersus* from South Africa in 1927, and in Yellow Eyed Penguins (*Megadyptes antipodes*) from near Stewart Island in New Zealand and Rock-Hopper Penguins (*Eudyptes crestatus*) from Gough Island in the southern Mid-Atlantic in 1929. In this study, blood smears from only a small number of birds of each species were examined, so prevalence estimates are probably of little value. Fantham and Porter touched on reasons for only a small number of infections being observed, including the possibility of the development of immunity by older birds.

Laird (1952) found three of 28 Fiordland Crested Penguins (*Eudyptes pachyrhynchus*) infected with *Plasmodium relictum* var. *spheniscidae* as well as one of two *Megadyptes antipodes* from Campbell Island infected with the same organism. This study was widened to embrace several species on Macquarie Island, but in examining blood samples from *Aptenodytes patagonica*, Gentoo Penguin (*Pygoscelis papua*), Rockhopper penguin (*Eudyptes chrysocome*), and Royal Penguin (*Eudyptes schlegeli*) no blood parasites were detected. In addition, no haemoparasites were observed in blood films from the Macquarie Island Shag (*Phalacrocorax traversi* =? *P. albiventer purpurascens*), and Elephant Seals (*Mirounga leonina*). The findings conformed to the perception that mosquitos were required as vectors as these are not present on Macquarie Island, and it was suggested that species from which *Plasmodium* had been recorded would only become infected when visiting the land masses in the northern parts of their ranges.

Antwerp Zoo proved an interesting locality for penguin protozoologists with a further case of *Plasmodium relictum* infection in a Humboldt Penguin (*Spheniscus humboldti*) in 1938, then two cases of *Plasmodium praecox relictum* Grassi and Felleti infection in Chinstrap Penguins (*Pygoscelis antarctica*) (Rodhain and Andrianne, 1952). Exoerythrocytic stages were observed in the lungs at necropsy in the latter cases. In addition, a Guillemot (*Uria aalge*) found dead in the zoo was found to have been infected with *Plasmodium praecox relictum*. It was again thought that the infections could have been acquired at the zoo because of the availability of vectors in the warm months.

Blood samples from 18 *Eudyptula minor* from Montagu Island, NSW were examined in an investigation of a perceived die off, but no haemoparasites were found (Mykytowycz and Hesterman, 1957).

Plasmodium elongatum infection of penguins was observed for the first time in *Spheniscus demersus* at the National Zoo in Washington. Whether the infections originated in the wild or were acquired in captivity was not known (Huff and Shiroishi, 1962).

A detailed account of the pathology of *Plasmodium praecox* (Grassi and Felletti, 1890) infection in *Spheniscus humboldti* and *Spheniscus demersus* from the zoo at Schönbrunn in Vienna noted the disease was only observed in the warm seasons. The pathological findings included massive invasion of the liver, spleen and lungs by schizonts in acute infections, and involvement of other organs when the clinical course was prolonged (Grunberg and Kutzer, 1963).

In another study of *Plasmodium elongatum* infection in *Spheniscus demersus* at the Baltimore Zoo, an acute and rapidly fatal disease was described (Fleischman, Squire, Sladen, and Melby, 1968). A clinical course of disease of between three hours and three days was observed, with vomiting being followed by death. Diagnosis was by examination of peripheral blood smears, histopathology and inoculation of canaries and ducks with kidney and spleen homogenates. Gross findings at necropsy were hepatosplenomegaly, subcutaneous and pulmonary oedema and hydropericardium. An important histopathological finding was exoerythrocytic schizonts in lung, spleen and heart, and in fewer numbers in kidney, brain, intestine, bone marrow and skeletal muscle. Amongst other observations infiltrates of lymphocytes, plasma cells and histiocytes in the lung, liver and spleen were seen. There was also focal coagulative necrosis of the lung. These authors suggest the possibility of misdiagnosis of malaria as toxoplasmosis because of a resemblance of the exoerythrocytic schizonts of *Plasmodium* to tissue cysts of *Toxoplasma*. In examining the possible source of infection for these birds, the authors noted that two genera of mosquitoes (*Culex* and *Aedes*) can transmit *Plasmodium elongatum* and that three species in these genera are present locally, along with wild birds which harbour the parasite. Their discussion suggested that the severity of disease might suggest the parasite is poorly adapted to this host.

