REVIEW ARTICLE

Bacterial, fungal and parasitic infections in the ostrich (*Struthio camelus* var. *domesticus*)

Ross G. COOPER

Department of Physiology, University of Central England, Birmingham, UK

ABSTRACT

The ostrich is susceptible to microorganisms of bacterial, fungal and parasitic origin. Anthrax, caused by *Bacillus anthracis*, is dangerous to other livestock and humans. *Salmonella* is transmitted from rodents or wild bird reservoirs. Pausterellosis caused by *Pasteurella multocida* results in air sac infections in ostriches. Colibacillosis is caused by *Escherichia coli*. Tuberculosis caused by *Mycobacterium avium*, is very rare in ostriches. Aspergillosis principally afflicts chicks. Zygomycosis, a secondary fungal infection of the upper gastrointestinal tract, is caused by *Basidia*, *Mucor* and *Rhizopus*. *Leucocytozoon struthionis* and *Plasmodium* spp. are harmless protozoa transmitted from flying arthropods. The tapeworm, *Houttuynia struthionis*, is dangerous in young ostriches. The adult ratite fluke (*Philophthalmus gralli*) is transmitted to ostriches following ingestion of infected freshwater crustaceans. Tick infestations of ostrich skin in Africa include *Amblyomma* spp., *Haemaphysalis punctata*, *Hyalomma* spp., *Rhipicephalus turanicus* and *Argus* spp. The ostrich quillmite (*Pterolichus bicaudatus*) and louse (*Struthioliperus struthionus*) may lower skin and leather quality via pruritis and/or excessive preening and feather loss. Nemododa infections are rare.

KEYWORDS: bacteria, fungi, ostrich, parasites, treatment.

INTRODUCTION

The ostrich (*Struthio camelus* var. *domesticus*) is an important animal in the commercial farming sector. Like with other livestock, productivity of this bird is at threat from diseases. The aim of the present review is to discuss bacterial, fungal and parasitic diseases afflicting the ostrich. It forms an extension of an earlier paper on viral infections (Cooper *et al.* 2004). An attempt, where possible, was made using the current literature to comment on each disease based on its etiology, pathology, transmission, zoonosis, diagnosis, prophylaxis and treatment, and control measures. The article also discusses diagnostic tests for microbial infections and the vaccination against some diseases of the birds, and suggests avenues for further research. It is hoped that the present article will encourage a focus on the usage of medicine in an attempt to maximize productivity on the farm.

BACTERIAL INFECTIONS

Anthrax

Aetiology

Anthrax is caused by *Bacillus anthracis* a highly zoonotic bacterial infection reported in ostriches in Africa (Snoeyenbos 1965; Ebedes 1997; Verwoerd 2000). Reports found in the archives include Robertson (1908) and Theiler (1912). It is, however, rarely seen in ostriches although producers are advised during anthrax outbreaks in other livestock, to allow

peracute necropsy of any bird that dies (Huchzermeyer 1997a).

Pathology

Pathological examination reveals extreme splenemegaly, hepatomegaly and vascular congestion. Anthrax bacteria are present in the blood stream and can be demonstrated in stained blood smears (Huchzermeyer 1997a). Due to the high infectivity of this disease it is essential that ostrich farms are not established in areas previously infected with anthrax, as all exposed birds will have to be killed.

Transmission

Ostriches may become infected by ingesting spores while grazing in areas of high soil contamination.

Zoonosis

Although there have been no reported cases of transmission to humans, it is possible that this may occur in slaughter personnel working in an ostrich abattoir.

Diagnosis

Bacterial growth is usually evident within 6–24 h. *Bacillus anthracis* may also be isolated from skin lesions (in the case of cutaneous anthrax) or respiratory secretions. Careful microscopic examination of stained smears of blood, vesicular fluid, or edema fluid may reveal the presence of *B. anthracis*. Edema fluid may be the first sample choice for suspected chronic infections, because *B. anthracis* usually only becomes bacteremic shortly before death.

Prophylaxis and drug treatment

Provost and Perreau (1978) discuss the use of combined vaccines in developing countries to increase convenience of use and efficiency of prophylactic projects in the field. Indeed, if biological compatibility of immunogens between anthrax and Newcastle disease were developed, it would be most beneficial in protecting birds from these diseases.

Control measures

Management of anthrax in livestock includes quarantine of the affected herd, removal of the herd from the contaminated pasture (if possible), vaccination of healthy livestock, treatment of livestock with clinical signs of disease, disposal of contaminated carcasses (preferably by burning), and incineration of bedding and other material found near the carcass.

Salmonellosis

Aetiology

Salmonellosis is a bacterial disease caused by *Salmonella*, microbes arising from contaminated facilities through contact with rodents or wild bird reservoirs. Higgins *et al.* (1997) reported salmonellosis involving cows, a goat and an ostrich aged more than 6 months. Although cases were diagnosed in four different regions, antimicrobial susceptibility patterns, phage-typing and pulse-field gel electrophoresis analysis demonstrated that all isolates belonged to one clone.

