

# Potential for appearance of transmissible diseases in farmed ratites in New Zealand

Infectious diseases and parasites, generally require a large population of susceptible birds before they pose a serious threat to flock health. Since there are at present only a few farmed ratites in New Zealand, the chances of a serious disease outbreak are small, but the high value of ratites has resulted in the generation of much information on the effects of a variety of infectious agents in these birds. These investigations have shown some interesting cross-species establishment of disease agents in ratites. As more is known about disease in poultry than other avian orders, much of the extrapolation is from poultry. Some of the pitfalls of this approach are highlighted. Since there are at this stage (1995) no animal remedies registered in New Zealand for use on emu and ostriches, any recommendations for treatment given by veterinarians should be tentative only, and owners of birds should be warned of this so that informed consent is possible.

## Emu and ostrich farming

Emus and ostriches are ratites (along with the kiwi, rhea and cassowary).

Ostrich farming has been practised in South Africa for 150 years and peaked in the early 1900s when the tail feathers were a fashion item. Ostriches were imported into Australia at that time, and after the collapse of the feather market were allowed to roam in the desert. Ostrich farms continued to exist in South Africa and interest began to pick up again slowly after 1945, when ostriches were farmed for their meat and skins. Ostrich farming also began in the then Rhodesia and South West Africa, and in the early '80s in Israel, Australia and North America. South Africa and Namibia have recently stopped the export of ostriches in an attempt to maintain dominance in the industry. Eggs have been imported to New Zealand from Australia, and live birds imported from the United Kingdom in 1995.

Emus are native to Australia and currently their export is not permitted. Emu farming for meat, skin and emu oil (from the abdominal fat) is well established in Western Australia. Birds have been sent to zoos and wildlife parks in New Zealand over the years and these formed the basis of the nascent New Zealand emu industry. In 1995 emus were hatched in New Zealand from eggs imported from Canada into medium security quarantine. General facts and figures on emus and ostriches are provided in Table I.

## Transmissible diseases of emus and ostriches

**Disease and the environment:** It is important to differentiate between infection with a disease agent (infection) and the presence of sick birds as a result of that infection. Often, infection becomes disease as a result of interaction with a stress, such as adverse environmental conditions, poor nutritional state or another disease agent. The same effect can be expected in birds with a naturally low level of resistance, such as very young birds. The establishment of a new farming industry, principally with imported stock, results in considerable stresses being placed on birds in pre-export isolation, in transport, in post-arrival quarantine, and even after release from quarantine through inexperienced management. The stresses of quarantine are an integral part of the quarantine process, but veterinarians need to take into account their existence when working with imported birds.

**Specific and non-specific disease agents:** A specific disease agent is one in the absence of which no disease occurs. For example, Newcastle disease does not occur without infection with Newcastle disease virus. Non-specific disease agents are frequently bacteria identifiable as the immediate cause of a problem in a sick

bird, but may be present in healthy birds from the same flock without causing a clinical problem. Non-specific bacterial septicaemia caused by *Escherichia coli*, *Salmonella* spp, *Proteus* spp, *Klebsiella* spp, and *Pseudomonas* spp appear as frequent findings in laboratory surveys of ostrich deaths<sup>(1,2)</sup>, especially involving chicks with retained yolk sacs. *Klebsiella* spp and *Pseudomonas* spp should be regarded as significant when cultured from swabs of hens' reproductive tracts.

## Specific bacterial diseases

**Tuberculosis:** *Mycobacterium avium* is the cause of tuberculosis in emus and ostriches. It is transmitted by sociable birds such as mynahs, starlings, and sparrows, and unusually for an infectious disease, is more likely to be a problem in older birds. An adult bird suffering chronic emaciation is the most likely presenting sign<sup>(3)</sup>. Tuberculin testing for *M. avium* has been carried out in poultry and in ostriches<sup>(4)</sup>, but a serum plate agglutination test is more usual in ratites. Human and bovine tuberculosis constitute a very small risk for emus or ostriches, and the industry should not be affected by any control measures that may be implemented to control the problem in cattle and deer. Significantly, humans are resistant to infection with *M. avium* so its zoonotic potential is low.

