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INTRODUCTION

The infraclass Palaeognathae includes ratites (ostrich, emu, rhea, cassowary, kiwi), and tinamous, a group of related but flighted birds (see Supplemental Table e1 for a list of the members of the Infraclass [Clade] Palaeognathae). Commercial ratite production for meat and leather (ostrich, emu, rhea), feathers (ostrich) and oil (emu) occurs worldwide. The management and diseases of the large ratites are similar. Information on the kiwi and tinamou derives primarily from in situ conservation programs and zoological collections (see the Supplemental Materials for a list of general resources related to ratite husbandry and health).

UNIQUE FEATURES

The basic principles of avian hematology and biochemistry are applicable to ratites. Reference intervals have been published and summarized for commercially raised species, particularly for ostriches. Several papers provide detailed descriptions of the composition of naturally voided ostrich urine and a few reports provide preliminary information on clinical pathology of the cassowary (see the Supplemental Materials for notable clinical pathology information and reference material).

Large and small ratites have several unique characteristic anatomic features. Some of the most notable are described later and shown in Figs. 26.1–26.4. Additional details and comparative anatomy across ratites are outlined in the Supplemental Materials Table e2.

Relative to other avian species and small ratites, large ratites have several anatomic modifications related to flightlessness: a flat keel with minimal pectoral musculature, poorly developed wings with modifications of the thoracic girdle, proportionately over developed legs, and elongated necks (Fowler, 1991). The ilia form an inverted bony shield dorsal to the synsacrum. Feathers lack barbules and hence have a fluffy appearance. There is no uropygial gland (Bezuidenhout, 1999).

All male ratites have a phallus in the proctodeum and the females of most species have a smaller analogous structure referred to as either a phallus or a clitoris. Ostriches must stand to urinate, thus birds recumbent for some time may have a coprodeum full of urine.

Due to the common occurrences of yolk sac retention in ostriches, an understanding of normal is critical. A microscopic description of the normal and abnormal yolk sac of the ostrich has been published by Dzoma and Dorrestein (2001). A predictive curve for yolk sac size was developed, with the amount of yolk predicted to be 33% of chick weight on day 1 after hatching, as compared to 4% at 14 days post-hatch. The mucosa of the yolk sac is initially composed of long villi lined by large vacuolated epithelial cells. These villi disappear by day 14, leaving a layer of single or stratified vacuolated epithelial cells.

In ostrich, emu, and rhea chicks, an important feature of the developing long bones is the presence of long medial and lateral cartilaginous cones composed of a solid sheet of chondrocytes with intervening blood vessels (Reece and Butler, 1984; Shivaprasad 1995). These cones extend from the growth plates and are surrounded by a thin osseous rim at the time of hatching. They gradually become separated from the growth plate, surviving as a central cartilaginous core until becoming fully remodelled into trabecular bone 6–8 weeks after hatching. In ostriches, the morphology and amount of cartilage varies among the long bones and among birds (Shivaprasad, 1995). Ossification at the growth plates occurs in the normal manner.

Kiwis have vestigial wings with a curved claw on the major digit. Feathers are long and hair-like at the
tips, without interlinking barbules. The beak is elongated and ventrally curved with slit-like nostrils at the tip; it is longer in the female than the male. Identifying earthworms and invertebrates in the soil is assisted by a sensory region at the tip of the beak that contains large numbers of Herbst and Grandry-like mechanoreceptors (Bang, 1971).

Female kiwis have two fully functional ovaries that can ovulate alternately, but only the left oviduct is functional (Kinsky, 1971). The follicle of the kiwi can attain 80 mm in diameter prior to ovulation; egg size would be more appropriate for a bird 6 times greater in weight (Fig. 26.3). The yolk comprises 61% of initial egg weight, with 48% of yolk mass still present at hatching and that can provide nutrition to the chick for up to approximately 14–17 days (Prinzinger and Dietz, 2002).

The tinamous are a large group of flighted, partridge-like birds. Although their wings are small, they have well-developed pectoral musculature and a highly pneumatized skeleton. Tinamous have well-developed ceca (Fig. 26.4), which vary considerably in appearance among species (McLelland, 1989). They are sacculated in the red-winged tinamou, and in the elegant crested tinamou are very...
unusual with numerous prominent external diverticula and a mucosa that resembles the reticulum of a ruminant.

NON-INFECTIOUS DISEASES

Nutritional

Sudden death due to a dissecting aortic aneurysm in ostriches and in a single emu has been suggested as being related to copper deficiency (Ferreras et al., 2001; Vanhooser et al., 1994). Histologic aortic lesions include fragmentation and disruption of the elastic laminae, cystic extracellular spaces, and an accumulation of what have been interpreted to be strongly sulfated acid mucopolysaccharides based on staining with Alcian blue and periodic acid Schiff (PAS). Copper deficiency is suspected based both on the character of the aortic lesions and the findings of low liver copper levels in a number of affected birds. A copper-dependent enzyme, lysyl oxidase, is required for the cross-linking of collagen and elastin fibres that provides strength to the vascular wall. Liver copper levels in affected birds are frequently below 3.5 ppm, in comparison to above 5 ppm in unaffected birds.

Developmental angular limb deformities are a significant cause of mortality in ratite chicks (Bezuidenhout and Burger, 1993; Huchzermeier, 1998). These abnormalities are undoubtedly multifactorial, with nutritional and management factors intricately intertwined in the pathogenesis. Imbalances of Vitamin D₃, calcium, phosphorus, manganese, zinc, copper, and selenium; riboflavin deficiency; and excess protein have all been implicated in disease pathogenesis. The most commonly described syndromes include unilateral or bilateral bowing or twisting of the leg bones, particularly of the tibiotarsus and/or tarsometatarsus (Fig. 26.5). Associated sequelae can include dislocation of the gastrocnemius tendon over the hock joint and lateral rotation of the major toe of the ostrich.

Rickets in ratite chicks has most often been associated with hypophosphatemia. The pathologic features have been described in affected rheas (rubber rhea syndrome) and ostriches (Gröne et al., 1995; Morrow et al., 1997). Characteristic lesions are pliability of long bones, angular deformity of the distal tibiotarsus and the tarsometatarsus. The normal (right) leg is on the left side of the image for comparison.
FIGURE 26.6  Phosphorus-deficiency rickets in young common rhea. Characteristic features include (A) Tibiotarsal curvature and thickening at the proximal growth plate, (B) enlargement of the metaphyseal plates, elongation of the zone of hypertrophic cartilage, and pathologic fractures in the underlying metaphyseal bone (arrow), and (C) pathologic fractures characterized by irregular, jagged edged clefts in the bone surrounded by irregular rests of hypertrophic cartilage. (Part B: Photo Courtesy of H.L. Shivaprasad, University of California Davis (UC Davis), California Animal Health & Food Safety Laboratory [CAHFS])
Atherosclerosis can develop in emu as a sequela to feeding a ration high in eggs (high cholesterol content) and fat for an extended period of time (Tomimura et al., 1970). Histologic lesions include partial arterial occlusion by large confluent atherosclerotic plaques containing cholesterol clefts, lipid, and lipid-laden macrophages, fatty degeneration and calcium deposition in the media, fragmentation of the internal elastic membrane, and muscular elastic thickening. In a described case, the abdominal aorta and coronary arteries were the most severely affected.

Marked pansteatitis associated with low liver Vitamin E levels has been seen in adult kiwi (Boardman, 1998).

Toxic

Monensin toxicity in ostriches has occurred due to errors in feed formulation. It presents clinically as anorexia, ataxia, loss of control of the neck, recumbency, and dyspnea prior to death. Expected elevations in creatine kinase, aspartate aminotransferase and lactate dehydrogenase, and classic histopathologic lesions of acute to subacute skeletal myodegeneration are seen (Baird et al., 2000).

Botulism due to Clostridium botulinum and associated with toxin types C and D has been described in ostriches and, presumptively, in a cassowary (Romer, 1997). Diagnosis in these cases is related to clinical history and identifying toxins via mouse bioassay. Affected ostriches may be acutely ataxic, paretic, or completely paralyzed (Huchzermeyer, 1998).

The larger ratites are indiscriminate eaters, and ingestion of metallic foreign bodies has resulted in absorption of toxic levels of iron, lead, copper, and zinc. Pancreatic exocrine atrophy and fibrosis accompanied by the presence of regenerative ducts has been described ostriches due to zinc toxicity (Carreira et al., 2011).

Toxicity associated with ingestion of certain plants and plant toxins within pasture is reported for: parsley (Petroselinum sativa, ostrich, photosensitization); avocado (Persea americana; ostrich; myocardial degeneration); oak leaves (Quercus agrifolia; double wattled cassowary; enteritis and nephrosis); wilted grass sprouts (ostrich; prussic acid poisoning); yew (Taxus baccata; emu; gastroenteritis and pulmonary edema); and a number of toxic southern African plant species (including ragwort [Senecio scleratus] and lantana [Lantana camara]; ostrich) (Cooper, 2007a; Huchzermeyer, 1998).

Mortality occurred in a flock of young emu chicks that ingested large numbers of blister beetles (Pyrota insulata) containing cantharidin, a vesicant. Resulting lesions included esophageal congestion and ulceration, submucosal vascular thrombosis and necrosis, and sloughing of the lining of the ventriculus (Barr et al., 1998).

Levamisole toxicity in kiwi resulted in acute bronchopneumonia with severe pulmonary edema and death in five of six affected birds, as well as hepatocellular degeneration, and vascular thrombosis and multifocal hepatic necrosis, each in one bird (Gartrell et al., 2005).

A list of several additional toxins described as causes of clinical disease and death in the larger ratite species is included in the Supplemental Materials. There are frequently minimal details on associated pathologic findings in these reports.

Congenital/Genetic

A form of mucopolysaccharidosis type IIIB, an autosomal recessive, inherited lysosomal storage disease caused by a deficiency of lysosomal alpha-N-acetylglucosaminidase (NAGLU) and subsequent intracellular accumulation of heparan sulfate, has been described in emus (Palmieri et al., 2015). Juvenile birds develop progressive neurologic signs; acute death due to abdominal hemorrhage is also described. Characteristic histologic lesions include widespread neuronal swelling and fine, intracellular vacuolation in the brain, spinal cord, retina, and autonomic ganglia (Fig. 26.7A). Material in vacuoles stains variably positive with PAS and strongly positive with Luxol fast blue. Foamy macrophages may also be present in a variety of tissues including liver, spleen, intestine, and the tunica media of the aorta. On transmission electron microscopy (TEM), membrane bound, electron-dense, lamellated cytoplasmic material (consistent with “zebra bodies”) is seen in neurons and ganglial cells (Fig. 26.7B). Storage material in retinal cells may differ and consist of a central granular, highly electron-dense core surrounded by membranous whorls, while in macrophages it may appear as well-circumscribed, electron-dense, homogenous circular bodies surrounded by stacks of rough endoplasmic reticulum.