Pathology

Salmonellae that are host-specific avian pathogens are avirulent in mammals and those that are non-specific are commensals in poultry (Henderson *et al.* 1999). Common serotypes of Salmonella in ostriches include *S. pullorum, S. gallinarum,* and *S. typhimurium* (Ley *et al.* 2000).

Transmission

Inadequate housing and nutrition of the birds may leave them susceptible to salmonellae infections (Tully & Shane 1996). Poorly constructed pens often result in severe injure to ostriches especially to chicks. Ostriches may also be exposed to bacteria following injury during loading particularly if the loading ramp is poorly constructed. Strict hygiene practices in abattoirs ensure that contamination of carcasses is highly unlikely (Cooper 1999a,b, 2000a).

Zoonosis

In ostrich abattoirs in Zimbabwe, the animal health inspector ensures good hygiene, a practice supported by Gopo and Banda (1997) who demonstrated that the main ostrich products destined for export (meat, bone meal and ostrich fillet) were negative for *Salmonella*.

Diagnosis

In a study (Gopo & Banda 1997), a total of 1429 samples were collected from fillet, liver, gizzards, blood-meal, skins, heart, feces, large and small intestines, carcasses, wash-water from feathers and from carcasses, and other sources during the ostrich processing. These were screened for *Salmonella* using a *Salmonella*-specific DNA probe (1.8 Kb). Dot-blot sensitivity tests were carried out to estimate the approximate concentration of *Salmonella* bacterial cells in each

positive sample. *Salmonella* positive samples included 16.9% of the total and 50.8% of all ostriches were positive for *Salmonella* upon arrival at the slaughterhouse. The authors decided that the birds might have been contaminated on the farm, during transportation, or at the abattoir. Products determined as *Salmonella* positive included gizzards (5%), skins (8.3%), bloodmeal (4.2%), large intestines (26.2%), small intestines (16.1%) and feces (44.2%). Of these, the only exportable products are the skins, although the low contamination is likely to be eliminated during tanning. It was suggested that Zimbabwean ostrich producers should enforce decontamination control measures and strict hygiene through establishment of separate handling facilities for handling birds and product processing. The source of bacterial infections of the skin may arise through fecal contamination during defeathering (Huchzermeyer 1997b; Geornaras *et al.* 1998).

Prophylaxis and drug treatment

Salmonella transmission from infected poultry to ostriches is common and occurs particularly on farms where poultry are allowed to wander around freely. The necessity to vaccinate chickens cannot be emphasized enough. A recent study investigated the efficacy of Mucosal Starter Culture (MSC) for preventing salmonellae in broiler chickens (Bailey *et al.* 2000). The use of MSC treatment in ostriches may prove useful.

Control measures

Reduction of the risk of subclinical carriers is prevented if a whole blood *S. pullorum* agglutination test is conducted before shipment and routine screening of birds is done by cloacal swabs during quarantine (Foggin 1992).

Escherichia coli infections

Aetiology

Bacterial disease syndromes involving associated *Escherichia coli* infection have been rarely described in ostriches, although there is one by Foggin (1992).

Pathology

Infection also occurs when the umbilicus is not disinfected (Foggin 1992). Symptoms include weakness and rapid death in neonatal chicks within the first 10 days. Chicks hatch very weak if infection occurs in the egg. Pathological symptoms include an inflamed, reddened yolk sac within the abdomen, sometimes with strands of pus or milky pus. The umbilicus is moist and 'cheesy' material is observed within the abdomen.

Transmission

Escherichia coli may cause omphalitis, a navel and yolk sac infection, due to wet bedding in the hatchery or neonatal-chick house.

Zoonosis

Transmission to man may occur in abattoir personnel. Septicemia may also be caused by *E. coli* infection. Ley *et al.* (2001) reported that ostrich carcasses sampled from eight slaughterhouses did not have *E. coli* O157:H7, although 91% (116/128) of the dressed carcasses sampled had *E. coli* present. For the large intestinal sampling, 149 of the 217 (69%) samples had *E. coli* present. Fifty of these 149 samples had *E. coli* levels ranging from 10^2 to 10^5 colony-forming units/g feces. Antimicrobial susceptibility testing on 131 intestinal *E. coli* isolates showed resistance to erythromycin (98%), neomycin (66%), netilmicin (34%), oxytetracycline (34%), streptomycin (40%), and trimethoprim (13%).

Diagnosis

This includes culture of swabs taken from the yolk sac, umbilicus and abdomen. An antibiotic sensitivity test is also performed. Prevention is vital as treatment is usually too late. It is important to provide aseptic conditions in the incubator and hatchery, to rapidly dry the umbilicus, and provide dry surfaces for neonatal chicks. Disinfection of the umbilicus is not normally a problem as most producers disinfect the navel with gentian violet spray (Cooper 2000b,c).

Prophylaxis and drug treatment

None is described. The producer should avoid routine administration of antibiotic in drinking water of neonatal chicks, as it will destroy the bacteria in the intestine. Coprophagia is advantageous as it allows ingestion of bacteria and the strengthening of the acquired immune response, and supplies essential B vitamins (Foggin 1992).

Control measures

It is important to provide aseptic conditions in the incubator and hatchery, to rapidly dry the umbilicus, and provide dry surfaces for neonatal chicks.

