

## Avian Parasites and Infectious Diseases

D. Keast

To cite this article: D. Keast (1977) Avian Parasites and Infectious Diseases, *Emu - Austral Ornithology*, 77:4, 188-192, DOI: [10.1071/MU9770188](https://doi.org/10.1071/MU9770188)

To link to this article: <https://doi.org/10.1071/MU9770188>



Published online: 22 Dec 2016.



Submit your article to this journal [↗](#)

---

# AVIAN PARASITES AND INFECTIOUS DISEASES

D. KEAST

## SUMMARY

KEAST, D. 1977. Avian parasites and infectious diseases. *Emu* 77: 188-192.

Current interest in the biological resources of Australia and the possible impact of large-scale ecological developments such as man-made lakes and national reserves demands that the parasites and infectious diseases (health patterns) of its wild fauna are fully delineated and studied in detail. A short selected review outlines aspects associated with avifauna, where contributions have been made to the understanding of the role of these organisms with respect to avifauna in general. Detailed study of the subject in Australia is lacking.

## INTRODUCTION

Lower invertebrates and micro-organisms such as protozoa, bacteria and viruses are without question involved in the development and architecture of both plant (Anon. 1970; Lieth 1974) and animal distributions (Davis *et al.* 1971) and hence environment on a larger scale. For the most part there is a balance of life in the environment, which sets the pattern of normality, and often it is only when some disturbance occurs to this balance that the micro-environment can express itself in a new or dramatic way or both.

Parasites are basically opportunists in that they can rapidly take advantage of, as well as engineer, conditions to their own reproductive advantage; the expression of this can be as disease of their hosts (Davis *et al.* 1971). One outcome is likely to be variation in the pattern of disease in the wildlife of the area or newly introduced animal population including man (Stanley and Alpers 1975). Thus a knowledge of the occurrence of disease and its frequency in wild fauna reflects environmental change, in particular at the level of the micro-ecosystem. The study of diseases of avifauna ought to give information on, and allow predictions in, several aspects, particularly with regard to the general health of the biological resources of a country and the likelihood of diseases being transmitted from the wildlife to man.

This review is not exhaustive but illustrates the subject.

## DISEASES OF BIRDS

Established patterns of disease characteristically express themselves in populations more often by lethal infections of susceptible young and old members of species than in other age-groups (Roitt 1971; Hungerford 1969). For any area there then becomes an established pattern of disease largely under the control of immunity within the host or genetic resistance or both through natural selection (Roitt

1971). The norm for a locality or geographical area is thus established and this *status quo* is upset only when the population of birds is put under stress or when a new disease organism is introduced. Representative parasitic and disease organisms of wild birds are listed in Table I.

Endemic diseases usually express themselves in individuals when they are under specific stress. For example, when wild birds are taken into captivity such diseases as psittacosis and ornithosis, bird pox, coccidiosis and virus-induced bronchial or pneumonal diseases can be fatal (Davis *et al.* 1971). Likewise the supply of new types of food often leads to an increase in the abundance of resident species and new species of birds can become established (Lack 1968). This may lead to the introduction of new diseases (Stanley and Alpers 1975), thus providing the double problem not only of the new disease but of the possibility of its expression in epidemic form. However, apart from epidemic disease, the real importance of disease in the control of wild birds is unknown and, although often mentioned as an ecological factor, it is rarely discussed in detail.

Predation probably exerts a strong influence in the control and the removal of weak members of wildlife communities (Lack 1954; Houston and Cooper 1975). It is well established that predatory birds take a high proportion of diseased animals (Rowley 1969, 1970) and that chronically diseased birds may be easier to shoot or trap (Lack 1966). Though Harris (1965) and Lack (1966) consider that over three per cent of a single species of bird suffering a single disease is insignificant in population dynamics, heavy parasitic levels may not lead to overt disease but may influence a bird's movements sufficiently to allow it to be taken by a predator. Therefore, in general, predation probably removes sick birds before they can be found by man. Thus research workers for the most part can only study diseases of wild animals in terms of carrier status and immunity of the fauna or when a large-scale epidemic disease occurs.

TABLE I

Examples of infectious organisms and parasites of wild birds with examples of their involvement in human health.