**Pseudotuberculosis:** *Yersinia pseudotuberculosis* causes disease in a variety of avian orders, and accordingly it is a possibility in ratites. It is commonly seen as a clinical entity (in orders where it is recognised) in stressed young birds, where death may follow a short bout of enteritis. In birds surviving the acute phase of the disease, progressive emaciation develops, as granulomas form in liver and spleen<sup>(5)</sup>. Diagnosis depends upon the isolation of the organism from spleen or liver of dead birds, or in a faecal sample. There are few recommendations as to an antibiotic of choice for treatment, but enrofloxacin has been effective in canaries. The organism is not very resistant in the environment.

**Erysipelas:** This disease is characterised by sudden death in stressed, usually youngish, birds. Postmortem features of this disease are distinctive, and usually involve haemorrhages around the heart. The organism is resistant in the environment, and gains entry to the bird's body via small cuts in the skin or mouth. Chronic infections do occur, and deaths due to heart failure result from growths on the heart valves<sup>(6)</sup>. It is unlikely that the first death on a property would be accurately diagnosed ante mortem but any subsequent cases would require rapid treatment with fast-acting penicillin or tetracycline injection, and the use of erysipelas vaccine on birds on the property.

Table I: General facts and figures on emus and ostriches

		Ostrich	Emu
Weight	Male	up to 150 kg	50 kg
	Female	up to 120 kg	40-50 kg
Egg weight		1.3 kg	600 g
Chick weight		800 g	400-450 g
Incubation period		42 days	56 days
Sexual maturity	Female	2-2.5 years	2-2.5 years
	Male	3 years	3 years
Eggs/year		40	30
Time of breeding		summer	winter
Intestinal length		long with functional caeca	short with rudimentary caeca
No. of toes		two	three
Economic products		leather	leather
		meat	meat
		feathers	oil

Erysipelas is mainly a disease of swine and turkeys, although it does occasionally affect sheep, in which species infection often takes place in contaminated docking pens<sup>(7)</sup>. Vaccination is carried out in both breeders and slaughter stock at a major emu farm in Western Australia.

**Pasteurellosis:** As far as is known, all species of birds are affected by fowl cholera<sup>(8)</sup>, with varying degrees of susceptibility, and therefore symptoms. Turkeys and wild waterfowl are most severely affected, with many sudden deaths. Chronic disease occurs in chickens, and this is the type of infection that would probably occur in ratites, manifesting as sporadic deaths in older birds in poor condition. That such an incident referred to by Huchzermeyer<sup>(9)</sup> occurred in a German zoo in 1900, together with the results of a serological survey of Zimbabwean ostriches<sup>(10)</sup>, which recorded very low levels of seroconversion to *P. multocida*, indicates that pasteurellosis is not a high risk infection in farmed ostriches. As specific signs of the disease are unlikely to be apparent, diagnosis would depend on culture from sinus swabs, or postmortem material. Treatment is by antibacterial (potentiated sulphonamide, enrofloxacin) injection. A commercial vaccine registered for use in poultry is available in New Zealand and would be the first line of defence if a problem property were identified.

**Campylobacter jejuni serotype 8:** This pathogen was consistently recovered from young (4 months) ostriches dying with a severe, extensive necrotising hepatitis, hydropericardium and ascites<sup>(11)</sup>. The birds produced a bright green urine prior to death, interpreted as being due to severe liver damage. Urine colour changes in ostriches occur normally with changes in diet<sup>(12)</sup>, and such changes should not be viewed in isolation. The affected birds were fed on lucerne pastures irrigated with water from a sewage treatment plant. The same organism was recovered from healthy birds in the flock.

**Chlamydiosis:** This condition is found in virtually all birds, and has been identified as a clinical problem in ostriches<sup>(13)</sup> and rheas<sup>(14)</sup>. The signs are of general malaise, especially in younger birds, or those with immune depression. Strains carried by different species of birds vary in virulence. For example, pigeons carry mild strains usually, whilst those associated with egrets and sea birds are virulent in a variety of other species, without any ill effects on the egrets or sea birds<sup>(15)</sup>. Postmortem examination of rheas dying of chlamydiosis has consistently shown enlargement of the spleen, with histological perivascular necrosis<sup>(14)</sup>. Liver necrosis has been a more variable finding, but was present together with necrotising splenitis in an outbreak of chlamydiosis in young ostriches recorded in South Africa<sup>(13)</sup>. A presumptive diagnosis may be made by finding intracytoplasmic chlamydial inclusions in splenic impression smears stained by the Gimenez meth-

od<sup>(16)</sup>, but definitive diagnosis requires isolation and identification of the organism in eggs or cell culture. An ELISA is available for detection of chlamydial antigen but lacks the specificity of egg inoculation or cell culture. A complement fixation test has proved useful in identifying seropositive turkeys and psittacines, but is unproven in other orders. Prolonged treatment (45 days) of affected birds and their flockmates with doxycycline is the therapy of choice.