Disinfection of the umbilicus is not normally a problem as most producers disinfect the navel with gentian violet spray (Cooper 2000c).

Colibacillosis

Aetiology

Colibacillosis is a bacterial infection caused by *E. coli*, a ubiquitous, Gram-negative enterobacterium that may be innocuous, although pathogenic strains result in primary disease. Reports of disease are rare. Kolb *et al.* (1993) reported pathological lesions in a group of ostrich chicks with high mortality including colibacillosis and isolation of *E. coli*. *Chlamydia* spp. were shown as being associated with the colibacillosis (Kolb *et al.* 1993).

Pathology

Chicks are susceptible to *E. coli* infection if afflicted by an underlying viral or fungal infection; nutritional deficiency or excess; and a dysfunctional immune system (Foggin 1992). At necropsy, small, yellowish-white nodules are found throughout the hepatic parenchyma. In the early stages, they may be sharply demarcated, while in the later stages, they coalesce. The early lesions have a milky content; older lesions may have a more cheese-like consistency.

Transmission

This occurs via fecal/oral routes and the agent is identified with cloacal swabs (Tully & Shane 1996).

Zoonosis

Although this has not been reported it is conceivable that farm laborers would be most at risk.

Diagnosis

Microscopically, the nodules are composed of hypertrophied bile ducts.

Prophylaxis and drug treatment

Antibiotic treatment is essential for the treatment of colibacillosis. Bacterins use cultures isolated from affected birds to boost immunity in resident animals. Antibiotic treatment is essential for the treatment of *E. coli* infection and autogenous bacterins are used when colibacillosis develops into a health problem on the farm. Bacterins use cultures isolated from affected birds to boost immunity in resident animals. The authors, however, note that the efficiency of bacterins

in ratites is unknown and the most suitable prevention measures for colibacillosis include proper management and nutrition, and reduction of stress in the flock or during transport.

Control measures

Treatment will not be successful unless a sanitation program is instituted simultaneously. Feed containers and water baths should not become contaminated with feces. Pens should be kept dry, and the accumulated feces removed frequently. Some chemicals, such as 10% ammonia solution, are lethal to oocysts and may be used to disinfect cages or ancillary equipment exposed to fecal material.

Pausterellosis

Pathology

Pausterellosis is a bacterial infection caused by *Pasteurella multocida* resulting in air sac infections in ostriches (Huchzermeyer 1997b). Intensive ostrich farming may increase the incidence of *Pasteurella* and exposure from the environment or through direct contact with recovered *Pasteurella* carriers provides ample opportunity for acquisition of this bacterium in immunosuppressed ratite flocks (Tully & Shane 1996). Ostriches lack lymph nodes in their organized lymphatic system. Their mechanism – common to all birds and reptiles – to prevent the spread of local infections to the blood (septicemia) consists of exuding fibrin into the infected area. This immobilizes the infectious agents but also the mobile blood cells of the immune system. This is effective as a first defence. The swelling resulting from the accumulation of fibrin is called 'fibriscess' (Huchzermeyer 1997b).

Diagnosis

The clinically diseased bird shows non-specific respiratory signs and generalized vascular congestion may be observed on gross necropsy specimens (Tully & Shane 1996). Injured ostriches do not have to be treated with antibiotics to prevent septicemia due to the accumulation of fibrin (Huchzermeyer 1997b).

Prophylaxis and drug treatment

There are no effective vaccination protocols for ostriches and exemplary standards of hygiene and quarantine are needed to prevent introduction of infections into ostrich flocks (Tully & Shane 1996).

Tuberculosis

Aetiology

Tuberculosis is a bacterial infection caused by *Mycobacterium avium*. Huchzermeyer (1997b) described the infection as being rare and generally limited to birds in zoos where there is contact with other avian species. *Mycobacterium avium* has been diagnosed in ostriches in the USA and Canada (Shane *et al.* 1993). Mycobacterium infection has also been reported in ostriches in Australia (Doneley *et al.* 1999; Cousins *et al.* 2000).

Pathology

In the study by Doneley *et al.* (1999) a three-year-old ostrich hen was examined to assess weight loss of several months. Physical examination revealed emaciation, scant urine output, small pelletized droppings, and reduced skin tone. Clinical pathology tests demonstrated the presence of leukocytosis (60×10^9/L, reference rangeL 10–24), monocytosis (16.2×10^9/L, 0–1), increased serum total protein concentration (110 g/L, 24–53) and increased serum globulin (79 g/L, 14–31). Cloacal palpitation was done to assess the degree of constipation and numerous small nodules were detected. White nodules (5–10 mm in diameter) were seen following eversion of the cloaca and they were covered with caseous pus. Histopathological analysis of a biopsied specimen of the cloaca revealed chronic granulomatous cloacitis with many acid-fast bacilli within macrophages in the lamina propria. Following euthanasia, the bird was necropsied. Emaciation was evident with bright yellow subcutaneous and internal body fat. The enlarged liver had multifocal, densely packed white nodules (<5 mm). Similar nodules were observed in the entire length of the colon and caeca, and extending into the cloaca. The caeca exhibited the most pathology with the surface of the distal two-thirds appearing as a yellow-fissured membrane. The lumen contained a clear, gelatinous fluid in which some flocculent material was suspended.