Classification	Disease syndrome in birds	Association with human disease
(1) <i>Parasitic</i>		
(a) Ectoparasites siphonaptera, fleas mallophaga, feather-lice diptera, mosquitoes	Non-specific malaise, loss of weight. High levels, loss of weight. Implicated in transmission of virus diseases. Irritation, blood loss, disease vectors.	? Carriers of arbovirus diseases, malaria
(b) Endoparasites nematodea, round-worms Many spp. cestodea, tapeworms  tremodea, flukes	Common non-lethal except at overload. Others e.g. gapeworm, cropworm, lethal by either numbers and obstruction or toxin production. General malaise, diarrhoea and death by starvation. Starvation, physiological stress, death.	Largely unknown
(c) Protozoa coccidia  trichomonadida, trichomonas plasmodia toxoplasma	+ 500 spp of coccidia often tend to be species specific. Carrier states common, diarrhoea and death. Often not lethal but can produce cankerous and upper respiratory syndromes and death. Avian malarias. Acute and chronic disease occurs. Toxoplasmosis. Widespread, can be non-pathogenic.	? Bilharzia, duck-shooters itch Plasmodia, Malaria Toxoplasmosis
(2) <i>Bacterial</i> Salmonella  Pasteurella Mycobacteria  Clostridia  Many others	Salmonellosis. Enteritis and death, carrier states common. Avian cholera. Epidemic deaths, enteric type. Tuberculosis. Chronic forms affecting various organs. Botulism. Seasonal disease, acute epidemic deaths, mainly associated with stagnant water. Chronic and fatal diseases common.	Salmonellosis   Botulism ?
(3) <i>Clamydial</i>	Psittacosis/ornithosis. Endemic and epidemic in form. Pneumonal, upper respiratory syndrome with diarrhoea.	Psittacosis
(4) <i>Mycotic</i> Aspergillus  Candidia	Avian aspergillosis. Chronic respiratory disease, mainly leads to death. Avian candidiasis. Respiratory and alimentary disease, acute/chronic leads to death.	Aspergillosis Candidiasis
(5) <i>Rickettsial</i>	Respiratory symptoms and death.	Q Fever
(6) <i>Viral</i> Parainfluenza—Newcastle disease virus Arbovirus—Encephalitic viruses Pox viruses	URTI, deaths, loss of weight.  Isolations of viruses have been made from birds with encephalitic symptoms. Tend to be host specific, produce acute fatal or self-limiting diseases, can be latent.	Encephalitis, polyarthritis, death

#### ROLE OF SEASONAL CHANGES IN THE ENVIRONMENT AND DISEASE

Little appears to be known of the effects of seasons on the patterns of disease in an area. Though most deaths are likely to occur in the young, newly

fledged individuals (Lack 1954), deaths from disease are probably not a result of any seasonal characteristic but represent the susceptible fledgelings experiencing the endemic diseases for the first time. However, some diseases are truly associated with the

season and this is best exemplified by botulism, a fatal epidemic disease occurring mainly in waterfowl and their predators (Lack 1954; Grubb 1964; Davis *et al.* 1971; Flegg *et al.* 1975). This disease is caused by a massive change in the micro-environment whereby a bacterium *Clostridium botulinum* can multiply under anaerobic conditions with the production of a lethal neurotoxin (Grubb 1964). These conditions are created by rotting vegetation in undrained stagnant pools and swampy areas mainly in late summer. Waterfowl consume the bacteria or toxin or both along with rotting vegetation and are killed. The birds as carcasses or sick animals can also be lethal to their scavengers and predators (Davis *et al.* 1971). This disease occurs often in urbanized areas such as parks, lakes and estuaries where man affects the flow of water and supplies of food of the waterfowl (Lack 1954; Grubb 1964; Davis *et al.* 1971; Flegg *et al.* 1975).

For the most part, seasonal factors such as climatic conditions and supply of food are likely to be responsible for epidemics (Lack 1954). These factors would lead to some periodicity of outbreaks of disease (Weise 1974) but this would only be recognized when the stress induced is high enough to lead to rapid depletion of numbers.

#### ROLE OF THE FOOD CHAIN

Life in the wild depends on food chains, which invariably end with the predators and scavengers (Lack 1954). Some of the natural parasitic diseases of birds depend on the food chain for completion of their life cycles (Rothschild and Clay 1957; Olsen 1974) and thus for their maintenance within the host pool. Once again it is only when these parasites come into contact with a weak undernourished naïve or physiologically stressed bird that overt disease is likely (Rothschild and Clay 1957).