**Mycoplasmosis:** Large numbers of mycoplasma isolates have been obtained from ratites including one from ostriches in New Zealand, and attempts have been made to type several dozen. Most do not fit with any characterised avian species; a few have been typed as *Acholeplasma laidlawii* or *Mycoplasma cloacale*, but their significance is unknown (SH Kleven, pers. comm.). The specific poultry mycoplasmas are not known to cause disease problems in ratites, and specific anti-mycoplasma control measures are not considered necessary at this stage. One untyped species isolated from conjunctival and nasal swabs from adult ostriches has been associated with poor viability and growth in chicks from mycoplasma-positive hens<sup>(17)</sup>.

If such problems appear in chicks, culture for mycoplasma should be part of a thorough disease investigation. Anti-mycoplasma therapy where vertical transmission of the agent is involved is a complex subject and expert advice should be sought.

**Clostridial diseases:** Mild gut problems (stasis, overeating) can lead to an overgrowth of anaerobic bacteria, leading to necrotic enteritis (*Clostridium perfringens*) or the more chronic ulcerative enteritis (*C. colinum*). *C. difficile*, which causes a fibrinous colitis, is a possibility in young chicks with intractable diarrhoea<sup>(18)</sup>. It is a difficult organism to culture.

Many antibiotics are effective against enteric clostridia, and the recent introduction of a suitable drinking water formulation of amoxicillin (*Paracillin SP*, Intervet) provides an effective and convenient treatment against clostridia. For farms where this is a recurrent problem, a variety of preventive drugs are available, such as bacitracin and avoparcin, which are unlikely to have any adverse effects on the normal gut flora.

Botulism in ostriches has been described from South Africa<sup>(19)</sup> as a paralytic condition causing varying degrees of paralysis from a mild ataxia to total paralysis and death. *C. botulinum* toxin type C was demonstrated in the carcass of one of the dead birds. Treatment of affected birds with type C antitoxin was effective in reversing clinical signs in affected birds. Given the intensive conditions under which ostriches are farmed in New Zealand, it is unlikely that outbreaks of botulism will occur here. Botulism outbreaks occur in waterfowl in late summer where birds

have access to ponds that are drying up. The *C. botulinum* bacteria multiply in the anaerobic sludge at the bottom of the pond, and toxin is especially concentrated in insect larvae feeding on rotting plant or animal material. Ingestion of these maggots by birds leads to ingestion of large amounts of botulinum toxin<sup>(20)</sup>. Drowsiness with droopy eyelids is likely to be seen before more severe paralysis. The period from ingestion of the toxin to appearance of signs has been recorded as being from 20 to 36 hours in chickens<sup>(21)</sup>. There are no postmortem changes that will confirm a diagnosis of botulism, but the finding of maggots or rotting plant material amongst the ingesta would provide evidence in support of a diagnosis of botulism. In South Africa, where ostriches are farmed more extensively, and the presence of carrion is more common, it is recommended that ostriches be vaccinated against botulism.

**Megabacteria:** These organisms have been recognised in budgerigars in New Zealand<sup>(22)</sup>. These large, bacteria-like organisms have been found to cause gastritis in 6 to 10-week-old ostrich chicks<sup>(23)</sup>. The organisms impair gizzard activity by invading deep within the koilin layer. Impaction of the gizzard is the clinical sequel of infection.

## Viral diseases of ratites

This review concentrates on those avian viral diseases known to be of significance in ratites, and mentions some well-known diseases of poultry that are unlikely to affect ratites. The most important viral diseases of ratites are Newcastle disease, avian influenza and fowlpox. Newcastle disease and avian influenza are exotic to New Zealand, whilst pox, although common from the Waikato northwards, is only rarely recorded further south.