Transmission

Transmission of *M. avium* occurs through infected feces from sick birds. The microbe is virulent remaining viable in the soil for up to 12 months (Tully & Shane 1996). As a consequence, spores are easily transmitted on shoes, vehicle tyres, and may be dispersed in dust, and in mud during rain showers.

Zoonosis

Tuberculosis is zoonotic as it persists in wild bird populations and may infect pigs, cattle and immunosuppressed humans (Tully & Shane 1996) particularly those individuals infected with human immunodeficiency virus (HIV 1 & 2). Cousins *et al.* (2000), in their study of 71 isolates of *M. paratuberculosis* from cattle, sheep, goat, alpaca and rhinoceros, recorded that most isolates were cattle strains falling into C1 ($n = 28$) and C3 ($n = 32$) groupings. All isolates from alpaca were type C1 and bovine isolates were C1 ($n = 15$) or C3 ($n = 28$). All sheep were infected with sheep strains, none of which were identified in cattle.

Diagnosis

Histopathological analysis revealed the presence of multiple granulomas in the mucosa and submucosa of the intestines. Most granulomas had a central area of coagulative necrosis surrounded by giant cells, epithelioid macrophages, plasma cells and lymphocytes. Immature tubercles composed of focal aggregations of epithelioid macrophages and occasional heterophils were also present.

Control measures

Mycobacterium avium attacks the avian gastrointestinal system and clinical signs are those of non-specific wasting. Confirmation of infection is done by necropsy. An intensive ante-mortem physical examination may be performed in which the acid-fast organism is isolated from a bacterial granuloma or a fecal screen (Tully & Shane 1996). The authors suggest, due to the unreliability of screening tests using fecal acid-fast methods, intradermal testing is recommended plus the maintenance of closed flocks, examination of newly acquired breeding stock and good management. Ostrich farms should be set up in areas free of tuberculosis.

FUNGAL DISEASES

Aspergillosis

Aetiology

Aspergillosis is a fungal disease caused by *Aspergillosis fumigatus* and other species. Ostrich chicks are particularly susceptible to aspergillosis, the most susceptible individuals being young birds kept in enclosed facilities and exposed to dust or hay which is alternatively wet or dry (Tully & Shane 1996). Indeed, chicks

housed in areas with high rainfall are susceptible to this disease by 32% (Cooper 2000c).

Pathology

Aspergillosis infection in eggs indicated by dead-in-shell embryos is rare due to stringent standards of hygiene in incubators (Cooper 2000b). Infected chicks show non-specific symptoms although coughing and respiratory distress may be seen. Affected chicks usually die rapidly although they may exhibit severe dyspnea prior to death.

Transmission

Spores of the fungus may accumulate in the incubator, hatchery and chick houses. The latter are particularly at risk if humidity levels are high and ventilation is poor, leading to the build-up of ammonia (Terzich & Vanhooser 1993). Other causes include dusty feed that may be inhaled, or mouldy feed which may be a source of infection (Foggin 1992). In older birds, chronic infection may lead to prolonged loss of condition (Foggin 1992).

Diagnosis

In ostriches auscultation over the cranial dorsal aspect of the coelomic cavity is useful during physical examination to listen for lung sounds. Lower respiratory infections, e.g. aspergillosis, may be evident as harsh lung sounds when the bird is ausculated (Tully 1998). Blue coloration of the membranes at the back of the mouth when the beak is open indicates cyanosis. Necropsy reveals *Aspergillus* granulomas in the lungs and/or air sacs (Tully & Shane 1996), which may be colored white, yellow or green, and contain 'cheesy' contents. Bronchoscopy may be used to view and to biopsy plaques within the bronchus and lower airways of sick birds exhibiting symptoms of aspergillosis infection. A highly intensive farming operation may make treatment unaffordable although itraconizole is the drug of choice. Chicks at risk may be treated with ketaconazole at a dose of 200 mg/20 kg bodyweight (bwt) daily for 7 days. Affected incubators or houses should be disinfected or preferably fumigated (Foggin 1992). *Aspergillus* spp. may also cause infection in humans (Tully & Shane 1996). Diagnosis of the disease is made by culture of fresh material and the histological examination of formalin-preserved tissues. Katz *et al.* (1996) developed a method for identifying DNA of *A. fumigatus* from ostriches using the polymerase chain reaction (PCR). The PCR primers based on the sequence of the alkaline protease gene from human isolates of *A. fumigatus* were used. It was found that DNA sequences of *A. fumigatus* isolates from ostriches were similar to that of human isolates. DNA sequences varied significantly among *A. fumigatus* isolates including those from affected ostriches in the same flock. Genetic variation may be used to trace aspergillus infection in ostrich flocks and to determine disease transmission through contact with infected birds.

Zygomycosis

Aetiology

Zygomycosis is a fungal infection of the upper gastrointestinal tract of ratites caused principally by Rhizomucor. Incidence of this disease is sporadic and rare.