A mechanobullous skin condition with low prevalence occurred in ostrich chicks on a large farm in Israel over a 2-year period (Perelman et al., 1995). Lesions, present only in skin, consisted of dermolytic blistering, skin peeling, and feather loss. Disease resulted in death or euthanasia of affected chicks by 14 weeks of age. Microscopic and TEM examination of skin showed epidermolysis bullosa-like lesions, including subepidermal bullae, splitting of the sublamina densa, dermal edema, and with time, inflammation and crusting. Lesions were considered likely to be inherited, but the parentage of the affected birds could not be traced.

Trauma

Trauma, misadventure, and capture myopathy are leading causes of death in adult age group ratites. In the wild, the most commonly recognized causes of mortality for cassowaries and kiwis are trauma, and trauma and predation, respectively. Tinamous are prone to injury and trauma when startled or frightened and must be handled carefully to avoid fractures.
Inflammatory Non-infectious

Amyloidosis has been reported in rhea and in the liver and/or spleen in young ostriches with mycotic airsacculitis and pneumonia (Akkoç et al., 2009; Cowan, 1968). Histologically, eosinophilic homogenous material positive with Congo red staining expands sinusoids and is associated with hepatocyte degeneration and atrophy. In the spleen, the material is within the tunica media of and surrounds splenic blood vessels. The presence of amyloid A type fibrils has been confirmed using immunohistochemistry (Akkoç et al., 2009).

Miscellaneous

Management-related factors are a major cause of mortality in captive ratite systems. In the hands of inexperienced ratite farmers, hatching, and neonatal chick survival rates can be abysmal. Improper egg collection and storage, incubation, and hatching parameters result in embryonic mortality and failure to hatch, weak and edematous hatched chicks, and poor chick viability. Acute generalized skeletal myopathy involving the complexus (pipping), hind limb, and costopulmonarís muscles has been described in newly hatched chicks and may be a result of exertion or physiologic difficulty at hatching (Philbey et al., 1991).

“Fading syndrome” and “gastric stasis” are major causes of stunted growth, wasting, and mortality in ostrich chicks up to 6 months of age; up to 70% of can be chicks affected to varying degrees. The cause is not fully understood but environmental parameters, maladaptation, and increased susceptibility to infectious agents are likely important factors (Button et al., 1996; Huchzermeier, 1998; Ocal et al., 2006). Necropsy lesions are non-specific but include emaciation and proventricular and ventricular changes including either lack of contents or distension without impaction, and mucosal hyperplasia and degeneration.

Ratites are indiscriminate eaters and gastrointestinal impaction and gastrointestinal foreign bodies are; therefore, important causes of anorexia, wasting, and death in birds of all ages (Reissig and Robles, 2001). Impaction with straw, shavings, coarse grass, and virtually any other ingestible substrate have been reported (Fig. 26.8A, B).

Pneumoniosis is common in captive kiwi and lesions can be marked. The condition is thought to be associated with dry, dusty substrates and enhanced by the birds’ sniffing behavior during feeding and substrate investigation (Smith et al., 1973).

Miscellaneous conditions identified in tinamous held in zoos include a myocardial infarct, aortic and carotid atherosclerosis, vegetative valvar endocarditis associated with chronic sinus infection, oviductal egg impaction, and nephritis (Griner, 1983). The pathologies in birds raised under more intensive conditions are somewhat different with uric acid nephrosis, amyloidosis, gastrointestinal foreign bodies, and the presence of the renal trematode Paratamaisia confusa and of Capillaria penidoi in the upper digestive tract as the most significant findings in one study of red-winged tinamous (Momo, 2007).

Neoplastic

Neoplastic diseases have rarely been reported in Struthioniformes, with multicentric lymphoid neoplasia in ostriches most commonly described. Single cases of lymphoid leukemia and a monoclonal gammopathy also exist (additional descriptive details for lymphoid neoplasia in ratites can be found in the Supplemental Materials Table e3). There are no reports of immunohistochemical or other typing of neoplastic lymphocytes. Additionally, single cases of intrathoracic hemangiosarcoma (Headley, 2005)
and capillary-type pulmonary hemangioma (Shathele et al., 2009) have been reported in young ostriches. Other neoplastic conditions undoubtedly exist but have not been formally described.

**INFECTION DISEASES**

Infectious diseases, particularly as caused by “garden-variety” bacterial and fungal agents, are significant causes of embryonic death, poor hatchability, and deaths in neonatal and growing chicks. Deficiencies in hygiene during incubation and hatching, suboptimal incubation and hatching parameters leading to weak chicks and failure of the umbilicus to close, retained yolk sacs, and immunosuppression resulting from environmental stress predispose to infections. This is exemplified by a summation of pathologic findings in 59 tinamous of eight species in a North American zoo: bacterial infections, most commonly omphalitis with *Escherichia coli* and *Pseudomonas* spp., pneumonia and pulmonary aspergillosis, and esophageal candidiasis were among the noted conditions (Griner, 1983).

Viruses reported as frequent or significant causes of infection and/or disease are described later; brief descriptions of additional viral agents affecting ratites are listed in the Supplemental Materials Table e4.

**DNA Viruses**

In 1992, an adenovirus outbreak caused 90%—100% mortality in ostrich chicks less than 2 months of age in Oklahoma, United States (Raines and Meridian, 1993). Egg transmission was highly suspected. The most unique clinical symptom was foul smelling, gray chalky feces. Gross necropsy findings were non-specific, including pulmonary congestion, pale foci in the liver, a pale spleen and airsacculitis. On microscopic examination, the most consistent lesions in chicks were splenic and bursal lymphoid depletion, hepatic necrosis, necrotic enteritis, and pulmonary congestion; adult hens laid chalky or wrinkled eggs (Fig. 26.9) and one had evidence of hepatitis. An adenovirus was isolated from dead chicks, the yolk sac of a dead in-shell chick, and the trachea of healthy adult birds. Inclusion bodies consistent with adenovirus were not seen and the virus was not further characterized.

In the United Kingdom, six adenoviruses similar to fowl adenovirus serotype 8 have been isolated from ostriches. The sources were the tracheas of an adult hen that laid eggs with chalky ridged shells and her mate, and the intestines of chicks with diarrhea (Gough et al., 1997). In Italy, an adenovirus isolated from a 4-month-old ostrich chick was associated with an enlarged, nodular pancreas and hemorrhagic enteritis. Inoculation studies in young guinea fowl chicks produced severe pancreatic lesions, including tissue swelling and hardening, loss of acini and inflammation,
and death. Intranuclear viral inclusions were present in surviving and regenerating cells. Restriction endonuclease analysis showed the virus to be similar to fowl adenovirus serotype 1 (Capua et al., 1994).

An avian pox virus was identified histologically and by sequencing as the cause of transient nodular cutaneous lesions in the skin of the legs of two immature North Island brown kiwi and near the beak in one of these (Ha et al., 2013). The histopathologic lesions were classical, with epidermal hyperplasia, epithelial swelling, and eosinophilic intracytoplasmic viral inclusions (Bollinger bodies).

RNA Viruses

Several of the RNA viruses that affect ratites cause OIE listed reportable diseases. These are avian influenza virus (AIV), Newcastle disease virus (Avian paramyxovirus 1), and eastern and western equine encephalitis viruses.

Avian influenza virus infection has been reported in ostriches, emus, rheas, and a cassowary. Infection with a variety of viral strains including those of both high (H5 and H7; HPAI) and low pathogenicity occur frequently in farmed ostriches in South Africa (Abolnik et al., 2016). Outbreaks are thought to result from exposure to wild birds. Clinical signs and pathology associated with AIV in ostriches in natural outbreaks and with experimental infection are variable. Mortality can be up to 100% and is dependent on age, virus strain, and secondary infections (Allwright, 1996; Capua et al., 2000; Cooper et al., 2007). Chicks and juvenile birds are more susceptible to infection and develop more significant clinical disease than adults, who frequently appear unaffected. Clinical signs include anorexia, depression, and poor growth rates as well as green discoloration of the urine (biliverdinuria), swelling of the throat and neck, ocular discharge and sneezing and evidence of respiratory difficulty. Gross necropsy findings are nonspecific and can include serous atrophy of fat and muscle atrophy; luminal bile and bile staining of the upper gastrointestinal tract; hepatic bile stasis; congestion and motting of the liver, spleen, kidneys, trachea and lungs; epicardial hemorrhages; and intestinal congestion to severe hemorrhagic enteritis. Histologic lesions also vary and include multifocal to diffuse necrosis of liver, spleen, and kidneys with a heterophilic response; pancreatic hemorrhage and necrosis with mononuclear cell inflammation; fibrinoid arteriolar necrosis and inflammation, particularly in spleen and brain; interstitial pneumonia; mild to marked air sacculitis; multifocal malacia and neuronophagia in the brain; and necrosis and hemorrhage in the intestine, particularly in the duodenum. Both gross and histologic lesions are affected by the presence of secondary infections, which have been prevalent in several reports. Infection with low pathogenic avian influenza viruses results in primarily a mild respiratory disease, with a reduction in egg production (Cooper et al., 2007).

In emus and rheas, respiratory disease is the predominant clinical presentation with HPAI H5N2 and H7N1 subtypes in the United States. Lesions are present in both the upper and lower respiratory tracts (Panigrahy et al., 1995; Woolcock et al., 2000). In China, an outbreak of low pathogenicity H9N2 influenza in emus was associated with 60% morbidity and 12.5% mortality (Kang et al., 2006). Gross lesions included hyperemia and hemorrhage in the mucous membranes, edema and congestion of the lungs and cloaca, epicardial hemorrhage, hepatic and renal swelling, and widespread gastrointestinal hemorrhage. Histologic lesions included desquamation of the bronchial mucous membrane; myocardial necrosis; and local liquefactive necrosis, satellitosis and neuronophagia, perivascular cuffing, and hemorrhage in the brain.

Standard methods of diagnosing AIV infection in ratites include PCR, virus isolation and typing, testing for pathogenicity in chickens, and standard serologic surveys. Tracheal swabs may be positive for virus earlier in disease than cloacal swabs (Allwright, 1996).

Newcastle disease virus (Avian paramyxovirus 1) has been identified in ostriches in zoos and commercial operations in several countries. Like avian influenza viruses, this is an OIE listed reportable disease. Virus has been isolated from tissues of birds with and without a history of clinical disease, and there are no pathognomonic gross or microscopic lesions. Chicks and juveniles appear most susceptible to disease development, with sudden death as the most common presentation. In adult birds, the predominant clinical signs and lesions include edema of the head; mucoid sinusitis, tracheitis and air sacculitis; splenomegaly; epicardial petechial hemorrhages; and neurologic abnormalities. Histologic lesions include neuronal chromatolysis, endothelial cell hyperplasia, perivascular round cell cuffing, and multifocal gliosis, generally limited to the brainstem, and less frequently, splenic vascular necrosis with fibrin exudation or lymphoplasmacytic pancreatitis (Huchzermeyer and Gerdes, 1993). Cerebrospinal fluid has been suggested as a source of material for direct EM, hemagglutination inhibition, and virus isolation, although this last can be difficult (Allwright, 1996).