A prospective study of *Spheniscus demersus* at the same zoo (Stoskopf and Beier, 1979) showed a relative lymphocytosis was characteristic of infected birds. Mortality was prevented in birds with parasitaemia by combined chemotherapy of primaquine phosphate 0.03 mg base/kg bodyweight *per os* each day for 3 days with chloroquine phosphate 10 mg base/kg *per os* as an initial dose, followed by 3 doses of 5 mg/kg at intervals of 6 hours. Ducks were inoculated with blood samples in an attempt to increase the sensitivity of detection of parasitaemia, but false negatives and the delay required led the authors to conclude that this technique was unsuitable in the clinical situation. Extensive surveillance of wild birds and mosquito vectors (Beier and Stoskopf, 1980) in the environs of the zoo, and examination of the epidemiology of disease strongly suggested a local origin for the disease.

Captive penguins at another North American zoo, San Diego, have been seriously affected by *Plasmodium relictum* (Griner and Sheridan, 1967; Griner, 1974). In July, August and September of 1965, deaths of the following species of penguins were recorded to be due to *Plasmodium relictum* infection: *Spheniscus humboldti*, *Spheniscus demersus*, *Aptenodytes patagonica*, *Pygoscelis papua*, and *Eudyptula minor*. A Laysan Albatross (*Diomedea immutabilis*) also died with this infection. Griner (1974) speculated that the source of the infection was a local reservoir and local vectors, and that the seasonal incidence of mortality might be related to heat stress of the birds allowing a fatal disease to be triggered.

Plasmodium relictum was the cause of the loss of 38 of a group of 46 Magellanic Penguins (*Spheniscus magellanicus*) at Blank Park Zoo in Iowa (Fix, Waterhouse, Greiner, and Stoskopf, 1988). The birds had been caught on an island off the South coast of Chile, held on the mainland for about a month, quarantined in Michigan for 38 days and then shipped to the zoo at the end of April. The first death occurred approximately 48 hours after arrival, and six more died over the next 13 days. Twenty more birds died during June and July, and then 13 more in September. Pathological findings were similar to previous studies, and the significance of schizogony, particularly in the capillary endothelia of spleen, liver and lung was stressed. The origin of infection was again unknown, but the authors presented evidence suggesting infection on arrival at the zoo, with the initial death due to acute preerythrocytic schizogony. The roles of stress, concurrent disease, and long term antibiotic therapies prophylactic against aspergillosis and malaria in the clinical events were discussed.

In an important extension of the work of Laird (1952), Jones (1988) drew together the negative results of several surveys of wild Antarctic and Subantarctic seabirds, and particularly penguins, for haemoparasites. He examined Giemsa stained thin smears from *Aptenodytes patagonica*, *Eudyptes schlegeli*, *Pygoscelis papua* from Macquarie Island and *Aptenodytes patagonica* and Macaroni Penguins (*Eudyptes chrysolophus*) from Heard Island and found no haemoparasites. Other species surveyed in earlier studies, without finding haemoparasites, included *Eudyptes crestatus*, Adélie Penguins (*Pygoscelis adeliae*), Emperor Penguins (*Aptenodytes forsteri*), *Pygoscelis antarctica*, and amongst non-Spheniscid hosts, Snow Petrels (*Pagodroma nivea*), South Polar Skuas (*Catharacta maccormicki*), and Wilson's Petrel (*Oceanites oceanicus*). The absence of vectors for *Plasmodium* in these latitudes has been noted. Ectoparasites which may act as vectors for other haemoparasites are available and Jones quotes Peirce and Prince's

observation of *Hepatozoon albatrossi* n.sp. in the Grey Headed Albatross (*Diomedea chrysostoma*), the Wandering Albatross (*Diomedea exulans*), and the Black Browed Albatross (*Diomedea melanophris*) on South Georgia. They speculated that *Ixodes uriae* might act as the vector.

Skuas held in quarantine at Taronga Zoo have been affected by *Plasmodium* (Hartley, 1992).

In a paper presented to the 2nd International Penguin Conference held earlier this year, Brossy (1992) described infections of *Spheniscus demersus* in South Africa by *Plasmodium relictum*, *Leucocytozoon*, and *Babesia*. The first two parasites were associated with disease with a high mortality, while the third appeared to be endemic and only of clinical importance in birds under stress.

At the same meeting, Spielman read a paper by his colleagues at Taronga Zoo (Cunningham, Stephens, Bennett, and Humphries, 1992) describing a haematological study of *Eudyptula minor* at Lion Island and Bowen Island in NSW, in which two morphologies of intraerythrocytic parasites were described. It is possible that one may be a ring form of a *Plasmodium* sp., but work on their identification was incomplete. A recent survey of 57 *Eudyptula minor* from southeast Tasmania (Jones and Woehler, 1989) using Giemsa stained thin smears found no *Plasmodium*, but did find a new species of blood trypanosme *Trypanosoma eudyptulae*.