Pathology

Birds become infected when fungal spores are ingested and these overwhelm the bird due to a suppressed immune status associated with concurrent bacterial, viral or parasitic infections (Tully & Shane 1996).

Transmission

The accumulation of hay, grass, leaves, fibrous material, sand, gravel and plastic in the proventriculi and ventriculi presumably resulted in nutritional deficiencies and subsequent weakening of immune function. Infection by fungi was facilitated through erosions and hemorrhagic ulcers of varying number and severity in the mucosae of both organs involved.

Diagnosis

Necropsy is needed to diagnose the infection that is usually associated with non-specific clinical signs. Gulbahar *et al.* (2000) reports three four-month-old ostriches with zygomycotic proventriculitis and ventriculus associated with impaction. Clinical signs included anorexia, chronic weight loss, weakness and lethargy, followed by scant feces for 7 days. Hemorrhagic necrosis of mucosal lesions, characteristic of fungal infection, was associated with 5–12 µm wide rarely separated zygomycotic fungal hyphae with non-parallel walls, irregular branching and occasional globoid distension.

PROTOZOA

Aetiology

A number of intestinal protozoa, including *Hexamita*, *Giardia*, *Trichomonas*, *Cryptosporidium*, and *Toxoplasma*

have been isolated from ostrich chicks. Their pathology is unknown, and immunosuppression may be required for disease to develop.

Pathology

The intestinal protozoa *Balantidium struthionis*, *Cryptosporidium* spp., *Histomonas meleagridis*, *Hexamita* spp., *Giardia* spp. and *Trichomonas* spp. cause gastrointestinal problems. Reported is a study on a 14 month old ostrich with small intestinal serosal hemorrhages during post-mortem inspection (Gray *et al*. 1998).

Transmission

Leucocytozoon struthionis and *Plasmodium* spp. are transmitted from flying arthropods to ostriches although they do not cause significant clinical illness (Foggin 1992).

Diagnosis

Histopathological examination of the intestine revealed a chronic lymphoplasmacytic to purulent enteritis with mucosal hyperplasia, muscular hypertrophy and numerous microsporidia. Oocysts of *Cryptosporidium* sp. have been reported in ostriches imported into Canada (8.5%, 14 of 165). The mean (±SD) size of 40 oocysts was 4.6 (±0.53) × 4.0 (±0.42) μm with a shape index (length/width ratio) of 1.15. Cross-transmission experiments showed that the species infecting ostriches failed to infect suckling mice, chickens, turkeys or quail (Gray *et al*. 1998). Yakimoff (1940) has described *Isospora struthionis*, but no indication of *Eimeria* in ostriches has been described (Ernst *et al*. 1970).

Prophylaxis and drug treatment

Following identification of the parasite in fecal and intestinal content examinations, treatment should follow promptly with antiprotozoan drugs. When the organisms are isolated, metronidazole at 10 mg/kg, can be administered.

NEMATODA

Aetiology

Ostriches are infected with many helminths including *Baylisascaris* spp., *Libyostrongylus douglassi*, *Paraonchocera struthionus*, *Struthiofilaria megalocephala*, *Ascaridia orthocerca*, *Deletrocephalus dimidiatus*, *Deletrocephalus casarpintoi*, *Dicheilonema rheae*, *Paradeletrocephalus minor* and *Chandlerella quiscali* (Tully & Shane 1996).

Pathology

Symptoms of infection include ataxia, muscle weakness, recumbence and death due to visceral larval migration into the central nervous system. *Libyostrongylus douglassi* is a proventricular parasite that causes massive mortality in ostrich chicks (Tully & Shane 1996). Pasture rotation is a useful control method (Tully & Shane 1996). This nematode is found in glands and under the lining of the proventriculus. The eggs are passed out in droppings and remain viable in excess of 3 years. The eggs develop into infective, third stage larvae in warm ambient temperatures and in the presence of moisture. Once ingested the larval form develops into an adult worm in 3 weeks. Infective symptoms include lethargy, anorexia, loss of condition, anemia (paleness in the back of the mouth) and constipation. Pathological signs include anemia characterized by pale carcasses and organs, watery blood, small, yellow livers and the presence of worms under the lining of the proventriculus.

Transmission

Baylisascaris spp. is transmitted to ostriches in the USA by skunks and racoons in fecal material in which the eggs remain viable in the soil for years (Tully & Shane 1996).

Diagnosis

Diagnosis is based on finding trichostrongyloid type eggs in the feces. Hoberg *et al*. (1995) detailed the morphological characteristics of L. dentatus recovered from the posterior proventriculus and under the koilon lining of the gizzard of ostriches. The species has a prominent, dorsal, esophageal tooth; male parasites have a dorsal ray and spicules; and female parasites have small eggs (52–62 μm), a sublateral vulva situated at 93% of the body length from the anterior, and a strongly curled, digitate tail with cuticular inflations at the anus.