However, avian predators and scavengers themselves are likely to transmit diseases of both vegetation (Anon. 1970) and animals (Houston and Cooper 1975). Also their nature of feeding and supplies of food make them recipients of toxins whether bacterial (Grubb 1964; Davis *et al.* 1971; Houston and Cooper 1975) or the results of chemical pollution of the environment, which have first taken a toll on performance of the predated animal (Davis *et al.* 1971; Naoya 1973). Though it is these factors in general that make the predatory bird or animal a monitor of environmental changes, in particular those at micro-environmental level, it ought not to be forgotten that some birds can be resistant to toxins (Sergent 1941; Cohen 1971) but themselves become poisonous to predators (Cohen 1971).

#### ROLE OF POLLUTION OF THE ENVIRONMENT

All life excretes waste to the environment and it is only when this becomes excessive that it pollutes. The effects of bacterial, viral and chemical pollutants on wildlife are now well known (Davis *et al.* 1971; Naoya 1973).

Outbreaks of salmonellosis in humans have been traced to areas of sewage disposal and seabirds have been implicated in the spread of the bacteria as well as being both real and potential reservoirs (Steiniger and Hahn 1953; Miller 1964). Human disease viruses of the arbovirus groups A and B have been implicated as being introduced or becoming established in reservoir form in immigrating birds following development of large man-made stretches of water (Stanley and Alpers 1975). Also the introduction of new diseases by these immigrating birds probably creates epidemic disease in what tend to be rather isolated bird communities (Stanley and Alpers 1975). Chemical pollution such as oil spills, pesticides and the fungal toxins from over-growth induced by pollution in the oceans have also been implicated in depletion of bird life (Naoya 1973). All of these examples fundamentally result from micro-environmental changes, induced on a large scale by man with almost complete disregard for anything other than his immediate environment.

#### ROLE OF HUMANS AND DOMESTIC ANIMALS

There is a range of diseases of birds that can also occur in man (Table I). Though the knowledge of some classical examples (psittacosis, see below) is fairly extensive, there is little detailed information on, and wide gaps in our knowledge of, many of the others. Often the organisms do not appear to cause disease in the wild birds but occur as latent reservoirs of parasites and micro-organisms awaiting environmental changes for explosive reproduction and the presence of man as the susceptible host (Stanley and Alpers 1975).

Probably the best-known human disease of wild-bird origin is psittacosis (Merrillees 1934; Meyer 1934, 1940, 1942, 1952; Burnet 1935, 1939; Pollard 1947; Lack 1954; Davis *et al.* 1971), which is endemic throughout Australia. However, the role of wild birds as reservoirs for arboviruses, in particular Murray Valley encephalitis virus and Ross River virus (Anderson 1953; Miles 1954; Shope and Anderson 1960; Doherty *et al.* 1966, 1970; Doherty 1972; Hore *et al.* 1973), is currently of greater interest because large reserves of water are being developed in tropical areas of Australia (Stanley and Alpers 1975). Likewise the re-introduction of malaria (Rothschild and Clay 1957) to Australia becomes more likely when one considers that migratory birds come to newly established lakes and

waterways of northern tropical Australia from areas to the north where malaria is endemic (Stanley and Alpers 1975). Furthermore the introduction of infected intermediate hosts (snails) by migratory birds may be all that is required to establish a complete vector cycle for traditional tropical diseases such as schistosomiasis (Stanley and Alpers 1975). Water, supply of food, populations of birds exploding both by breeding and immigration, insect vectors and human settlement, all contribute to an eco-system conducive to the establishment of diseases common to birds and man (Stanley and Alpers 1975).

Wild birds are probably hosts of some common disease viruses such as influenza (Alexander *et al.* 1974; Bahl *et al.* 1975) and fungal diseases can also present health hazards to workers with wild birds (Jackson 1973).

Obviously most diseases of wild birds are potential diseases of domesticated birds and the chain of contact between the two is all that is required for the transmission of disease (Lack 1954; Rothschild and Clay 1957). Nematodes and cestodes (Rothschild and Clay 1957), salmonellas and Newcastle disease virus are well established as means of cross infection (Davis *et al.* 1971). Furthermore large-scale bird migration as a result of development of large new areas of water in northern Australia may lead to the introduction of new disease organisms of veterinary importance. Probably one most feared is the virus of foot-and-mouth disease, which has been shown (Andrewes and Pereira 1967) to be present in areas over which migratory birds travel to Australia. Mechanical transmission by migratory birds is probably the only way of introduction of this virus and this is not likely (Murton 1964). Therefore changes in the micro-environment associated with birds, whether induced by massive modification of the macro-environment by man or not, can and do endanger man and his domesticated birds.