II. ***Contracaecum*** **Infection in Birds.**

Nematodes of the genus *Contracaecum* are robust, up to about 80 mm long, and as adults, inhabit the stomachs of a wide range of fish eating birds and mammals throughout the world. The pathological effects of several individual species in their natural hosts have been studied, and the broad outline of the life history of members of the genus is usually reported with confidence. Some interest in *Contracaecum* has been reflected from the research on species within the same subfamily Anisakidae, such as *Anisakis* spp., which are significant economically principally due to their pathogenic effect in humans.

It is necessary to use magnification, usually of specimens cleared in a solution such as lactophenol which renders the white cuticle transparent, to distinguish *Contracaecum* from other genera in the superfamily Ascaridoidea which occur in the stomachs of piscivores. At the anterior end, three lips of equal size are present. Between each lip lies a small digitate structure called an interlabium. Within the body of the nematode, approximately the anterior third is occupied by the oesophagus and a shorter structure lying parallel to it called the intestinal caecum. Male specimens have paired copulatory organs called spicules. Species are differentiated mainly on the morphology of the interlabia and spicule lengths.

Lifecycle.

Adult female nematodes in the stomach of mammals or the stomach or crop of birds pass eggs approximately 65 µm X 55 µm, containing an often bilobed embryo. Over 48 hours at room

temperature in saline, the first larval stage becomes visible within the egg. The time taken for development is prolonged with decreasing temperature, such that between 10°C and 0°C this may take two to six months, while eggs maintained at -20°C for two months may retain their viability (Davey, 1969). The larva undergoes a moult while in the egg, so that the second stage larva ensheathed in the cuticle of the first stage emerges from the egg after about one to two weeks. This larva has a single tooth and is vigorously motile, exhibiting wriggling actions within its loose cuticle sheath for another week at room temperature. After this time, the wriggling activity is diminished but may be stimulated again possibly by light or heat over another two or three weeks or more. The length of the second larval stage in its sheath is about 300 to 600 µm (Davey, 1969; Huizinga, 1967). These times and lengths are probably all dependent on species and temperature.

It is clear that *Contracaecum* is heteroxenous, that is individuals require more than one host to develop to sexual maturity, but the specificity of the requirements has been brought into doubt. Thomas (1937) found that eggs of *Contracaecum spiculigerum* from cormorants developed spontaneously in distilled water until two moults had occurred, after which the egg hatched, releasing the third larval stage with two cuticle sheaths. He recorded the third moult occurring in free living larvae, and that he was able to infect guppies (*Labistes reticulatus*) by feeding them free living larvae. Further, Thomas was able to infect black bass (*Aplites salmoides*) by feeding them infected guppies.

Davey (1969), working on *Contracaecum osculatum* from grey seals, demonstrated the harpacticoid copepods *Idya furcata* and *Amphiascus similis* ingesting free living ensheathed second stage larvae. The larvae did not develop further but exsheathed and penetrated the invertebrates' haemocoel and were able to survive for a number of months. He suggested that this might be a necessary phase of the life history in order that the larva could be ingested by a fish that might not otherwise be able to detect it as a food item, and that the larva would be able to penetrate the gut wall of the next host once the sheath was lost and the larvae's tooth exposed.

Johnston and Mawson (1945) found that although fish (species not indicated) readily ate the free larvae of *Contracaecum spiculigerum* and *Contracaecum bancrofti*, none became infected.

Huizinga (1967) recapitulated the phases described by Davey using *Contracaecum multipapillatum* from the water turkey (*Anhinga anhinga leucogaster*), except that he used *Cyclops vernalis* as the copepod host. He described minor development of larvae in the haemocoel of the copepod, and infected guppies (*Lebistes reticulatus*) by feeding infected copepods. Guppies did not become infected by directly feeding on free living larvae. In the guppies, larvae were found encapsulated in the mesenteries, liver, intestinal wall and pericardial sac. These larvae moulted but did not shed the cuticle of the second larval stage. Largemouth bass (*Micropterus salmoides*) were infected by feeding them infected guppies, but no further development occurred.