Western equine encephalitis (WEE) is also an OIE listed reportable disease. Infection in emus is predominantly a neurologic disease with a reported morbidity of 10%–50% and mortality of approximately 10% (Ayers et al., 1994; Randolph et al., 1994). Birds as young as 3 months of age can be affected. Clinical signs include depression, anorexia, and evidence of weakness and/or ataxia including muscle tremors, recumbency, and unnatural positioning of the head on the back (opisthotonus). Watery diarrhea, green urates, and pericardial effusion may also be present (Ayers et al., 1996). In China, an outbreak of low pathogenicity H9N2 influenza in emus was associated with 60% morbidity and 12.5% mortality (Kang et al., 2006). Gross lesions included hyperemia and hemorrhage in the mucous membranes, edema and congestion of the lungs and cloaca, epicardial hemorrhage, hepatic and renal swelling, and widespread gastrointestinal hemorrhage. Histologic lesions included desquamation of the bronchial mucous membrane; myocardial necrosis; and local liquefactive necrosis, satellitosis and neuronophagia, perivascular cuffing, and hemorrhage in the brain.

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Microscopic lesions in the brain include mild to moderate infiltrates of lymphocytes, plasma cells, heterophils, and/or histiocytic cells perivascularly and in the meninges. Neuronal satellitosis, increased numbers of oligodendroglia, and axonal degeneration and empty axon sheaths can also occur (Randolph et al., 1994). Extensive vasculitis with necrosis and inflammation in a wide range of tissues is common. Diagnosis can be made by virus isolation and PCR.

A single outbreak of WEE in rheas in the United States has been described. Diagnosis was based on seroconversion and response to vaccination (Randolph, 1995). Clinical signs were generally non-specific and included anorexia, drowsiness, stupor, ataxia, hyperpnea, and loose stool for a period of 48–72 h prior to death. Twenty two of 90 birds died; at least 30 showed mild illness and recovered. Gross findings were restricted to mild hepato- and splenomegaly. On microscopic examination, there was mild multifocal subacute pericholangitis, diffuse hepatic sinusoidal leukocytosis and moderate diffuse splenic histiocytosis and plasmacytosis.

Eastern equine encephalitis (EEE) is primarily a disease of emus. It can affect all ages of birds and has a mortality rate up to 87% in juveniles and adults. Death is frequently acute, but reported clinical signs include marked depression and bloody vomitus and diarrhea. The predominant gross necropsy findings include large amounts of blood in the small and large intestines and systemic serosal petechiation to ecchymoses (Fig. 26.10). Histologically, consistent lesions include fibrinoid degeneration of splenic sheathed arterioles with necrosis of endothelial macrophages and diffuse lymphoid necrosis; hepatic congestion and hemorrhage with widespread hepatocellular necrosis; and changes in the intestinal lamina propria including the presence of large amounts of necrotic cellular debris, hemorrhage, a mild heterophilic infiltrate, and necrosis of lymphoid follicles. It is notable that lesions are not present in the central nervous system (Brown et al., 1993; Veazey et al., 1994). Emus experimentally infected as part of a vaccine trial developed severe clinical signs and shed virus in feces, oral secretions and regurgitated material; infection spread horizontally in the absence of mosquito vectors (Tengelsen et al., 2001).

Naturally occurring EEE infection has also been described in six captive cassowaries in the United States (Guthrie et al., 2016). Both juvenile and adult birds were affected. Clinical signs included sudden death, lethargy, weakness, and ataxia and convulsions. Gross necropsy lesions included coelomitis and diarrhea. The most consistent histologic lesion was vasculitis in the liver and spleen. Mild lymphocytic encephalitis and hepatitis, splenitis, pneumonia, and nephritis were also seen. The presence of a number of intercurrent diseases had an impact on the range of pathologic changes. Notably, the affected cassowaries did not develop hemorrhagic enteritis.

The diagnosis of EEE can be confirmed by virus isolation from tissues including liver, spleen, blood and brain; TEM; and PCR. Surviving birds develop both hemagglutinating and neutralizing antibodies; however, antibodies can also result from maternal transfer, inapparent infection, and vaccination (Day and Stark, 1996).

A prolonged outbreak of bornavirus-associated neurologic disease occurred in ostrich chicks on a series of farms in Israel between 1988 and approximately 1993. Clinical signs included a brief period of incoordination followed by spastic paresis and eventually death due to secondary complications. Mortality varied but was up to 20%. There were no consistent gross lesions. Histologic lesions were restricted to the lumbosacral spinal cord and included neuronal degeneration and neuronophagia, infiltrating glial cells (satellitosis), and the formation of glial nodules. The disease was experimentally transmitted to ostrich chicks using the brains of infected chicks and on microscopic examination notable perivascular cuffing developed. Extensive laboratory investigation into the cause of the syndrome provided convincing evidence of infection by Borna disease virus (Malkinson et al., 1995).

Aquatic bird bornavirus-1 was identified as the cause of prolonged, subtle gastrointestinal, and neurologic clinical signs in an emu in a zoological park. Histopathologic lesions included marked lymphoplasmacytic encephalomyelitis with prominent perivascular cuffing, and multifocal neuritis associated with the gastrointestinal tract. The disease was assumed to have been transmitted from wild Canada geese (Nielsen et al., 2017).
Bacteria

Bacterial infection is a common cause of embryonic death and of disease and mortality in young hatched ratite chicks. Yolk sac infection, generally due to ascending infection through an open umbilicus, is common in incubated chicks and can lead to bacteremia. Bacteria that cause localized (particularly respiratory and conjunctival), enteric, and systemic infection in ratites are no different from those that affect other species of birds and include a variety of Gram positive and Gram negative environmental and opportunistic organisms. Select bacterial diseases that are frequently reported in ratites or are of particular note are described in the Supplemental Materials Table e5. These include diseases due to Bacillus anthracis, Clostridium perfringens, C. difficile, C. chauvoei, Erysipelothrix rhusiopathiae, Brachyspira hyodysenteriae (also see Fig. 26.11), Bordetella avium, B. bronchiseptica, Campylobacter jejuni, Chlamydia psittaci, Lawsonia intracellularis, Hemophilus spp., Salmonella spp., and mycobacterial (also see Fig. 26.12) and mycoplasmal infections.

Fungi

Infection of the upper and lower respiratory systems with Aspergillus spp., particularly A. fumigatus and A. niger, is a common cause of disease in captive ostriches, emus, and rhea (Huchzermeier, 1998). Outbreaks are frequent in young chicks (e.g., brooder pneumonia). Clinical signs and gross and pathologic lesions are similar to those in any avian species and include pale gray to yellow nodules and plaques in the lungs and air sacs (Fig. 26.13). Histopathologic lesions are typical for avian aspergillosis, with central areas of necrosis that contain parallel-walled, dichotomously branching, fungal hyphae and conidia (at air interfaces) and are surrounded by heterophilic and granulomatous inflammation with multinucleate giant cells and (in chronic lesions) an outer layer of fibroblasts or granulation tissue. Fungal elements are often easily identified with routine hematoxylin and eosin staining; silver or PAS stains highlight the organisms. Aspergillus spp. have also been reported as the cause of dermatitis in ostriches. Lesions can be localized or extensive (Kuttin and Perelman, 1996). Regardless of site, fungal culture is confirmatory and PCR can be used for additional characterization and differentiation of species. Predisposing factors to infection include contamination of eggs and hatcheries, overwhelming exposure to fungal spores through contaminated feed and bedding, and immunosuppression; high humidity and/or poor hygiene were associated husbandry conditions in cutaneous aspergillosis.
Infection of the upper digestive tract; the oral cavity, pharynx, and esophagus, by opportunistic yeasts is reported frequently in ostrich chicks and is primarily associated with *Candida* spp. Proliferative and necrotizing lesions containing intraleisonal 3–5 µm diameter yeast and interspersed nonseptate pseudohyphae or hyphae are characteristic. Organisms can be highlighted in histologic section with either silver or PAS staining. Overuse of antibiotics is a suspected predisposing factor (Huchzermeyer, 1998) but infections can occur with no associated antibiotic administration.

Lesions of *proventricular* and *ventricular zygomycosis* in ostrich chicks include mucosal erosion and ulceration, suberosal edema, hemorrhage, and intraleisonal fungus. A thick layer of mucus covering the lining of the ventriculus, a result of mucosal mucous cell hyperplasia, appears to be a characteristic finding (Jeffrey et al., 1994; Kuttin and Perelman, 1996). Fungal hyphae are broad (6–20 µm diameter), irregular in appearance, paucisepitate, and branch at 45–90 degrees. As with other fungal organisms, the fungi can be highlighted with silver or PAS staining. Infection is considered opportunistic or secondary to lesions from proventricular and/or ventricular impaction. *Macrorhabdus ornithogaster* is an ascomycete yeast that is found in ostriches and common rhea and is associated with a clinical syndrome of failure to thrive and progressive emaciation despite an initially good appetite (Huchzermeyer, 1998; Martins et al., 2006). Mortality can be up to 100%. Gross necropsy findings include emaciation, a dilated but not impacted proventriculus, reduced digesta and ulceration of proventricular and ventricular mucosa. Organisms can be seen on fecal smears or proventricular or ventricular scrapings where they resemble oversized, elongate, rod-shaped bacteria. Histologically, they are present in the koilin where they appear as rigid, parallel, stacked rectangular structures. The organisms are Gram variable. Giemsa staining highlights the nuclei on cytology, and PAS and silver staining highlight the organisms in histologic sections. Associated inflammation is generally minimal.

**Fungal dermatitis** has been infrequently reported in rafites. A dermatophyte with an endothrix pattern in feather shafts, possibly a *Trichophyton* sp., resulted in feather loss and proliferative crusting lesions on the head and upper neck in several ostrich chicks (Onderka and Doornenbal, 1992). *Microsporum gypseum* was described as a cause of rows of small cutaneous lesions that resulted in downgrading of skins from commercially raised ostriches in South Africa (Huchzermeyer, 1998).

Four cases of infection with *Cryptococcus bacillisporus* (previously *C. neoformans var. gatti*) have been reported in North Island brown kiwi (Hill et al., 1995; Malik et al., 2003). Disease was limited to the respiratory tract in two cases and disseminated in two. Multifocal to diffuse necrosis, hemorrhage, and histiocytic inflammation with intraleisonal fungus was present in affected organs, which included the heart, liver, kidney, oviduct, pancreas, proventriculus, and intestinal serosa. Organisms were spherical, 8 µm in diameter, and surrounded by a 1.6 µm capsule that could be highlighted by staining with PAS or Best’s mucicarmine. Narrow-necked budding, a characteristic feature of *Cryptococcus* spp., was present. The low body temperature of the kiwi, the use of eucalyptus mulch, and the species’ habit of sniffing while probing, were all considered as predisposing factors.