The ideal parasite is one adapted to take maximum advantage of the host without killing it (Rothschild and Clay 1957) and almost all the disease organisms of wild birds appear to occur usually in the endemic state in non-infectious and non-lethal form. Only when conditions in animals or the environment or both change to produce circumstances that encourage large-scale reproduction or metabolic changes will these micro-organisms become lethal.

Australian work in general appears to have concentrated mainly on diseases of wild birds likely to be lethal to man (zoonoses) such as psittacosis (Burnet 1935, 1939) and viral diseases of arbovirus origin (Anderson 1953; Miles 1954; Shope and Anderson 1960; Doherty *et al.* 1966, 1970; Doherty 1972; Hore *et al.* 1973). However, there has been

some detailed study of worm parasites (Bancroft 1889; Johnston 1912; Baylis 1934; Johnston and Mawson 1940). The whole problem of endemic diseases of Australian wildlife and their likely importance takes on new importance at the present time. Australia is looking in detail at its biological resources and large tracts of land are being set aside as national parks and reserves. Developments in the tropics (Stanley and Alpers 1975), in mining and wood-chipping, are likely to cause unprecedented rapid modification to the environment. All of these developments may have a profound influence on wildlife and as such may lead to a significant change in disease patterns, the importance of which is largely unknown in Australia.

## REFERENCES

- ALEXANDER, D. J., W. H. ALLAN, J. W. HARKNESS and S. A. HALL. 1974. Isolation of influenza virus from psittacines. *Res. vet. Sci.* 17: 125-127.
- ANDERSON, S. G. 1953. Murray valley encephalitis: a survey of avian sera, 1951-1952. *Med. J. Aust.* 1: 573-576.
- ANDREWES, C., and H. G. PEREIRA. 1969. *Viruses of vertebrates*. London: Bailliere, Tindall & Cassell.
- ANON. 1970. Birds may spread plant diseases far and wide. *New Sci.* 47: 457.
- BAHL, A. K., B. S. POMEROY, B. C. EASTERDAY and S. MANGUNDIMEDJO. 1975. Isolation of type A influenza virus from the migratory waterfowl along the Mississippi flyway. *J. Wildl. Dis.* 11: 360-363.
- BANCROFT, T. L. 1889. On the filariae of birds. *Proc. R. Soc. Qld* 6: 58 (Not sighted).
- BAYLIS, H. A. 1934. Some parasitic worms from Australia. *Parasitol.* 26: 129-132.
- BURNET, F. M. 1935. Enzootic psittacosis amongst wild Australian parrots. *J. Hyg.* 35: 412-413.
- . 1939. A note on the occurrence of fatal psittacosis in parrots living in the wild state. *Med. J. Aust.* 1: 545-546.
- COHEN, G. M. 1971. Studies on the resistance of roosters and vultures to type A botulinum toxin. *Dise. Abs.* 31B: 7545-7546.
- DAVIS, J. W., R. C. ANDERSON, L. KARSTAD and D. O. TRAINER. 1971. *Infectious and Parasitic Diseases of Wild Birds*. USA: Iowa St. Univ. Press.
- DOHERTY, R. L., B. M. GORMAN, R. M. WHITEHEAD and J. G. CARTEY. 1966. Studies of arthropod-borne virus infections in Queensland. V. Survey of antibodies to group A arboviruses in man and other animals. *Aust. J. exp. Biol. med. Sci.* 44: 365-377.
- DOHERTY, R. L., E. J. WETTERS, B. M. GORMAN and R. H. WHITEHEAD. 1970. Arbovirus infection in Western Queensland: serological studies 1963-1969. *Trans. R. Soc. trop. Med. Hyg.* 64: 740-747.
- . 1972. Arboviruses of Australia. *Aust. vet. J.* 48: 172-179.
- FLEGG, J. J. M., K. T. STANDRING and W. R. P. BOURNE. 1975. Mysterious bird deaths. *BTO News* 74: 4.
- GRUBB, W. B. 1964. Avian botulism in Western Australia. *Aust. J. Biol. Med. Sci.* 42: 17-24.
- HARRIS, M. P. 1965. Puffinosis on Skokholm. *Br. Birds.* 58: 426.