A number of artificial cultivation techniques, including *in vitro* cultivation (McClelland and

Ronald, 1974) and surgical implantation in lab animal peritoneal cavities (Fagerholm, 1988; Deardorff and Overstreet, 1980) have been used in investigations of the life history, taxonomy and pathology of *Contracaecum* spp.. An attempt to infect a seal with *in vitro* cultivated larvae of *Contracaecum osculatum* failed (McClelland and Ronald, 1974). Third stage larvae of *Contracaecum multipapillatum* from fish failed to become established in chicks, ducklings or rats after oral infection (Deardorff and Overstreet, 1980), although third stage *Contracaecum osculatum* larvae from fish moulted to the fourth stage in the stomachs of rats and hamsters after two to five days (Fagerholm, 1988)

Ascaridoid larvae thought to belong to the genus *Contracaecum* are reported encysted in the mesenteries of fish (Johnston and Mawson, 1945; Deardorff and Overstreet, 1980; Szalai and Dick, 1990; Valtonen, Fagerholm, and Helle, 1988), though the identification of larvae to generic and specific level presents difficulties. Infections of definitive hosts have been achieved by feeding naturally infected intermediate host fish (Greve, Albers, Suto, and Grimes, 1986).

From this it can be seen that in general, an invertebrate paratenic host feeding on a free living second larval stage of *Contracaecum* sp. can be infective for a fish intermediate host. The third stage larva which develops in the fish is infective for a bird of mammal host, although a second fish intermediate host may be interposed in the life history between the first fish and the definitive host. However it is unclear whether there is any specificity either on a physiological or ecological basis in natural populations of *Contracaecum* spp. for paratenic and intermediate hosts.

Pathology.

The pathology of natural *Contracaecum* sp. infections in pelicans, cormorants (Huizinga, 1971; Greve, Albers, Suto, and Grimes, 1986) and fledged Little Penguins (Obendorf and McColl, 1980; Harrigan, 1988; Harrigan, 1992) has been described. Piscivorous birds are probably infected as nestlings when parent birds feed them by regurgitating fish. This is the case in Brown pelicans (*Pelecanis occidentalis*) (Courtney and Forrester, 1974; Greve, Albers, Suto, and Grimes, 1986) and Cormorants (*Phalacrocorax* spp.) (Huizinga, 1971) in the USA. Because of the deficiencies in knowledge of the pace of development of larval forms, it is unclear whether the parent birds pass on their own adult nematodes or just infective larvae. Seabirds such as penguins can regurgitate adult *Contracaecum* sp. nematodes when feeding chicks. Cormorant chicks three weeks old may pass *Contracaecum spiculigerum* eggs (Huizinga, 1971). Therefore it is likely that both forms of infection may occur.

Adult and larval *Contracaecum* sp. nematodes may be found attached by their anterior ends to the mucosae of the stomach and lower oesophagus in Little Penguins. Acute and chronic ulceration may be present at these attachment sites. Often a number of nematodes may be attached at a single site and ulcers may be up to about 20 mm in diameter (Obendorf and McColl, 1980). A cephalic cap structure by which larval forms of *Contracaecum osculatum* attached themselves to flask walls in in vitro culture has been described (McClelland and Ronald, 1974). Ulcers may have a caseous plaque firmly adherent to the underlying granulation

tissue. Obendorf and McColl (1980) described this structure and the histopathology associated with acute and chronic ulcers. The haemorrhagic foci of acute ulcers in their study were succeeded in chronic ulcers by deep erosions into the submucosa, infiltration by lymphocytes, plasma cells and fibroblasts, with heterophils in the peripheral tissues. Burdens of nematodes reaching 300 are encountered, and there may be haemorrhage or a tenacious mucus covering over the gastric mucosa. Penetration of the wall into the pleuroperitoneum may occur.

Harrigan (1988; 1992) and Obendorf and McColl (Obendorf and McColl, 1980) give detailed epidemiological information on their respective series. From these and other references (McOrist and Lenghaus, 1992; Norman, Du Guesclin, and Dann, 1992) it can be seen that it is very common for Little Penguins to have some burden of *Contracaecum* sp., and severe gastric ulceration is normally associated with heavy burdens, however healthy birds may have some infection, with mild ulceration without clinical effect. Mortalities of mature birds attributed to starvation may not have any *Contracaecum* sp.