**Metazoa**

A small number of parasitic diseases are or have been associated with substantial morbidity and or mortality in rafites. As in any other species, intensive rearing of birds and environmental contamination increase the prevalence and severity of disease. Raffites will ingest the feces of other species, thus the presence of parasite eggs in their feces can reflect pseudoparasitism rather than true infection. Supplemental Table e6 contains a list of the major metazoan endoparasites that affect larger raffles along with their host(s), geographic distribution, and associated disease. Parasites of particular significance include: *Libyostrongylus douglassii*, *Deletrocephalus dimidiatus*, *Codiostomum struthionis*, and *Houttuynia struthionis*. The endoparasites of the kiwi were tabulated by McKenna (2010); additional details are provided later. The ingestion of earthworms, which serve as intermediate and paratenic hosts for many nematode parasites, may predispose to the range of helminth infections described in kiwi (Clark, 1981). A variety of helminth parasites have also been recorded in both captive and free-ranging tinnamous; thickened oral and esophageal mucosa and fetid diarrhea being the most common associated pathologic changes/signs (Marques et al. 2012; Silveira et al., 2001).

Migration of aberrant parasites is a concern in all raffite species (Fig. 26.14A, B). *Visceral larval migrans* is frequently described in kiwi, most often in the North Island brown kiwi and most often as an incidental finding with granulomas containing fragments of or intact nematode larvae particularly in lung and liver (van Zyl, 2014). Neural larval migrans has also been reported in the North Island brown kiwi, and is likely of greater clinical significance. The species of nematode(s) causing these lesions is unknown, but does not appear to be *T. canis* or *T. catti*.

**Cutaneous larval migrans** has been described in brown kiwi chicks held on a côteche island as part of a captive propagation program (Gartrell et al., 2015). Infection was associated with feather loss, and a “scurfy” dermatitis was present in the abdominal area and along the vent margin. Histologically, there was mild to moderate epidermal hyperplasia and hyperkeratosis with perivascular and
nodular lymphoid infiltrates; lower numbers of heterophils and eosinophils; and in some cases the presence of intraepithelial pustules, granulomas, and necrotic debris. Intraluminal nematode larvae were identified, and were closely related to *Trichostrongylus axei* using DNA sequencing. It was speculated that the parasites might be of sea bird or marine mammal origin.

Intraventricular *Cyrnea apterycis* (a spirurid nematode) and colonic *Heterakis apterycis* appear to be common incidental findings in kiwi (*Clark and McKenzie, 2009*). Adult *Toxocara cati* have been identified in the small intestine of a North Island brown kiwi (this is the only report of adult worms of this species in an avian host) (*Clark and McKenzie, 2009*).

**Protozoa**

The majority of information pertaining to enteric protozoa in ratites derives from the results of fecal examinations. In the absence of correlation to clinical signs or histologic lesions, it is difficult to associate these findings with disease. As well, the use of molecular techniques has shown that the morphologic identification of this group of parasites is often unreliable. Notable protozoal infections of ratites are described below (also see Supplemental Table e7 for a list of intestinal coccidia identified in the feces of large ratites).

**Intestinal and extraintestinal coccidia** cause significant disease in both free-ranging and captive kiwi. Birds are commonly simultaneously infected with more than one species of parasite. Four *Eimeria* species have been described and additional species are suspected to occur in the North Island brown kiwi (*Morgan et al., 2017*). One species, *Eimeria kiwi* is present in the intestinal epithelium. An unusual feature of infection is the presence of large macromeronts (*Morgan et al., 2012*) (*Fig. 26.15*). Other identified species include *E. parairii* (suspected to play a role in the development of colorectal polyps), *E. mantelli* (has been identified in feces but has not yet been associated with clinical disease), and *E. apterxyii* (causes renal coccidiosis in the North Island brown kiwi, rowi, and tokoeka) (*Morgan et al., 2012, 2017; Thompson and Wright, 1978*). Additionally, asexual stages of unknown coccidial species have been found in the liver, spleen, lung, and pancreas. Periodic acid Schiff staining of kidney to enhance parasite recognition is recommended as part of routine histopathologic examination (*Morgan et al., 2013*). Eimeriosis has also been described in red-winged tinamous. Infection with the causative agent, *E. rhynchothi*, caused apathy and mal-digested, fetid, pasty feces as well as duodenal hemorrhage and thickening (*Freitas et al., 2006; Marques et al. 2012*). Experimentally infected birds died between 1 and 6 days of patent infection.

**Cryptosporidia** are frequent, incidental findings in the feces of apparently healthy ostriches, with greater prevalence in chicks and juvenile birds (*Wang et al., 2011*). However, an associated syndrome of *cloacal prolapse* is well recognized in ostrich chicks, particularly males. Infection of epithelial cells in the distal rectum and cloaca results in mucosal swelling, epithelial hyperplasia, and mixed heterophilic and mononuclear cell inflammation in the lamina propria (*Penrith et al., 1994; Santos et al., 2005*). Infection with necrosis and inflammation may also occur in the pancreatic ductal epithelium (*Jardine and Verwoerd, 1997*). Molecular investigations have identified *Cryptosporidium baileyi* and *Cryptosporidium avian genotype II* in ostriches (*Meireles
Enteric coccidiosis in a North Island brown kiwi. An *Eimeria* megalomerozoite (macromeront) is present in the villar lamina propria of the small intestine. Coccidiosis is an important cause of morbidity and mortality in young kiwi, with a number of incompletely described *Eimeria* species occurring intra- and extraintestinally. Macromeronts are an uncommon feature of avian coccidiosis. (Photo Courtesy of K. Morgan, Massey University)

**FIGURE 26.15**

North Island brown kiwi due to disseminated protozoal infection led to the identification of *Plasmodium elongatum* in North Island brown kiwi and rowi that were part of captive breeding and captive-rearing creche programs (Banda et al., 2013). Early trophozoites were present in blood smears collected at the time, with a prevalence of 68% by microscopy and 78% by PCR. Parasitemias were low, and subsequent testing did not show the continuing presence of the parasite. The most severe histologic lesion in the sentinel bird was severe interstitial pneumonia, with congestion, edema, and small blood vessel thrombosis. Hypertrophied pulmonary endothelial cells, hepatic Kupffer cells, and macrophages in the lung, liver, and spleen contained intracytoplasmic merozoites or hemozoin (hemoglobin breakdown product).

*Babesia kiwiensis*, a novel, intraerythrocytic parasite, has been identified in both captive and free-living North Island brown kiwi chicks (Jefferies et al., 2008; Peirce et al., 2003). Parasites were present as ring forms, schizonts, and merozoites within erythrocyte cytoplasm, without displacing the host nucleus. Single birds were coinfected with *Hepatozoon kiwii*, also a new species. The oval or elongated gametocytes of *H. kiwii* were present in the cytoplasm of monocytes and, less frequently, lymphocytes, where they distorted and compressed the nucleus of the host cell. *Ixodes anatis*, also known as the kiwi tick, is commonly found on kiwi and is postulated as a vector for these organisms. The significance of these infections is currently unknown.

**Ectoparasites**

Lice and mites are common ectoparasites of ostriches and rheas worldwide (Craig and Diamond, 1996; Huchzermeyer, 1998; Mohammed and Malang, 2015). *Struthiolipeurus struthionis*, the ostrich louse, feeds on the body scales and feather debris of ostriches and causes irritation and pruritus. Adult lice are 3–4 mm long and readily visible; eggs (nits) can be found on the underside of feathers attached to the barbs near the shaft. They live in the groove of the shaft and feed on the pulp of growing feathers. Parasite feeding and excessive grooming can lead to feather damage and loss. Acute parasitic, cystic, lymphoplasmacytic dermatitis, nodular trombiculinosis, has been described in a juvenile ostrich in Brazil as a result of infestation with the trombiculinid mite *Apolonia tigipioen* (Ornelas-Almeida et al., 2007) (also see Supplemental Table e8 for a list of additional select lice and mites of large ratites). In addition to lice and mites, international importations of ostriches and in-contact birds should be examined for the presence of ticks as a range of species of ectoparasitic arthropods have been identified on birds imported from Africa and Europe (Mertins and Schlater, 1991). Feeding by hippoboscid flies and black flies (*Simulium spp.*)
can cause irritation and anemia, and severe dermatitis and conjunctivitis has been described in ostriches swarmed by thrips (*Limothrips denticornis*) (Cooper, 2007b; Huchzer-meyer, 1998).

Tinamous are commonly infected by one or more species of ectoparasite, with up to 15 species reported from a single great tinamou (Marques et al., 2012). Kiwi can host several species of ticks; eight species of chewing lice and five species of feather mites have also been identified (Heath, 2010). The significance of ectoparasitism in these two groups of birds is unclear.

**Prions**

A spongiform encephalopathy similar to that seen with prion disease has been described in ostriches and emus in Germany. Neurological signs were present in most, but not all birds. Lesions included marked vacuolation of the neuropil in the brain stem and medulla, as well as neuronal cytoplasmic vacuolation, particularly in the red and vestibular nuclei and the reticular formation. Mild gliosis, isolated necrotic neurons, and neuronophagia were present in the anterior brainstem. The prion protein PrP was not identified in these cases using IHC labeling (Bello et al., 2017).
There is widespread lymphoplasmacytic perivascular cuffing, most severe at the level of the cerebellar peduncle at the junction of the grey and white matter, accompanied by endothelial hyperplasia and numerous reactive astrocytes. eSlide: VM05216

Avian bornavirus encephalitis, emu, brain. Immunohistochemistry (rabbit polyclonal antiserum against parrot bornavirus 2 (Psittaciform 1 bornavirus) nucleocapsid protein) is positive with nuclear, and to a much lesser degree cytoplasmic, staining in neurons and glial cells. Staining was present in moderate intensity in the cerebellar peduncle and brainstem, and was sporadically positive in the granular layer of the cerebellum and in the cerebral cortex. eSlide: VM05217

Clostridial typhlitis (Clostridium perfringens), ostrich, cecum. There are multifocal to coalescing areas of severe mucosal necrosis with a marked accumulation of cellular debris, heterophils, and bacteria (bacilli) over the surface and in the cecal lumen. eSlide: VM05231

Clostridial enteritis, ostrich, cecum. Brown and Hopps. There are multifocal to coalescing areas of severe mucosal necrosis with a marked accumulation of cellular debris, heterophils, and bacteria (bacilli) over the surface and in the cecal lumen. The positively staining bacilli are highlighted within a mixed bacterial population with Brown and Hopps staining. Brown and Hopps. eSlide: VM05232

Normal yolk sac, ostrich, yolk sac. The yolk sac is normal, and shows the long mucosal villi lined by large vacuolated epithelial cells. These villi are present in the yolk sacs of ostrich chicks until approximately day 14 of age. eSlide: VM05233

Rickets, rhea, bone. The zones of proliferation and prehypertrophy are unremarkable, but the zone of hypertrophy has multiple thick bands of cartilage alternating with thin spicules of bone that extend deep into the diaphysis. There is thin calcification of the cartilaginous matrix, the osteoblast population is poor and there are multifocal clusters of osteoclasts. (see Fig. 26.6). eSlide: VM05236