**Newcastle disease:** Caused by a paramyxovirus classified as PMV1, Newcastle disease is exotic to New Zealand and Australia (although lentogenic, non disease-producing strains of the virus exist in both countries), but is the chief disease threat to all avian species world-wide. Newcastle disease is endemic in South America, Africa, Asia and parts of Europe, and is starting to encroach upon some of the Pacific Islands. The virus causing the disease comes in strains of a single genetic make-up, but highly different virulence. Some of the least virulent strains occur in Australia and New Zealand. Newcastle disease causes a variety of respiratory, intestinal and neurological problems in ostriches<sup>(24)</sup>. In a recent South African report<sup>(25)</sup>, the involvement of a paramyxovirus (not Newcastle disease) in a diarrhoeal syndrome was highlighted. A paramyxovirus was implicated by early investigations into the deaths of ostriches in Australia in 1995, but these deaths were later shown primarily to be due to nutritional and husbandry-related problems (J Edwards, pers. comm.). Experience with outbreaks

of Newcastle disease in European zoos suggest that ostriches are more likely to succumb to the disease than are emus or rheas<sup>(26)</sup>. In parts of the world where Newcastle disease is endemic, it is controlled in ostriches by means of live and killed vaccines. No measures specifically aimed at controlling this disease are required in New Zealand at present, but all suspicious deaths or illness should be investigated. Given the value of ostriches and emus, one would expect this as a matter of course.

**Avian influenza:** In contrast to Newcastle disease, avian influenza comes in myriad of strains, which are variable both in virulence and antigenic make-up, making vaccination virtually useless as a control measure, except in the face of an outbreak with an identified strain of influenza virus, which can be made into an inactivated vaccine<sup>(27)</sup>. Avian influenza has been recorded as causing disease in farmed ostriches<sup>(28)</sup>, as well as a range of other avian species<sup>(27)</sup>. Migrant birds are important in its spread, and of all the exotic avian diseases, influenza is the most likely to affect ratite stock in New Zealand. Avirulent avian influenza has been recorded in New Zealand waterfowl<sup>(29)</sup>. Drought conditions, which lower the resistance of wild birds, and encourage their congregation around water supplies, are important factors in the appearance of avian influenza in captive or farmed outdoor birds.

**Fowlpox:** This disease occurs in most avian species, and is characterised by growths/lumps on the featherless parts of affected birds. Clinical effects are proportional to the interference with an individual's ability to eat and drink. Pox is found in poultry mainly in the northern half of the North Island, where biting insects are important in the spread of the disease from farm to farm. It has also been recorded from wekas in the same area. Antibiotic treatment aimed at limiting secondary infection is vital in affected birds, whilst long-term control by means of an attenuated fowlpox vaccine registered for use in day old poultry has proved a viable option in a number of species in addition to poultry. Fowlpox vaccine has been used successfully in ostriches in Israel<sup>(30)</sup>.

### Other avian viral diseases

**"Poultry viruses":** Poultry, particularly chickens, suffer from a number of well-studied viral diseases and there is some interest as to whether these could infect ratites. Field cases of the neoplastic diseases Marek's disease (herpesvirus)<sup>(31)</sup> and lymphoid leucosis (retrovirus) are virtually unknown in species other than the domestic fowl<sup>(32)</sup>. Whilst a possible isolate of infectious bursal disease virus from an ostrich in Florida is under investigation in the USA (P Lukert, pers. comm.) and there are reports of positive antibody titres amongst ostriches<sup>(10)</sup> and other wild birds<sup>(33)</sup>, there is no evidence

to alter the statement that chickens are the only birds known to develop clinical signs of infectious bursal disease<sup>(34)</sup>. Hence the recent introduction of infectious bursal disease to New Zealand should not have any effects on the ratite farming industries here.

In a serological survey of ostriches in Zimbabwe<sup>(10)</sup>, 99% of 149 birds gave a positive result in an ELISA for turkey rhinotracheitis (TRT) antibodies. Since the equivalent disease in broilers, known as swollen head syndrome, had been recognised as 'dikkop' in South Africa for some years prior to the first isolation of the causative pneumovirus there in 1981<sup>(35)</sup>, the status of avian pneumoviruses in ostriches should be investigated further. The Zimbabwean work was carried out with commercial plates developed for use with turkey serum, and investigation of ostrich sera in the United Kingdom using a serum neutralisation test for TRT antibodies failed to show any evidence of their presence. (RC Jones, pers. comm.).