- HORE, D. E., J. CAMPBELL and A. J. TURNER. 1973. A serological survey for viral antibodies in wild birds. *Aust. vet. J.* 49: 238-239.
- HOLSTON, D. C., and J. E. COOPER. 1975. The digestive tract of the White-back Griffon Vulture and its role in disease transmission among wild ungulates. *J. Wildl. Dise.* 11: 306-313.
- HUNGERFORD, T. G. 1969. Diseases of poultry including cagebirds and pigeons. Australia: Angus & Robertson.
- JACKSON, J. A. 1973. Histoplasmosis—an occupational hazard for bird-banders? *Inland Bird-Banding News* 45: 52.
- JOHNSTON, T. H. 1912. Internal parasites recorded from Australian birds. *Emu* 12: 105-112.
- , and P. M. MAWSON. 1940. Some filarial parasites of Australian birds. *Trans. R. Soc. S. Aust.* 64: 355-361.
- LACK, D. 1954. *The Natural Regulation of Animal Numbers*. 1970 ed. Oxford: OUP.
- . 1966. *Population Studies of Birds*. Oxford: OUP.
- . 1968. *Ecological Adaptation for Breeding in Birds*. London: Methuen.
- LIETH, H. 1974. *Phenology and Seasonality Modelling*. London: Chapman & Hall.
- MERRILLEES, C. R. 1934. Psittacosis in Australia. *Med. J. Aust.* 2: 320-321.
- MEYER, K. F. 1934. Psittacosis in native South Australian Budgerigars. *Proc. Soc. exp. Biol. Med.* 31: 917-919.
- . 1940. Psittacosis. *Auk* 57: 330-332.
- . 1942. The ecology of psittacosis and ornithosis. *Medicine* 21: 175.
- . 1952. Reservoirs of the psittacosis agent. *Acta trop.* 9: 204-206.
- MILES, J. A. R. 1954. Infection of birds with Murray Valley encephalitis (X disease). *Aust. J. exp. Biol.* 32: 69-78.
- MÜLLER, G. 1965. Salmonella in bird faeces. *Nature, Lond.* 207: 1315.
- MURTON, R. K. 1964. Do birds transmit foot and mouth disease? *Ibis* 106: 289-298.
- NAOYA, A. 1973. A bibliography of man's environmental pollution affecting bird life. *Proc. Yamashina Inst. Orn.* 7: 96-137.
- OLSEN, O. W. 1974. *Animal Parasites: Their Life Cycles and Ecology*. 3rd ed. USA: Univ. Park Press.
- POLLARD, M. 1947. Ornithosis in sea-shore birds. *Proc. Soc. exp. Biol. Med.* 64: 200-202.
- ROITT, I. 1971. *Essential Immunology*. Oxford: Blackwell.
- ROTHSCHILD, M., and T. CLAY. 1957. *Fleas, Flukes and Cuckoos*. London: Collins.
- ROWLEY, I. 1969. An evaluation of predation by 'crows' on young lambs. *CSIRO Wildl. Res.* 14: 153-179.
- . 1970. Lamb predation in Australia: incidence, predisposing conditions and the identification of wounds. *CSIRO Wildl. Res.* 15: 79-123.
- SERGEANT, E. 1941. Les cailles empoisonneuse dans la bible, et en Algérie des nos jours. *Arch. Inst. Past. Algérie* 19 (2): 161-167.
- SHOPE, R. E., and S. G. ANDERSON. 1960. The virus aetiology of epidemic exanthem and polyarthrititis. *Med. J. Aust.* 1: 156-158.
- STANLEY, N. F., and M. P. ALPERS. 1975. *Man-made Lakes and Human Health*. London: Academic Press.
- STEINIGER, F., and E. HAHN. 1953. Über den nachivas von Keimen der typhus—paratyphus enteritis-gruppe aus vogelkot von der stora karlso. *Acta pathol. microbiol. Scand.* 33: 401-406.
- WEISE, C. M. 1974. *Phenology and Seasonality Modelling*. H. Leith (Ed.). London: Chapman & Hall.

D. KEAST, *Department of Microbiology, University of WA, Nedlands, WA 6009.*