Baylisascaris larval migrans, tinamou, brain. Randomly affecting predominantly the white matter tracts are multifocal, variably sized areas of vacuolation and cavitation, with variable, predominantly, histiocytic infiltrates. Within these foci are three cross-sections of nematode larvae, 75 µm in diameter, with a smooth cuticle, lateral alae, platymyarian musculature and prominent lateral cords (consistent with Baylisascaris sp.). Axonal swelling and degeneration are contained within dilated myelin sheaths. (see Fig. 26.14). eSlide: VM05234

Bacterial yolk sacculitis, emu, yolk sac. The yolk sac is surrounded by a fibrous wall containing scattered heterophils. Centrally, there are regions of mineralization and of vacuolated material in the epithelial cells, as well as clusters of small coccoid bacilli and rare globules and clusters of amphophilic yolk droplets. eSlide: VM05237

Myodegeneration and myonecrosis, ostrich, pipping muscle. There is edema of the peri- and epimysium, and mild to moderate infiltration of the interstitium with heterophils. There is severe multifocal necrosis of myofibres with increased eosinophilia, contraction bands, fibre fragmentation, and mineralization of the degenerate sarcolemma. On cross section, swelling of myofibres and empty endomysial sheaths are also seen. eSlide: VM05245
GENERAL RATITE MANAGEMENT AND HEALTH REFERENCE MATERIALS

Information on ratites can be found in the biology/ecology, animal management, and veterinary databases and literature. The South African ostrich industry is historic and legendary, and the source of considerable veterinary literature. Many texts and detailed conference proceedings were produced in North America in the 1990s, a time when there was an explosive increase in the breeding of ostriches, emus and, to a lesser extent, common rheas on this continent. The product markets did not; however, expand at the same rate as the breeding programs and the heyday of the North American ratite industry ended within a decade or so. A scan through the more recent literature reveals an increase in publications concerning diseases of ratites emerging from South America and Asia. The following information is intended to provide the reader with a list of the major texts to act as additional sources of information on the management and diseases of a variety of ratite species:

**Basic ornithological information:**

Cabot, J., 2012. Family Tinamidae (Tinamous). In: del Hoyo, J., Elliot, A., Sargatal, J, (Eds.), Handbook of the Birds of the World, Vol. 1. Lynx Edicions, Barcelona, Spain, pp. 112–139.

Folch, A., 2012. Family Apterygidae (Kiwis). In: del Hoyo, J., Elliot, A., Sargatal, J, (Eds.), Handbook of the Birds of the World, Vol. 1. Lynx Edicions, Barcelona, Spain, pp. 104–108.

Folch, A., 2012. Family Casuaridae (Cassowaries). In: del Hoyo, J., Elliot, A., Sargatal, J, (Eds.), Handbook of the Birds of the World, Vol. 1. Lynx Edicions, Barcelona, Spain, pp. 90–97.

Folch, A., 2012. Family Dromaiidae (Emu). In: del Hoyo, J., Elliot, A., Sargatal, J, (Eds.), Handbook of the Birds of the World, Vol. 1. Lynx Edicions, Barcelona, Spain, pp. 98–102.

Folch, A., 2012. Family Rheidae (Rheas). In: del Hoyo, J., Elliot, A., Sargatal, J, (Eds.), Handbook of the Birds of the World, Vol. 1. Lynx Edicions, Barcelona, Spain, pp. 84–88.

Folch, A., 2012. Family Struthionidae (Ostrich). In: del Hoyo, J., Elliot, A., Sargatal, J, (Eds.), Handbook of the Birds of the World, Vol. 1. Lynx Edicions, Barcelona, Spain, pp. 76–82.

**Sources of Information: Ostrich, emu, rhea**

Deeming, D.C. (Ed), 1999. The Ostrich. Biology, Production and Health. CABI Publishing, Wallingford, Oxon, UK, 358 pp.

Hallam, M.G., 1992. The Topaz Introduction to Practical Ostrich Farming. M.G Hallam, Harare, Zimbabwe, 142 pp.

Huchzermeyer, F.W., 1998. Diseases of Ostriches and Other Ratites. Agricultural Research Council, Onderstepoort Veterinary Institute, Onderstepoort, Republic of South Africa, 296 pp.

Jensen, J., Johnson, J.H., Weiner, S.T., 1992. Husbandry and Medical Management of Ostriches, Emus, and Rheas. Wildlife and Exotic TeleConsultants, College Station, Texas, USA, 129 pp.

Kummrow, M.S., 2014. Ratites or Struthioniformes: Struthiones, Rheae, Cassuarii, Apteryges (Ostriches, Rheas, Emus, Cassowaries, and Kiwis), and Tinamiformes (Tinamous). In: Miller, R.E., Fowler, M. (Eds.), Fowler’s Zoo and Wild Animal Medicine, Vol. 8., Elsevier Health Sciences, St. Louis, Missouri, USA, pp. 75–82.

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Minnaar, P., Minnaar, M., 1992. The Emu Farmer’s Handbook. Induna Co., Groveton, Texas, USA. 177 pp.

Scataglini, A., Torti, M.V., Arantes, I.G., 2001. Order Rheiformes (Rheas). In: Fowler, M.E., Cubas, Z.S. (Eds.), Biology, Medicine, and Surgery of South American Wild Animals, Iowa State University Press, Ames, Iowa, USA, pp. 65–71.

Tully, T.N.J., Shane, S.M. (Eds.), 1996. Ratite Management, Medicine and Surgery. Krieger Publishing Company, Malabar, Florida, 188 pp.

Tully, T.N.J., Shane, S.M. (Eds.), 1998. The Veterinary Clinics of North America: Food Animal Practice (Ratites), vol. 14(3), 550 pp.

**Sources of Information: Cassowaries**

Biggs., J.R. (Ed.), 2013. Captive Management Guidelines for the Southern Cassowary Casuarius casuarius johnsonii. Cairns Tropical Zoo, Cairns, Australia. Available from: [http://aszk.org.au/wp-content/uploads/2015/05/2013_FINAL_bird_hm_casso_COMPLETE.pdf](http://aszk.org.au/wp-content/uploads/2015/05/2013_FINAL_bird_hm_casso_COMPLETE.pdf)

Latch,P., 2007. National recovery plan for the southern cassowary Casuarius casuarius johnsonii. Report to Department of the Environment, Water, Heritage and the Arts,
Canberra, Australia. Environmental Protection Agency. 38 pp. Available from: http://www.environment.gov.au/system/files/resources/79235f07-9c32-45fa-b868-eb248691e945/files/stth-cassowary.pdf

Sources of Information: Tinamous
Silveira, L.F., Höfling, E., Gaglianone Moro, M.E., do Nascimento, A.A., Gouveia Arantes, I.M., 2001. Order Tinamiformes (Tinamous). In: Fowler, M.E., Cubas, Z.S. (Eds.), Biology, Medicine and Surgery of South American Wild Animals. Iowa State University Press, Ames, Iowa, USA, pp. 72–80.

Sources of Information: Kiwis
Fraser, I., Johnson, T. with updates by Barlow, S., Travers, C., 2015. Brown Kiwi (Apteryx mantelli) Husbandry Manual, Version 3. Zoo and Aquarium Association, Auckland, New Zealand, 46 pp.

Morgan, K.J., 2008. Kiwi First Aid and Veterinary Care. Science and Technical Publishing, Department of Conservation, Wellington, New Zealand, 103 pp. Available from: http://www.doc.govt.nz/documents/science-and-technical/sap245entire.pdf.

NOTABLE CLINICAL PATHOLOGY INFORMATION AND REFERENCE MATERIALS IN RATITES

Variably detailed information on the hematology and serum or plasma biochemistry of ostriches, emus and, to a lesser extent, rheas, is included in specific ratite texts and general veterinary clinical pathology reference texts that may also include tables of reference intervals. Only a limited amount of the primary literature is summarized. A small number of original research investigations evaluate the effects of blood collection or storage techniques.

A variety of anticoagulants have been recommended for hematology samples in ratites, including lithium heparin, EDTA (preferably in liquid form), and sodium citrate for laser flow cytometry. However, a direct comparison on ostrich blood suggested that heparin resulted in less hemolysis than EDTA.

Biochemistry is often performed on plasma rather than serum, in part due to the development of large gelatinous clots from which serum can be difficult to extract.

Information on the hematology and biochemistry of cassowary, kiwi, and tinamou species is scant and available through the Species360 Zoological Information Management System (https://www.species360.org) and in the primary literature or in conservation workshop proceedings. Lithium heparin should be used for both hematology and biochemistry in the kiwi.

Urine composition has been described for the ostrich.

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TOXIC ELEMENTS DESCRIBED AS CAUSES OF CLINICAL DISEASE AND DEATH IN LARGE RATITE SPECIES

Therapeutic/pest control compounds: colistin, dynaminulin, furazolidone, lincomycin, lindane, mineral oil, morantel, furan, thiabendazole, streptomycin, warfarin.
Feed-related compounds and forage: gizzerosine (causing erosion of the gizzard), mycotoxins (aflatoxin, ochratoxin, T2 toxin, sporodesmin, zearalenone), salt, selenium.