**Arboviruses:** Ostriches and emus are known to suffer from infection with a variety of arthropod-borne viral diseases, and new candidates appear from time to time. The significance of these for New Zealand depends on the presence of suitable vectors among the insects present in New Zealand. The potential for the spread of a variety of indigenous and exotic arboviruses amongst the human population has been reviewed<sup>(36)</sup>, and insects capable of spreading these viruses are found in New Zealand. It is not known whether the insects here would be able to initiate outbreaks should they feed on viraemic travellers or imported birds, or whether long-term hosts of the various viruses exist amongst the native and introduced birds and mammals here.

**Eastern/western equine encephalomyelitis:** These are insect-borne diseases affecting ratites in North America. Transmission to emus and ostriches depends on the presence of large numbers of passerine birds, which serve as a reservoir of infection for the mosquito vectors. Neurological signs are not always present in ratites, and one report attributed an acute haemorrhagic colitis to EEE infection<sup>(37)</sup>. Vectors recorded in North America include mosquitoes of the genera *Culex*, *Mansonia* and *Aedes*<sup>(38)</sup>. Whether suitable vectors would exist among related New Zealand mosquitoes is unknown, hence precautions are needed to ensure that the agents are not imported from America. These diseases are not transmitted through the egg. Serological freedom from these infections is a requirement for importation of ostriches to New Zealand from Canada, even though the disease usually occurs in southern states of the USA.

**Wesselsbron disease:** This is a sporadically occurring disease of sheep in southern Africa. It is caused by a flavivirus,

and is transmitted primarily by mosquitoes. In 1992, the virus was isolated from a flock of 250 ostrich chicks, after 90% had died at about 4 months, an age at which chicks are fairly hardy. Splenomegaly was the only finding at postmortem examination. The investigators were unable to exclude egg transmission in ostriches<sup>(39)</sup>.

**Murray Valley encephalitis, Ross River virus:** These are Australian arboviruses. Antibodies to MVE have been found in representatives of a wide variety of avian orders in Australia, but there is no record of emus having been tested. The potential significance of MVE in ostriches is unknown at this stage. Ross River virus (RRV) was the cause of a major epidemic in humans in Fiji but died out because there was no suitable mammalian maintenance host<sup>(35)</sup>. It has probably been introduced by travellers to New Zealand, but not become established here. Available evidence suggests that most birds are quite refractory to infection with RRV<sup>(40)</sup>.

**Borna disease:** This has been isolated in Israel from young ostriches with paralysis<sup>(41)</sup>.

**Reoviruses, adenoviruses and herpes viruses:** There are large numbers of isolations of these viruses reported from various avian species, only a few of which are related to specific diseases, for example viral arthritis of chickens (reovirus) or herpesvirus infection of cranes<sup>(42)</sup>. Recently, the consistent isolation of an adenovirus from ostrich chicks showing liver lesions was reported<sup>(17)</sup>. An adenovirus was isolated from ostrich chicks in Italy<sup>(44)</sup> and was shown to be capable of producing pancreatitis in guinea fowl keets similar to earlier reports of adenoviral pancreatitis in guinea fowl. Given the high value of ostriches and emus, there will be a disproportionately large effort put into the isolation of viruses from ratites, and many will be recovered. Interpreting their significance will not be easy as ostrich chicks are unlikely to be available for passage, and isolates will have to be passaged in other species.

**Spongiform encephalopathy:** Three adult birds died in a German zoo in the late 1980s showing clinical and histological signs analogous to those found in cattle with bovine spongiform encephalopathy<sup>(45)</sup>. The investigators did not have access to fresh material to check for the presence of scrapie fibrils. The birds were fed on a mixed diet including animal protein.

### Fungal infections

**Aspergillosis:** Inhaled fungal spores develop on the air sacs and in the lungs (usually) to cause gasping breathing, loss of weight and, eventually, death in young (5 days to 8 weeks) birds. Birds seen to be affected when very young have probably been infected in the hatchers. Aspergillosis in such young birds develops into a

systemic infection with heavy losses. In older chicks, respiratory distress is not always a feature<sup>(46)</sup>; the birds may simply fail to thrive and have a higher than normal mortality. This emphasises the importance of carrying out postmortem examinations of chicks under these circumstances.