Prophylaxis and drug treatment

Treatment includes regular administration to chicks every 3 weeks until the age of 4 months, and periodically thereafter one of the following: Levamisole (Tramisol, ICI, Princeton, NJ, USA), 30 mg/kg; Levamisol (Ripercol oral, Janessen, Johannesburg, South Africa), 30 mg/kg; Fenbendazole (Panacur, Hoechst, Johannesburg, South Africa), 15 mg/kg; and Oxfendazole (Systamex, Coopers, Johannesburg, South Africa), 5 mg/kg. Alternating between two treatments is

important to prevent development of drug resistance by the nematode. Fenbendazole (15 mg/kg) administered alone or in combination with Resorantel reduced the total burden of *L. douglassi* by more than 98%. Levamisole (30 mg/kg) was only 28% effective, and in combination with Resorantel its efficacy was 67%. The low efficacy with Levamisole was an indication of anthelmintic resistance in ostriches (Malan *et al.* 1988).

Control measures

Effective biosecurity measures include removal of definitive host populations around ostrich facilities and proper feed storage. Wild birds should not be kept on the same farm as ostriches (Foggin 1992).

CESTODA

Aetiology

The tapeworm *Houttuynia struthionis* is common in Africa and seen only sporadically in the USA. The intermediate host is not known. It mainly affects the small intestine of young ostriches.

Pathology

Ostrich chicks infected with the worm show loss of condition, anemia and the presence of tapeworms in the small intestine.

Diagnosis

The worm is white, segmented and grows up to 60 cm long. Fourie *et al.* (1997) describes a scanning electron microscope examination of *H. struthionis* in which the scolex differed from other subfamilies in the family Davaineidae by lacking scale-like spines covering the base of the rostellum and replaced by small hooks. The worm develops to an immature stage possibly in an insect or mite living on pasture. When the ostrich eats this intermediate host, it develops into an adult worm. Diagnosis is done at post-mortem, although segments of the worm may be passed in the dung and eggs identified by flotation techniques (Foggin 1992).

Prophylaxis and drug treatment

The author recommends one of the following oral treatments every 6 weeks: Niclosamide (Lintex, Bayer, Johannesburg, South Africa), 100 mg/kg; Fenbendazole (Panacur, Hoechst), 25 mg/kg; Oxfendazole (Systamex, Coopers), 5 mg/kg; and Resorantol (Terenol, Hoëchst), 130 mg/kg Gruss *et al.* (1988) showed that

Resorantol (130 mg/kg) was highly effective against *H. struthionis* in ostriches when dosed alone or in combination with Fenbendazole or Levamisole.

TREMATODA

The adult ratite fluke is found outside the nictitating membrane in affected birds. The intermediate host is a freshwater snail and ostriches acquire the parasite through ingestion of freshwater crustaceans or other objects (Foggin 1992). Producers should ensure that their birds do not come into contact with standing water (Tully & Shane 1996). Studies on trematoda infection are lacking due to the rarity of this condition.

ARTHROPODA

Three types of arthropods affect ostrich – lice, ticks, and quill mites. Biting lice, *Struthioliperurus struthionis*, result in skin and feather damage. The mites can be seen on the feather shaft.

Mites and lice

Pathology

Mites and lice may be found infesting ostrich feathers. Infection by the ostrich quillmite (*Pterolichus bicaudatus*) and the ostrich louse (*S. struthionus*) are important for skin and leather quality as irritation from these ectoparasites may cause pruritis and/or excessive preening and feather loss (Foggin 1992). Infestation with these parasites causes stress and predisposes birds to secondary infections and gastrointestinal disorders such as impaction. Mites live in the shaft of feathers causing damage to feather follicles.

Diagnosis

This is made following submission of damaged feathers to the laboratory for examination. The feather mite of ostriches lives in the vein on the underside of the feather and feeds on blood. They can be visualized as small, reddish, dust-like particles.

Treatment

For mites Ivermectin is used (Tully & Shane 1996) dosed by mouth at 0.2 mg/kg at 4 week intervals (1 mL Ivermectin per 50 kg bwt) (Foggin 1992). Lice are treated with 1–5% Malathion dust applied to the bird (Foggin 1992). The author also suggests Alugan wash (Hoechst, Johannesburg, South Africa) or

Flumetherin (Bayer, Johannesburg, South Africa) applied by sponge to the feathers.

Ticks

Aetiology

Ticks commonly infest ostrich skin in Africa and include *Amblyomma* spp., *Haemaphysalis punctata*, *Hyalomma* spp., *Rhipicephalus turanicus* and *Argus* spp. (Craig 1993). Foggin (1992) describes infections of various ixodid ticks in Zimbabwe especially brown ear tick (*Rhipicephalus appendiculatus*), bont-legged tick (*Hyalomma* spp.) and bont tick (*Amblyomma* spp.). Mertins and Schlater (1991) report 10 species of adult ixodid ticks on ostriches imported into the United States from Africa and Europe. These include *Amblyomma gemma*, *A. lepidum*, *A. variegatum*, *Haemaphysalis punctata*, *Hyalomma albiparmatum*, *H. luscitanicum*, *H. marginatum rufipes*, *H. truncatum*, *Hyalomma* sp. and *Rhipicephalus turanicus*.

Pathology

Tick infestations are particularly prevalent in high rainfall and more heavily vegetated areas especially if there is a preponderance of undipped wildlife. Ticks normally attach on the head and neck and immature forms may not be easily seen. Ticks may transmit viral diseases and heavy infestations result in ill thrift, slow growth and low egg production.