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Shivaprasad, H.L., 1993. Neonatal mortality in ostriches: an overview of possible causes. Proceedings of the Annual Conference of the Association of Avian Veterinarians, Nashville, Tennessee, USA, 282–293.
| Order                  | Family              | Geographic Origin | Common Names                                      | Genus and Species                          |
|------------------------|---------------------|-------------------|---------------------------------------------------|--------------------------------------------|
| Apterygiformes<sup>a</sup> | Apterygidae         | New Zealand       | Great spotted kiwi                               | Apteryx haastii                             |
|                        |                     |                   | Little spotted kiwi                             | Apteryx owenii                              |
|                        |                     |                   | North Island brown kiwi                          | Apteryx mantelli                            |
|                        |                     |                   | Okarito kiwi (rowi)                              | Apteryx rowi                                |
|                        |                     |                   | Southern brown kiwi (tokoeka)                    | Apteryx australis                           |
| Casuariiformes         | Casuariidae         | Australasia       | Dwarf cassowary                                  | Casuarius bennetti                          |
|                        |                     |                   | Northern cassowary                               | Casuarius unappendiculatus                  |
|                        |                     |                   | Southern cassowary                               | Casuarius casuarius                         |
| Casuariiformes         | Casuariidae         | Australia         | Emu                                              | Dromaius novaehollandiae                    |
| Struthioniformes       | Struthionidae       | Africa            | Common ostrich                                   | Struthio camelus                            |
|                        |                     |                   | Somali ostrich                                    | Struthio molybdophanes                      |
| Rheiformes             | Rheidae             | South America     | Lesser rhea                                      | Rhea pennata                               |
|                        |                     |                   | Greater rhea                                      | Rhea americana                              |
| Tinamiformes           | Tinamidae<sup>b</sup> | South America   | Tawny-breasted tinamou                           | Notohcercus julius                          |
|                        |                     |                   | Highland tinamou                                 | Notohcercus bonapartei                      |
|                        |                     |                   | Hooded tinamou                                   | Notohcercus nigrocapillus                   |
|                        |                     |                   | Gray tinamou                                      | Tinamus tao                                 |
|                        |                     |                   | Solitary tinamou                                 | Tinamus solitarius                          |
|                        |                     |                   | Black tinamou                                     | Tinamus osgoodi                             |
|                        |                     |                   | Great tinamou                                     | Tinamus major                               |
|                        |                     |                   | White-throated tinamou                           | Tinamus guttatus                            |
|                        |                     |                   | Berlepsch's tinamou                              | Crypturellus berlepschi                     |
|                        |                     |                   | Cinereous tinamou                                | Crypturellus cinereus                       |
|                        |                     |                   | Little tinamou                                    | Crypturellus soui                           |
|                        |                     |                   | Tepui tinamou                                     | Crypturellus pteropei                       |
|                        |                     |                   | Brown tinamou                                     | Crypturellus obsoletus                      |
|                        |                     |                   | Undulated tinamou                                | Crypturellus undulatus                      |
|                        |                     |                   | Pale-browed tinamou                              | Crypturellus transfasciatus                 |
|                        |                     |                   | Brazilian tinamou                                | Crypturellus strigulosus                    |
|                        |                     |                   | Grey-legged tinamou                              | Crypturellus duidae                         |
|                        |                     |                   | Red-legged tinamou                               | Crypturellus erythropus                     |
|                        |                     |                   | Yellow-legged tinamou                            | Crypturellus notecnagrae                    |
|                        |                     |                   | Black-capped tinamou                             | Crypturellus atrocapillus                   |
|                        |                     |                   | Thicket tinamou                                  | Crypturellus cinnamomeus                    |
|                        |                     |                   | Slaty-breasted tinamou                            | Crypturellus boucardi                       |
|                        |                     |                   | Choco tinamou                                     | Crypturellus kerriae                        |
|                        |                     |                   | Variegated tinamou                               | Crypturellus variegatus                     |
|                        |                     |                   | Rusty tinamou                                     | Crypturellus brevirostris                   |
|                        |                     |                   | Bartlett's tinamou                               | Crypturellus bartletti                      |
|                        |                     |                   | Small-billed tinamou                              | Crypturellus parvirostris                   |
|                        |                     |                   | Barred tinamou                                    | Crypturellus casiquare                      |
|                        |                     |                   | Tataupa tinamou                                   | Crypturellus tataupa                        |
|                        |                     |                   | Red-winged tinamou                               | Rhynchosotus ruifescens                     |
|                        |                     |                   | Huayco tinamou                                    | Rhynchosotus maculicollis                   |
|                        |                     |                   | (Rhynchotus)<sup>c</sup>                           |                                            |
|                        |                     |                   | Taczanowski's tinamou                             | Notohroctta taczanowskii                    |
|                        |                     |                   | Ornate tinamou                                    | Notohroctta ornata                          |
|                        |                     |                   | Chilean tinamou                                   | Notohroctta perdicaria                      |
|                        |                     |                   | Brushland tinamou                                 | Notohroctta cinerascens                     |
|                        |                     |                   | Andean tinamou                                    | Notohroctta pentlandii                      |
|                        |                     |                   | Curve-billed tinamou                              | Notohroctta curvimorristis                  |
|                        |                     |                   | White-bellied nothura                             | Notohroctta boraquira                       |
|                        |                     |                   | Lesser nothura                                    | Notohroctta minor                           |
|                        |                     |                   | Darwin's nothura                                  | Notohroctta darwini                         |
|                        |                     |                   | Spotted nothura                                   | Notohroctta maculosa                        |
|                        |                     |                   | Chaco nothura                                     | Notohroctta chacoensis                      |
|                        |                     |                   | (Nothura)<sup>d</sup>                              |                                            |
|                        |                     |                   | Dwarf tinamou                                     | Taoniscus nanus                             |
|                        |                     |                   | Elegant crested tinamou                           | Eudromia elegans                            |
|                        |                     |                   | Quebracho crested tinamou                         | Eudromia formosa                            |
|                        |                     |                   | Puna tinamou                                      | Tinamotis pentlandii                        |
|                        |                     |                   | Patagonian tinamou                                | Tinamotis ingoufi                           |

<sup>a</sup>Gill, F.B., Donsker, D.B. (Eds.), 2018. IOC World Bird List (v 8.1), doi:10.14344/10C.ML.8.1.
<sup>b</sup>Sales, J., 2005. The endangered kiwi: a review. Folia Zool. 54, 1–20.
### Table e2. Comparison of Select Anatomic Features Among Large Ratites\(^{a,b,c,d}\)

| Anatomic features   | Ostrich\(^{e,f}\)                                                                 | Emu\(^{m,n}\)  | Rhea                                                                 | Cassowary                                                                 |
|---------------------|----------------------------------------------------------------------------------|----------------|----------------------------------------------------------------------|----------------------------------------------------------------------------|
| Wing structure      | Large wing, three digits, cornified hooks on tips of alula- and distal phalanx of two anterior digits. Scapula, coracoid and clavicle fused in adult. | Vestigial wing, single clawed digit. | Medium sized wing, claw on alular digit and sometimes other two digits. | Small wing, curved claw on major digit.                                     |
| Pelvic and leg structure | Large digit three with strong toenail, smaller digit four with or without toenail. Ischial and pubic bones form a pubic symphysis. Unfused tarsal bone could be mistaken for the patella, which is absent.\(^g\) | Digits two, three, and four with strong toenails. Unfused tarsal bone could be mistaken for the patella, which is absent.\(^h\) | Digits two, three, and four with strong toenails. | Digits two, three, and four with strong toenails. Nail on digit two is an elongated sharp spike. |
| Gastrointestinal system |                                                                                   |                |                                                                      |                                                                            |
| Oral cavity and esophagus | Smooth, blunt, U-shaped tongue and well-developed pharyngeal tonsils with crypts. Taste buds not evident.\(^i\) | Tongue has a serrated edge. Lateral projections of the pharyngeal folds form pharyngeal tonsils. Taste buds present. | Tongue and tonsils have pointed V-shaped tips. |                                                                            |
| Proventriculus      | Large, curves over ventriculus, thin walled or saccular with small glandular patch on greater curvature (dorsal and caudal walls). Large proventricular-ventricular opening. Frequently contains large amounts of water. | Large, thick walled, spindle shaped with entire surface glandular. | Small, thin walled with glandular patch on the greater curvature. | Large, spindle shaped.                                                     |
| Ventriculus         | Heavily muscled.                                                                  | Ventriculus larger than proventriculus, muscling of wall varies with diet. | Large, sac-like. | Elongated, moderate muscling.                                          |
| Gall bladder        | Not present                                                                      | Present        | Present                                                              | Present                                                                   |
| Liver               | Left lobe subdivided into three smaller lobes, larger right lobe undivided.       |                |                                                                      |                                                                            |
| Intestines          | Unique secondary duodenal loop. Hepato-enteric duct opens into the descending loop of the duodenum; the pancreatic duct opens into the ascending loop near the jejunal junction. Coiled jejunum and ileum. Elongated ceca with a sacculated appearance except at the thick-walled apices, complex frilled internal spiral fold. Right cecum is generally longer than the left, single blended opening to the intestinal tract. Voluminous large intestine: proximal thin walled sacculated portion; narrow thin-walled distal portion. Dilatation of a sac-like area with feces proximal to rectocoprodeal fold is described.\(^l\) Small intestine, cecum, and colon form 36, 7, and 57% of the total length of the gastrointestinal tract, respectively. | Elongated small intestine, muscularis progressively thins from the jejunum to the distal ileum. Short tubular ceca with longitudinal rugae and villi extending most of the length. Small ceca and proportionally short large intestine with mucosal folds. Large rectal villi. Small intestine, cecum, and colon form 90, 3, and 7% of the total length of the gastrointestinal tract, respectively. | Proportionally longest ceca, sacculated with a valvular transverse fold in two alternating longitudinal layers. Small intestine, cecum, and colon form 61, 21, and 17% of the total length of the gastrointestinal tract, respectively. | Short to vestigial non-functional ceca. Short large intestine. |
| Cloaca | The coprodeum or proctodeum (depending on author) stores urine like a bladder; a well-developed rectoproctodeal fold prevents retrograde flow of urine and urates. Urodeum may or may not be present (depending on author). Well-developed coproproctodeal fold. Bursa of Fabricius is an integral part of dorsal and lateral walls of the proctodeum. | Bursa of Fabricius is an integral part of dorsal and lateral walls of the proctodeum. | Bursa of Fabricius is a cranial appendix of the proctodeum. |
| Urogenital system | Phallus with a phallic sulcus (no internal cavity) that folds on ventrum of proctodeum.¹ | Testes are black. Spiral phallus has cavity which gives appearance of urethra but does not communicate to urinary tract, much of phallus lies like an inverted glove finger in a pocket below the mucosa of the proctodeum. | Dilated pouch of ureter stores urine. Ureters are described as opening into the coprodeum, rather than the urodeum, in an adult male (as compared to an adult female and a juvenile male). Spiral phallus has cavity which gives appearance of urethra but does not communicate to urinary tract, much of phallus lies like an inverted glove finger in a pocket below the mucosa of the proctodeum. |
| Respiratory system | Femur is the only pneumatized bone. | Femur is the only pneumatized bone. Prominent discontinuity in the ventral tracheal rings cranial to the thoracic inlet that communicates with a large subcutaneous membrane lined pouch in adult birds. | Information on bone pneumatization not available. Information on bone pneumatization not available. |
| Integumentary system | Prominent cutaneous callosities on ventral pressure points: sternum, plantar metatarsal pad, below the prominences of the pubic bones. Large featherless areas (apteria). | Feathers have a double rachis. | Sternal callosity. Firm, keratinized casque, or helmet, on the top of the head. Feathers appear to have a double rachis due to large hypopenna. Flight feathers are thin rods of solid keratin. |

(Continued)
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Table e2. Comparison of Select Anatomic Features Among Large Ratites\(^{a,b,c,d}\) (Cont.)

| Anatomic features | Ostrich\(^{a,e,f}\) | Emu\(^{m,n}\) | Rhea | Cassowary |
|-------------------|---------------------|--------------|------|-----------|
| Thymus            | Varies, may be rounded or longer and lobulated, at base of the neck cranial to first ribs. | Long, flat, J-shaped, and multilobulated. | Flattened hemisphere. | No published description. |
| Spleen            | Oval and bean-shaped to elongated or sausage-shaped and triangular on transverse section. | Long and cylindrical. | Cylindrical, bean-shaped. | Flattened, irregularly polygonal in section. |