Treatment is seldom effective, but some success has been achieved by nebulising *Clinafarm* spray (enilconazole) or *Clinafarm* smoke bombs. Enilconazole is not readily available in New Zealand, but the human analogue itraconazole (available as *Sporanox* 100 mg capsules) is effective, especially when given as a prophylactic medication to high risk hatchings. Alternately fogging premises or birds with *Virkon S* (0.5%) (*Antec*) may be useful. Control measures include fumigating hatcheries and setters with formalin, *Virkon S* or *Clinafarm* spray or smoke bombs.

Maintenance of good brooder hygiene by fumigation as above prior to use, and avoiding damp conditions in brooders are also important.

Aspergillosis also causes chronic weight loss in older birds<sup>(47)</sup>.

**Zygomycosis and thrush:** These infections occur as an overgrowth in the upper gastro-intestinal tract (gizzard, proventriculus and oesophagus) of affected chicks, usually after prolonged treatment with oral antibiotics that kill the bacteria normally resident in these parts<sup>(48)</sup>. The bacteria produce natural antifungal substances. Nystatin or copper sulphate have been recommended for use in treating these fungal conditions. The use of a narrow spectrum antibiotic for treating intestinal conditions, where a diagnosis makes this possible, will lessen the adverse effects of antibiotic therapy on the intestinal flora.

**Mycotoxins:** Aspergillosis and zygomycosis are the direct result of fungal infection inside the bird. If the fungus grows in the feed, or feed grain prior to harvest, it may produce toxins which when ingested cause problems in the birds. There have been a large variety of mycotoxins identified from maize, barley, wheat and soya meal. Indeed, any feed ingredient infected with a fungus is a potential source. Mycotoxins are difficult to diagnose and impossible to treat. Care with choice of feed supplier, and ensuring feed is stored in a cool dry place, used in the order it was bought, and not stored for too long, will help to avoid problems.

## Protozoal diseases

**Coccidiosis:** Infections with coccidia are found in all species of birds but are very specific disease agents. Coccidia of chickens do not cause disease in turkeys, let alone in ratites. Coccidia usually cause an enteritis, but occasionally cause problems in kidneys (goose) or liver (rabbit).

Coccidia, both *Isospora* and *Eimeria*, are found very rarely in ostriches<sup>(49)</sup> and emus<sup>(50)</sup>. Coccidiosis is very much a disease of intensification, and given the rarity of the condition in South Africa's more intensive industry, and the low numbers of birds present in New Zealand, an outbreak of coccidiosis is unlikely. The treatment of choice would be toltrazuril at 12.5 mg/kg.

**Histomoniasis (blackhead):** This protozoal infection is very common in peafowl, turkeys and chickens on range. It is carried in the eggs of caecal worms (*Heterakis* spp.) of these birds, and when these eggs are ingested, they protect the protozoan through the harsh environment of the proventriculus and gizzard. When the eggs hatch out in the small intestine and the worms begin their development, the protozoan is liberated and begins its development. If the eggs of a galliform parasite are eaten by a ratite, the worm eggs do not develop into adult worms, but the protozoan is still liberated in the small intestine and able to develop. The disease is more significant in ostriches because of the development of their caeca, but also occurs in emus. Control involves the prophylactic feeding of dimetridazole to young birds sharing range with galliforms. Dimetridazole should not be given to breeding emus or ostriches.

Other flagellated protozoa have been recovered from faecal samples of ostriches in quarantine. These appear to be trichomonads, but their relationship to *Trichomonas gallinae*, the cause of trichomoniasis in pigeons, is unknown. Flagellate protozoans of the lower intestinal tract of birds have not been shown to be pathogenic in avian hosts<sup>(51)</sup>.

**Cryptosporidia:** These are closely related to coccidia, but behave somewhat differently in that the parasites infect the respiratory tract in addition to the gut. Further, they have a dual lifecycle, with double walled oocysts, as do coccidia, and single walled oocysts which do not pass out of the body but re-infect the same host to give a rapid build up in infection. There is cross species infectivity. That is, the same parasites could infect game birds and ratites. Clinical disease is not common but cryptosporidiosis should be considered in cases of mild enteritis or runny nose/respiratory distress, especially in young birds. Cryptosporidia have also been found infecting pancreatic ducts in birds with pancreatic necrosis<sup>(52)</sup>. No treatment is registered against cryptosporidia, but toltrazuril would probably help. Most cases resolve if the general condition of the stock is rectified.