Treatment

Control of tick infestations may be achieved by application of pyrethroid pour-on dips to the head and neck by sponge. Approximately 2–4 mL per bird of Flumetherin (Bayer) or Deltamethinin (Coopers), are effective in keeping off ticks for over 2 weeks (Foggin 1992). Craig (1993) describes treatment with 5% carbaryl dust at 14-day intervals for heavy infestations.

Miscellaneous arthropod infestations

Miscellaneous arthropod infestations occur frequently in ostriches resulting in blood loss, irritation and stress, and may facilitate transmission of other parasites (Craig 1993). *Glossina pallidipes* and *G. longipenmis* are blood-sucking arthropods and frequently feed on bushbuck, and also on ostriches, elephant, buffalo and warthog (Sasaki *et al.* 1995). The investigators used NG2G traps (Brightwell *et al.* 1991) and collected 1952 of the former and 1098 of the latter, of which 339 individuals (11.1%) had blood meals. Mertins and

Schlater (1991) report the occurrence of hippoboscid flies (*Struthiobosca struthionis*) in ostriches.

CONCLUSION

Ostriches are susceptible to numerous diseases of bacterial, fungal or parasitic origin. For some diseases afflicting ostriches, new methods of control are poorly documented and an attempt was made to apply methods used in poultry biosecurity to ostriches. Diseases normally result in significant losses to producers and in order to maintain a healthy and profitable enterprise, producers must implement, with assistance from the local veterinary authority, comprehensive, practical and effective methods of health management and preventative medicine. Clearly much research needs to be carried out to gather more information on diseases in ratites, including gaining information of the regulations for meat or eggs after drug administration for a particular ailment.

REFERENCES

Bailey JS, Stern NJ, Cox NA. 2000. Commercial field trial evaluation of mucosal starter culture to reduce *Salmonella* incidence in processed broiler carcasses. *Journal of Food Protection* **63,** 867–870.

Brightwell R, Dransfield RD, Kyorku C. 1991. Development of a low-cost trap and odour baits for *Glossina pallidipes* and *G. longipennis* in Kenya. *Medical and Veterinary Entomology* **5,** 153–164.

Cooper RG. 1999a. Ostrich meat, an important product of the ostrich industry: a southern African perspective. *World's Poultry Science Journal* **55,** 389–420.

Cooper RG. 1999b. *Critical Success Factors for the Zimbabwean Ostrich Industry*. MBA Dissertation, Nottingham Business School, Nottingham Trent University, Nottingham.

Cooper RG. 2000a. Meat from the ostrich. Slaughtering, meat inspection and health risks. *Fleischwirtschaft International* **1,** 36.

Cooper RG. 2000b. Critical factors in ostrich (*Struthio camelus australis*) production: a focus on southern Africa. *World's Poultry Science Journal* **56,** 247–265.

Cooper RG. 2000c. The management of ostrich (*Struthio camelus*) chicks. *World's Poultry Science Journal* **56,** 33–44.

Cooper RG, Horbanczuk JO, Fujihara N. 2004. Viral diseases of the ostrich (*Struthio camelus var. domesticus*). *Animal Science Journal* **75,** 89–95.

Cousins DV, Williams SN, Hope A, Eamens GJ. 2000. DNA fingerprinting of Australian isolates of *Mycobacterium avium* subsp. paratuberculosis using IS900 RFLP. *Australian Veterinary Journal* **78,** 184–190.

Craig T. 1993. Natural parasites of ratites. *Proceedings of Annual Ratite Conference*, 9–10 September, pp. 1–2. College of Veterinary Medicine, Texas A & M University, Texas.

Doneley RJ, Gibson JA, Thorne D, Cousins DV. 1999. Myco-bacterial infection in an ostrich. *Australian Veterinary Journal* **77,** 368–370.

Ebedes H. 1997. Anthrax epizooties in Etosha National Park. *Madoqua* **10,** 99–118.

Ernst JV, Cooper WL, Frydendall MJ. 1970. Eimeria sprehni Yakimoff, 1934, and *E. causeyi* sp. n. (Protozoa: Eimeridae) from the Canadian beaver, *Castor canadensis*. *Journal of Parasitology* **56,** 30–31.

Foggin CM. 1992. Veterinary problems of ostriches. In: Hallam MG (ed.), *The Topaz Introduction to Practical Ostrich Farming.*, pp. 61–96. The Ostrich Producers' Association of Zimbabwe, Harare.

Fourie HJ, Van Amelsfoort AF, Michael LM, Putterill JF. 1997. A scanning electron – microscope examination of the scolex of *Houttuynia struthionis*. *Onderstepoort Journal of Veterinary Research* **64,** 47–50.

Geornaras I, De Jesus AE, Von Holy A. 1998. Bacterial populations associated with the dirty area of a South African poultry abattoir. *Journal of Food Protection* **61,** 700–703.

Gopo JM, Banda GN. 1997. Occurrence of *Salmonella* on meat and products in an ostrich abattoir as determined with a DNA probe. *South African Journal of Animal Science* **27,** 1–6.