\(^{a}\)Cho, P., Brown, R., Anderson, M., 1984. Comparative gross anatomy of ratites. Zoo Biol. 3(2), 133–144.
\(^{b}\)Fowler, M.E., 1991. Comparative clinical anatomy of ratites. J. Zoo Wildl. Med. 22(2), 204–227.
\(^{c}\)McLelland, J., 1989. Anatomy of the avian cecum. J. Exp. Zool. 252(3), 2–9.
\(^{d}\)Berens Von Rautenfeld, D., Budras, K.-D., 1982. The bursa cloacae (Fabricii) of struthioniforms in comparison with the bursa of other birds. J. Morphol. 172(1), 123–138.
\(^{e}\)Bezuidenhout A.J., 1986. The topography of the thoraco-abdominal viscera in the ostrich (Struthio camelus). Onderstepoort J. Vet. Res. 53(2), 111.
\(^{f}\)Bezuidenhout, A.J., 1999. Anatomy. In: Deeming, D.C. (Ed.), The Ostrich. Biology, Production and Health. CABI Publishing, Wallingford, UK, pp. 13–49.
\(^{g}\)Bezuidenhout, A., Burger, W.P., 1993. The incidence of tibiotarsal rotation in the ostrich (Struthio camelus). J. South Afr. Vet. Assoc. 64(4), 159–161.
\(^{h}\)Tivane, C., 2008. The morphology of the oral cavity, pharynx and oesophagus of the ostrich (Struthio camelus). MSc thesis, University of Pretoria, Pretoria, Republic of South Africa.
\(^{i}\)Skadhauge, E., Erlwanger, K.H., Ruziwa, S.D., Dantzer, V., Elbrønd, V., Chamunorwa, J.P., 2003. Does the ostrich (Struthio camelus) coprodeum have the electrophysiological properties and microstructure of other birds? Comp. Biochem. Physiol. Part A Mol. Integr. Physiol. 134(4), 749–755.
\(^{j}\)Gandini, G.C.M., Keffen, R.H., 1985. Sex determination of the South African ostrich (Struthio camelus). J. South Afr. Vet. Assoc. 56(4), 209–210.
\(^{k}\)Herd, R.M., 1985. Anatomy and histology of the gut of the emu (Dromaius novaehollandiae). Emu, 85(1), 43–46.
\(^{l}\)Herd, R.M., Dawson, T.J., 1984. Fiber digestion in the emu, Dromaius novaehollandiae, a large bird with a simple gut and high rates of passage. Physiol. Zool. 57(1), 70–84.
### Table e3. Lymphoid Neoplasia in Large Ratites

| Species | Age   | Sex | Published Diagnosis | Description                                                                                                                                 |
|---------|-------|-----|---------------------|--------------------------------------------------------------------------------------------------------------------------------------------|
| Ostrich | 7 years | F   | Lymphosarcoma<sup>a</sup> | 4 cm firm ulcerated mass partially occluding cloacal lumen and resulting in clitoral prolapse. Resembled lymphoid leucosis in poultry in that there was “lymphatic spread.” |
| Ostrich | 20 months | M   | Lymphoma<sup>a</sup> | Multilobulated subcutaneous mass at the base of the neck cranial to the thoracic inlet. Similar nodules throughout lung, liver, and spleen. |
| Ostrich | 3 years | F   | Lymphoid leukemia<sup>b</sup> | Ascites and hepatomegaly with whitish tumor nodules in spleen, large intestine, kidney and cloacal region. On histopathology, the lesions consisted of coalescing foci and diffuse infiltrates of immature lymphoid cells. Cells had large uniform nuclei, a thin rim of basophilic cytoplasm, and little pleomorphism. Mitotic figures were scanty. The enteric nervous system was not affected. |
| Ostrich | 3 years | F   | Multicentric, diffuse lymphoma of intermediate differentiation in the leukemic phase<sup>c</sup> | 3 × 5 cm bilobed mass in the right atrium and immediately cranial and caudal to the thoracic inlet, hepatomegaly with 3–5 mm random white firm masses on capsule and in section. Neoplastic tissue consisted of sheets of small lymphocytes with oval nuclei, small amounts of clumped chromatin, scant eosinophilic cytoplasm. Diffuse bone marrow effacement was present. The ostrich also had a lymphocytic leukocytosis and monoclonal gammopathy in the late beta to early gamma ranges, with a faint trailing oligoclonal peak in the late gamma range. |
| Ostrich | Unknown | F   | Lymphoid leukemia<sup>d</sup> | Affecting reproductive tract, abdominal viscera, and mediastinal “nodes.” |
| Emu     | 6 months | F   | Lymphosarcoma<sup>a</sup> | Skin lump highly suggestive of lymphosarcoma on cytological examination. |
| Emu     | Unknown | F   | Lymphoma<sup>a</sup> | No details provided. |

<sup>a</sup>Cook, 2008. Disease entities of farmed ratites in New Zealand. Surveillance 24(8), 10–12.

<sup>b</sup>Garcia-Fernandez, R.A., Perez-Martinez, C., Espinosa-Alvarez, J., Escudero-Diez, A., Garcia-Marin, J.F., Nunez, A., Garcia-Iglesias, M.J., 2000. Lymphoid leukemia in an ostrich (*Struthio camelus*). Vet. Rec. 146(23), 676.

<sup>c</sup>VanDer Heyden, N., Fulton, R.M., DeNicola, D.B., Hicks, K., 1992. Lymphoma in an ostrich (*Struthio camelus*). Proceedings of the Annual Conference of the Association of Avian Veterinarians, New Orleans, Louisiana, USA, pp. 310–312.

<sup>d</sup>Hicks, K.D., 1993. Ostrich reproduction. J. Zoo Wild Anim. Med. 3, 203–206.

<sup>e</sup>Adby, M.J., Howarth, E.W. 1995. Zinc levels in ratites in the southeastern United States. Proceedings of Joint Conference of the American Association of Zoo Veterinarians, Wildlife Disease Association, and American Association of Wildlife Veterinarians. East Lansing, Michigan, USA, pp. 428–429.
### Table e4. Viral Infections in the Large Ratites

| Viral Family | Viral Species | Common Name of the Disease | Species Affected and Sample Geographic Locations | Disease Characteristics |
|--------------|---------------|---------------------------|-------------------------------------------------|-------------------------|
| **DNA Viruses** | | | | |
| Adenoviridae | Presumptive Aviadenoviruses | Adenovirus infection | Ostrich (Italy, UK, USA); emu (USA) | Untyped adenoviruses and adenoviruses related to Fowl Adenoviruses 1 and 8 have been associated with diarrhea and high mortality in ostrich chicks and with the laying of chalky shelled eggs by female ostriches. See text chapter for additional details and references. |
| Circoviridae<sup>a</sup> | Gyrovirus; Chicken anemia virus | | Ostrich (Spain) | Virus was identified by immunohistochemistry in ostrich chicks with anorexia, diarrhea and dyspnea and 100% mortality. There was severe splenic lymphoid depletion with eosinophilic inclusions in macrophages. |
| Herpesviridae<sup>b</sup> | | | Ostrich (South Africa) | Virus was isolated from the livers of 3-week-old ostrich chicks during a brief mortality event. Gross lesions included pale friable livers; on histology there was severe biliary hyperplasia and hepatocellular necrosis. Disease could not be experimentally reproduced. The results of microneutralization tests suggested the presence of a novel avian herpesvirus. |
| Poxviridae<sup>b–g</sup> | Avipox, Fowlpox virus | Avian pox | Ostrich (Australia, Israel, South Africa, USA) | Dry form affecting the head is most common. Gross and histopathologic lesions are characteristic for the disease with cytoplasmic inclusion bodies generally present. Mortality can be high as a result of blindness and secondary infection. Domestic poultry are most probably the source of infection; however, in one South African report chickens could not be infected with ostrich origin virus. |
| **RNA viruses** | | | | |
| Birnaviridae<sup>h–j</sup> | Avibirnavirus; Infectious bursal disease virus | Infectious bursal disease (OIE reportable) | Ostrich (South Africa, UK, USA); rhea (USA) | Birna-like viruses have been isolated or visualized by EM from the bursa of Fabricius of an ostrich chick with ulcerative cloacitis deriving from a group of 8–to–25–day–old chicks that died with severe fibrinonecrotic ulcerative cloacitis and colitis and bursal lymphoid necrosis; intestinal contents of rhea chicks with enteritis and lymphoid necrosis in the bursa of Fabricius; intestinal contents and liver and pancreas of an impacted ostrich chick; and intestinal contents of adult ostriches. Mean seropositivity was 15% in 149 juvenile farmed ostriches from Zimbabwe by ELISA, and 61% positive by VN in 74 breeding ostriches from the UK. Virus cultured from two ostrich chicks in the UK had a close relationship with IBD type 2 virus. |
| Bornaviridae | Bornavirus; Mammalian 1 bornavirus | Bornavirus | Ostrich (Israel) | An approximately five year outbreak of neurologic disease affected ostrich chicks in Israel. Laboratory investigation indicated the cause as being infection with Bornavirus Disease virus. See text chapter for additional details and references. |
| Bornaviridae | Bornavirus; Waterbird 1 bornavirus | Avian bornavirus/Aquatic bird bornavirus 1 | Emu (Canada) | A prolonged clinical course involving gastrointestinal and neurological signs preceded a necropsy diagnosis of infection with an avian bornavirus in a captive emu. See text chapter for additional details and reference. |
| Family          | Genus, Species                                    | Species                                           | Notes                                                                                                                                                                                                 |
|-----------------|---------------------------------------------------|---------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Bunyaviridae    | Nairovirus; Crimean-Congo hemorrhagic fever virus  | Ostrich (South Africa)                            | Ostriches become viremic for 1–4 days after experimental infection, and can become naturally infected through bites of infected tick vectors. The birds do not develop disease but can seroconvert, and can act as a source of infection to humans at slaughter. |
| Coronaviridae   | Presumptive coronavirinae                         | Enteric coronavirus, rhea (USA)                   | Particles resembling coronaviruses have been seen on transmission EM from intestinal contents or enterocytes in sick or dead young chicks with histologic lesions consistent with those seen with coronaviral enteritis in other species (flattening and loss of superficial villar epithelium, villar atrophy and fusion, crypt epithelial necrosis and hyperplasia). Hemorrhagic typhlitis has also been associated with coronaviral presence. Both explosive outbreaks and more chronic “fading chick” syndrome have been seen. The frequent occurrence of secondary clostridial-associated necrotizing enteritis was described from South Africa. There are no reports of viral isolation. |
| Flaviviridae    | Flavivirus; Wesselsbron virus                      | Wesselsbron disease                               | Virus was isolated from the spleens of 4-month-old ostrich chicks undergoing an outbreak of high mortality. Subsequent inoculation of 1-day-old, 6-month-old, and adult birds did not result in illness or mortality. Seropositivity in tested slaughter birds was up to 50%. In retrospect, the mortality outbreak was considered likely not to be the result of Wesselsbron virus infection. |
|                | (unconfirmed)                                      |                                                   | Virus was isolated, but not fully characterized, from young ostriches with cholangiohepatitis characterized by nonspecific clinical signs, high morbidity but low mortality, and necrosis of biliary epithelium and a mild round cell infiltrate. |
| Orthomyxoviridae| Orthomyxovirus; Influenza virus A                  | Avian influenza (OIE reportable)                  | Avian influenza has been diagnosed in all the large ratite species, with a range of high and low pathogenic strains identified. Disease ranges from subclinical to 100% mortality, with the respiratory system frequently affected. Wild birds are generally the initial source of infection. See text chapter for additional details and references. |
| Paramyxoviridae | Avulavirus; Newcastle disease virus                | Avian paramyxovirus-1 (OIE reportable)           | Newcastle disease virus has been isolated from ostriches with and without associated disease; there are no pathognomonic lesions in this species. Sudden death is the most common presentation in chicks and juveniles. See text chapter for additional details and references. |
| Paramyxoviridae |                                                        |                                                   | Paramyxoviral particles have been seen in the feces of 2– to 3-month-old ostriches with enteritis. The virus has been cultured and experimental vaccination reduced clinical disease, implying causality. Other enteric pathogens are frequently also present and thus lesions vary. |
| Picornaviridae  | Enterovirus                                       | Ostrich (South Africa, USA)                       | An enterovirus was identified by electron microscopy in the feces of ostrich chicks less than one week of age that had diarrhea and nervous signs. Catarrhal enteritis was seen on gross and microscopic examination. The virus could not be cultured. |
| Reoviridae      | Enteric reovirus                                  | Ostrich (South Africa, USA)                       | Reoviruses have been isolated from ostrich chicks with enteritis, including an outbreak with high mortality in South Africa. Chicks also had necrotic enteritis with Clostridium sp. isolated. The significance of these reoviruses is unknown. |