## Intestinal parasites

**Lybostromylus douglassi:** The proventricular worm of ostrich chicks is carried by adults and is pathogenic in chicks, causing a haemorrhagic gastritis and

proventriculitis. Heavy infestations result in high mortality rate in young birds on intensively grazed pasture.

**Codiostomum struthionis:** This is a larger, but apathogenic, worm of the lower intestine of ostriches. As the eggs are very similar to *L. douglassi*, it is possible to misdiagnose the presence of the latter unless a larval culture is carried out. In addition, ostriches on range will also ingest sheep and cattle helminth eggs which can be passed out in the faeces.

**Tapeworms:** A large tapeworm over 60 cm in length, *Houttuynia struthionis* has been recorded from the small intestine of ostriches. It causes ill thrift in young birds on pasture in South Africa<sup>(53)</sup>. The significance of tapeworms in emus is unknown. If treatment is needed, use praziquantel (*Droncit*) at 7.5 mg/kg. All tapeworms have an intermediate host, which in the case of land-living birds is usually an insect (fly or beetle). As the precise intermediate host in South Africa of *H. struthionis* is unknown, it is not possible to say whether it could become a problem in New Zealand. It has not been recorded in Australia.

**Cerebral nematodiasis:** In USA a cerebral worm has been recorded as a cause of neurological problems in emus of about 3-5 months of age<sup>(54)</sup>. The normal host is the raccoon, and the presence of raccoons in ratite pens in North America is discouraged. Neurological signs result. Ivermectin treatment at 400 µg/kg (2x sheep dose) is recommended. Alternately 200 µg/kg monthly from 12 weeks to 24 months in affected areas.

At present, there are too few emus/ostriches in New Zealand to build up infection to levels likely to cause disease, but prophylactic anthelmintic treatment is advisable at about 4 months and when birds are moved onto a property for the first time.

## Lice and mites

**Feather louse:** *Struthiolipeurus struthionis* is a large louse 2-5 mm long. Eggs are white, spindle-like and 1-2 mm long. They are laid in a herringbone pattern up the main shaft of the feather. Lice can be treated by spraying or dusting with malathion, repeated after 2 weeks, and then again after a moult when new feathers emerge<sup>(56)</sup>.

**Ostrich feather mite:** *Pterolichinus* spp are 1-2 mm long and lays brown eggs 0.75 mm diameter along the barbs perpendicular to the shaft. Mites can be treated with malathion, but may be difficult to get rid of.

Mites are frequently found around the head of young emus. Whilst ivermectin will help, a spray administered by a knapsack or hand-held garden sprayer would be suitable. Concentrate on area around the ears (protect eyes) and vent.

## Potential effects of transmissible disease of ratites in New Zealand

New Zealand has a unique avifauna, including native ratites in the three species of kiwi. Since the current introductions of emus and ostriches are for farming purposes, the chances of contact between them and kiwis are minimal at present. However, new farming ventures have failed in the past, and the introduced species left to roam, so precautions are justified. Emus and ostriches have been held in the same institution in New Zealand (Orana Park, Christchurch) for some years with no apparent ill effects. These birds came from Australian zoological collections and therefore constitute a lesser risk than birds imported from countries of poor avian disease status. Kiwis and other New Zealand native birds held in overseas institutions have at least indirect contact with birds in countries where many of the avian diseases discussed in this review occur. Serological screening of these overseas birds for some of the agents discussed here might yield interesting information as to which disease agents New Zealand natives have been exposed to without clinically apparent effects.

The avian bacterial diseases discussed have occurred in New Zealand, some in native species<sup>(22)</sup>, and the rise of ratite farming poses minimal extra risks so far as bacterial disease is concerned. Metazoan parasites tend to be host specific, but there have been unfortunate cross species transfers. Extensive testing and treatment during quarantine will reduce the risks of any introduction of such organisms. The main risks lie with the viral diseases where cross species changes in disease manifestation occur and are difficult to predict. With the dismantling of non-tariff barriers to trade, there have to be sound technical reasons for preventing importation of a livestock species. The possibility that they might have unknown effects in unspecified indigenous species should not be considered as adequate grounds to prevent the development of a commercial venture in New Zealand.

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