Gray ML, Puette M, Latimer KS. 1998. Microsporidosis in a young ostrich (*Struthio camelus*). *Avian Diseases* **42,** 832–836.

Gruss B, Malan FS, Roper NA, Du Plessis C, Ashburner AJ. 1988. The anthelmintic efficacy of resorantel against *Houttuynia struthionis* in ostriches. *Journal of the South African Veterinary Association* **59,** 207–208.

Gulbahar MY, Agaoglu Z, Biyik H, Yuksek N. 2000. Zygomy-cotic proventriculitis and ventriculitis in ostriches (*Struthio camelus*) with impaction. *Australian Veterinary Journal* **78,** 247–249.

Henderson SC, Bounous DL, Lee MD. 1999. Early events in the pathogenesis of avian salmonellosis. *Infection and Immunity* **67,** 3580–3586.

Higgins R, Desilets A, Cantin M, Messier S, Khakhira R, Ismail J, Mulvey MR, Daignault D, Caron H. 1997. Out-break of *Salmonella give* in the province of Quebec. *Canadian Veterinary Journal* **38,** 780–781.

Hoberg EP, Lloyd S, Omar H. 1995. *Libyostrongylus dentatus* n. sp. (Nematoda: Trichostrongylidae) from ostriches in North America, with comments on the genera Libyostrongylus and Paralibyostrongylus. *Journal of Parasitology* **81,** 85–93.

Huchzermeyer FW. 1997a. Animal health risks associated with ostrich products. *Revue Scientifique et Technique* **16,** 111–116.

Huchzermeyer FW. 1997b. Public health risks of ostrich and crocodile meat. *Revue Scientifique et Technique* **16,** 599–604.

Katz ME, Love SC, Gill HS, Cheetham BF. 1996. Develop-ment of a method for the identification, using the poly-merase chain reaction, of *Aspergillus fumigatus* isolated from ostriches. *Australian Veterinary Journal* **74,** 50–54.

Kolb J, Kankondi R, Hubschle OJ. 1993. Isolation of *Chlamy-dia* spp. from ostriches (*Struthio camelus*) (short report). *Deusche Tierernahrung Wochenschrift* **100,** 454.

Ley EC, Morishita TY, Brisker T, Harr BS. 2001. Prevalence of *Salmonella, Campylobacter,* and *Escherichia coli* on ostrich carcasses and the susceptibility of ostrich-origin *E. coli* iso-lates to various antibiotics. *Avian Diseases* **45,** 696–700.

Ley EC, Morishita TY, Harr BS, Mohan R, Brisker T. 2000. Serologic survey of slaughter-age ostriches (*Struthio cam-elus*) for antibodies to selected avian pathogens. *Avian Diseases* **44,** 989–992.

Malan FS, Gruss B, Roper NA, Ashburner AJ, Du Plessis CA. 1988. Resistance of *Libyostrongylus douglassi* in ostriches to levamisole. *Journal of the South African Veterinary Association* **59,** 202–203.

Mertins JW, Schlater JL. 1991. Exotic ectoparasites of ostriches recently imported into the United States. *Journal of Wildlife Diseases* **27,** 180–182.

Provost A, Perreau P. 1978. Combined vaccines in veterinary medicine in the developing countries. *Developments in Biol-ogy Standardisation* **41,** 349–360.

Robertson W. 1908. Case of anthrax in an ostrich. *Journal of Comparative Pathology and Therapeutics* **21,** 261–262.

Sasaki H, Kang'ethe EK, Kaburia HF. 1995. Blood meal sources of *Glossina pallidipes* and *G. longipennis* (Diptera: Glossinidae) in Nguruman, southwest Kenya. *Journal of Medical Entomology* **32,** 390–393.

Shane SM, Camus AC, Strain MG, Thoen CO, Tully TN. 1993. Tuberculosis in commercial emus (*Dromaius novae-hollandiae*). *Avian Diseases* **37,** 1172–1176.

Snoeyenbos GH. 1965. Anthrax. In: Biester HE, Schwarte LH (eds). *Diseases of Poultry*, 5th edn, pp. 432–435. Iowa State University Press, Ames, Iowa.

Terzich M, Vanhooser S. 1993. Post-mortem findings of ostriches submitted to the Oklahoma Animal Disease Diagnostic Laboratory. *Avian Diseases* **37,** 1136–1141.

Theiler A. 1912. Anthrax in the ostrich. *Journal of the Union of South Africa* **4,** 370–379.

Tully TN. 1998. Health examinations and clinical diagnostic procedures of ratites. *Veterinary Clinics of North America – Food Animal Practice* **14,** 401–402.

Tully TN, Shane SM. 1996. Husbandry practices as related to infections and parasitic diseases of farmed ratites. *Revue Scientifique et Technique* **15,** 73–89.

Verwoerd DJ. 2000. Ostrich diseases. *Revue Scientifique et Technique* **19,** 638–661.

Yakimoff WL. 1940. Isospora struthionis n. sp., coccidie de l'autrucheafricaine. *Annales de Societe Belge de Medecine Tropical, Brussels* **20,** 137–138. (in French with English abstract)