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Table 1. Contracaecum spp. described from avian hosts in Australia and adjacent regions

Species	Host	Locality
Contracaecum eudyptulae Johnston & Mawson, 1942	*Eudyptula minor* (Type)[7]	Broughton Is., NSW (Type) WA, SA, Vic.
Contracaecum rudolphii Hartwich, 1964 [Synonym:*Contracaecum spiculigerum* (Rudolphi, 1809) of Johnston & Mawson, 1941 - part.]	*Phalacrocorax carbo*[5][2] *Phalacrocorax sulcirostris*[5][2] *Phalacrocorax melanoleucus*[5][2] *Phalacrocorax fuscescens*[5][10][2] *Notophoyx pacifica*[5][2] *Notophoyx novaehollandiae*[5][2] *Botaurus poeciloptilus*[5][2] *Nycticorax caledonicus*[5][2] *Phalacrocorax varius*[5][2] *Phalacrocorax colensoi*[13] *Larus novaehollandiae scopulinus*[13] *Phalacrocorax magellanicus*[1][2] *Phalacrocorax verrucosus*[10][2] *Phalacrocorax brevirostris*[10][2][9] *Phalacrocorax atriceps nivalis*[10][2] *Phalacrocorax purpurascens*[10][2]	SA, NSW, Qld SA, Qld SA, NSW, Qld Kangaroo Is., SA; Tas. Qld NSW SA SA WA Auckland Is. Campbell Is. Hermite Is., Cape Horn Kerguelen Is. NZ Heard Is. Macquarie Is.
Contracaecum micropapillatum (Stossich,1890) [Synonym: *Contracaecum bancrofti* Johnston & Mawson, 1942]	*Pelecanus conspicillatus* (Type)[5] (Type of *C.bancrofti* J&M,1942)	Burnett River, Qld (Type *C.bancrofti*) SA, Qld, ?NSW
Contracaecum clelandi Johnston & Mawson,1942	*Pelecanus conspicillatus* (Type)[5]	Perth, WA (Type)
Contracaecum sinulabiatum Johnston & Mawson, 1942	*Plotus novaehollandiae* (Type)[5] *Phalacrocorax carbo*[5] *Phalacrocorax melanoleucus*[5]	Burnett & Thompson Rivers,Qld Qld SA

Table 1. Contracaecum spp. described from avian hosts in Australia and adjacent regions (Continued)

Species	Host	Locality
Contracaecum magnicollare Johnston & Mawson, 1942	*Anous stolidus* (Type)[5]	Capricorn Group, Qld (Type)
	Anous minutus[3][16]	Qld, Coral Sea, Lord Howe Is., New Caledonia
	Sula serrator[9]	NZ
	Diomeda cauta[8]	SA
Contracaecum microcephalum (Rudolphi, 1809)	*Anas bochas*[5]	Lord Howe Is.
	Anas superciliosa[5]	NSW
	Anas superciliosa[13]	Auckland Is.
	Larus novaehollandiae scopulinus[13]	Auckland Is.
Contracaecum tricuspe (Gedoelst, 1916)	*Plotus novaehollandiae*[5]	Qld
Contracaecum spp. (larvae)	*Xenorhynchus asiaticus*[5]	-
	Egretta alba[5]	SA
	Pelecanus conspicillatus[5]	SA
	Notophoyx novaehollandiae[5]	SA
	Eudyptula minor[5]	SA
Contracaecum spp.	*Pelecanus conspicillatus*[5]	WA
	Notophoyx pacifica[5]	Qld
	Notophoyx novaehollandiae[5]	SA
	Plotus novaehollandiae[5]	Qld
	Megadyptes antipodum[13]	Auckland Is.
	Himantopus leucocephalus[15]	SA
	Microcarbo melanoleucus[11]	SA
	Chlidonias leucopareia[11]	SA

Table 1. Contracaecum spp. described from avian hosts in Australia and adjacent regions (Continued)

Species	Host	Locality
Contracaecum heardi Johnston & Mawson, 1953	*Pygoscelis papua* (Type)[14] *Eudyptes crestatus*[14] *Eudyptes chrysolophus*[14] *Aptenodytes patagonica*[14]	Heard Is. (Type) Heard Is. Heard Is. Heard Is.
Contracaecum antarcticum Johnston, 1938 (larvae only - adult unknown)	*Pygoscelis adeliae*[14] *Aptenodytes forsteri*[14]	
Contracaecum eudyptes Johnston & Mawson, 1953	*Eudyptes crestatus* (Type)[13] *Megadyptes antipoda*[13]	Auckland Is. (Type) Auckland Is.
Contracaecum nycticoracis Johnson & Mawson, 1941	*Nycticorax caledonicus*[6]	NSW
Contracaecum pelagicum Johnston & Mawson, 1942	*Diomedea melanophris* (Type)[8] *Diomedea chlororhynchus*[8]	NSW (Type) NSW
Contracaecum podicipitis Johnston & Mawson, 1949	*Podiceps cristatus* (Type)[12]	SA (Type)
Contracaecum erraticum Johnston & Mawson, 1940	?*Plotus novaehollandiae*[4]	

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