(Continued)
**Table e4. Viral Infections in the Large Ratites (Cont.)**

| Viral Family | Viral Species | Common Name of the Disease | Species Affected and Sample Geographic Locations | Disease Characteristics |
|--------------|---------------|----------------------------|-----------------------------------------------|-------------------------|
| *Togaviridae* | Alphavirus; Eastern and Western encephalitis viruses | Eastern (EEE) and western (WEE) equine encephalitis. (both OIE reportable) | Emu, cassowary (EEE, USA) Emu, rhea (WEE, USA) | EEE causes high mortality in emus; disease has also been described in cassowaries. Pathologic findings primarily reflect vascular damage. WEE also primarily affects emus, but has been described in rheas. Disease is predominantly neurological. Both viruses are arthropod borne. See text chapter for additional details and reference. |

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Table e5. Select Bacterial Diseases in Large Ratites

| Bacterial Agent | Species Affected and Sample Geographic Locations | Disease Characteristics |
|-----------------|-----------------------------------------------|-------------------------|
| **Gram-positive Bacteria** | | |
| *Bacillus anthracis*<sup>a–c</sup> | Ostrich (South Africa, Namibia) | Ostriches are believed to be the avian species most susceptible to anthrax, possibly due to their lower body temperature. Individual cases and outbreaks of sudden death were reported from farmed ostriches in the late 1800s and early 1900s in South Africa, in wild ostriches in Etosha National Park, Namibia as part of a large outbreak between 1966 and 1974 (associated with overuse of contaminated artificial waterholes) and in an outbreak in farmed ostriches in Indonesia in 2001. As in mammals, diagnosis has been based on visualization of *B. anthracis* in blood smears. Classical necropsy findings; generalized congestion, hemorrhages, and poorly coagulated blood, are also seen in ostriches. |
| *Clostridium perfringens*<sup>d–h</sup> | Ostrich, lesser rhea (single outbreak), emu (single case) (worldwide) | *Clostridium perfringens* in an important cause of necrotizing enteritis in ostrich chicks, with mortality approaching 100% in some outbreaks. Diagnosis is based on the presence of characteristic gross and microscopic lesions in the small intestines, including the finding of Gram-positive rod-shaped bacteria on intestinal smears or in section in association with necrotic tissue; *C. perfringens* isolation from intestinal content or tissue; and toxin typing using multiplex PCR. *C. perfringens* toxin type A is most common, with genes for the alpha and beta toxins, enterotoxin, and netB identified. Surveys have not shown a difference in the isolates from ostriches with and without intestinal disease; carriage is common in healthy birds. |
| *Clostridium difficile*<sup>i,j</sup> | Ostrich, common rhea, emu (South Africa, USA) | *C. difficile* infection has been the cause of outbreaks of severe acute necrotizing typhlitis and colitis, and a cause of necrotizing and granulomatous hepatitis in young ostrich chicks. Toxins A and B have both been identified. |
| *Clostridium chauvoei*<sup>k</sup> | Ostrich (Israel) | *C. chauvoei* was cultured and identified using fluorescein-labeled antiserum in association with recumbency, toxemia, and acute mortality in two adult ostriches in Israel. Lesions included hemorrhagic enteritis, multifocal necrosis in liver and kidney, and fibrinous exudation into the pericardial sac. |
| *Clostridium sordelli*<sup>h,l</sup> | Ostrich, emu, lesser rhea (USA) | *C. sordelli* has been reported as a cause of acute necrotizing hepatitis in a group of young ostrich chicks, and as well was cultured in combination with *C. perfringens* from an emu and a lesser rhea with necrotizing enteritis and typhlocolitis, respectively. |
| *Erysipelothrix rhusiopathiae*<sup>m–p</sup> | Emu (worldwide) | The majority of reported cases are of sudden death as a result of septicemia in juvenile emu up to approximately 1-year of age. Serotypes 1b, 5 and 21 have been identified. Most prevalent lesions include widespread congestion, cardiac and serosal petechiation, multifocal parenchymal necrosis with colonies of bacteria, and thromboemboli. Poor environmental conditions may predispose to disease, and vaccination can be protective. |
| **Gram-negative bacteria** | | |
| *Brachyspira hyodysenteriae*, *(known also as Serpulina sp., Treponema sp., intestinal spirochetosis), rarely B. pilosicoli*<sup>q–u</sup> | Common rhea (predominantly USA, probably worldwide)<sup>p</sup> | An important cause of high mortality in common rhea, particularly chicks and juvenile birds, as a result of fibrinonecrotizing typhlocolitis. Cecal cores and mucosal ulceration with pseudomembranes are characteristic; histologic changes vary but include heterophilic to granulomatous inflammation associated with necrosis and the presence of spirochetes. Tentative diagnosis can be made by finding spirochetes on cecal or intestinal wet mounts or smears, or in silver-stained microscopic sections. Laboratory confirmation by standard means (PCR, immunofluorescence, etc.). Earlier reports described coinfection with *Trichomonas*-like protozoa. |
| *Bordetella avium*<sup>e,v,w</sup> | Ostrich (worldwide) | Causes of upper respiratory infection and pseudomembranous tracheitis in ostrich chicks. |

(Continued)
Table e5. Select Bacterial Diseases in Large Ratites (Cont.)

| Bacterial Agent | Species Affected and Sample Geographic Locations | Disease Characteristics |
|-----------------|-------------------------------------------------|-------------------------|
| Campylobacter jejuni jejuni C. coli | Ostrich, common rhea, emu (bacterial isolation only) (worldwide) | Campylobacter sp. organisms have been frequently identified from the intestinal (in particular cecal and colonic) contents, cloacal swabs, and feces of healthy ostriches on the farm or at slaughter, and at necropsy have been isolated from the intestines of birds with and without pathologic lesions. Disease states linked with Campylobacter sp. infection include sudden death in ostrich chicks and young rheas as a result of typhlocolitis and/or multifocal necrotizing or granulomatous hepatitis. Affected livers were swollen with variably sized pale foci on the surface and cut section. Histologically, there was acute to chronic multifocal hepatic necrosis with lesions particularly around portal tracts, cholangitis, bile duct proliferation, periportal fibrosis, and portal vein thrombosis. Infiltrating inflammatory cells included heterophils, lymphocytes and plasma cells; in some birds, granulomatous inflammation was the striking finding. Argyrophilic curved and serpentine bacilli may be noted in smears or section. C. jejuni was also isolated from the yolk sacs of neonatal ostrich chicks with granulomatous yolk sacculitis. |
| Chlamydia psittaci | Ostrich, rhea (worldwide) | Clinical presentation and pathology are similar to those in any avian species; including sudden death with high mortality, systemic evidence of inflammation and necrosis, and conjunctivitis. Diagnosis is performed using standard techniques. The sources of infection for ratites are thought be local wild birds, particularly Columbiformes. Seropositivity has been identified in apparently healthy captive and wild (common rhea, Argentina) birds. |
| Lawsonia intracellularis | Ostrich, common rhea, emu (USA and Germany) | Initially reported from a group of emu chicks with 23% morbidity and 16% mortality associated with rectal prolapse due to cloacitis. The distal rectal mucosa was thickened and hyperplastic with dilated glands full of mucus and large numbers of intracellular, silver-staining, comma-shaped bacteria. Necrotizing lesions were also present in the rectal mucosa and cloaca. Diagnosis was based on TEM and indirect immunofluorescence staining. Proliferative enteritis was described in an ostrich and a rhea - the ileal mucosa was thickened with cryptal hyperplasia and large numbers of curved intracellular silver-staining bacteria in the proliferative epithelial cells. PCR was used to confirm the diagnosis in both cases. |
| Hemophilus spp. | Ostrich (Israel, probably USA) | Serous to purulent rhinitis, sinusitis, and conjunctivitis are described in ostrich chicks. The disease can be highly contagious. |
| Salmonella spp. | Ostrich, emu, common and lesser rhea, southern cassowary. (worldwide) | There are two separate bodies of information on Salmonella spp. infections in ratites—one relating to clinical disease and the second to carriage and shedding rates, seropositivity, and the public health risk in slaughter birds. A wide range of Salmonella spp. has been identified in or from ratites, likely reflecting local sources of infection. Description of disease resulting from S. pullorum is notable by its absence, although seropositivity has been identified. Experimental infection of emu with S. pullorum resulted in seroconversion but no evidence of organ infection or shedding. Seroconversion also occurred in experimentally infected ostriches, with 1/8 birds developing lesions from which S. pullorum was reisolated. Salmonella spp. can be important causes of embryo death. Clinical and pathologic scenarios associated with salmonellosis include acute death and more chronic disability; omphalitis; yolk sacculitis; oophoritis (S. arizonae); necrotizing enteritis, typhlitis, and colitis; hepatitis; and pneumonia. A rectal strictur, identical to the lesion associated with salmonellosis in pigs, was described in an adult ostrich; however, no Salmonella sp. was isolated. |
Although frequently described as an uncommon disease, the frequency of description makes one wonder whether the condition is particularly uncommon. Disease reports involve captive animals, with the exception of diagnoses in wild southern cassowary in Australia. Gross lesions are as per other avian species: granulomatous nodules particularly in spleen, liver, abdominal serosa, and intestine and cloaca that frequently have an ulcerated mucosal surface. Areas of predilection also appear to be the ocular conjunctiva, the oropharynx in association with the pharyngeal tonsils, and subcutaneously at the base of the neck. Granulomatous nodules can be very large, for example, 30 cm in diameter or 2 kg in mass. The microscopic appearance is consistent with that in most avian species, small lesions are frequently composed of solid sheets of epithelioid macrophages whereas in larger lesions a central, caseonecrotic core without mineralization, and surrounded by fibrous connective tissue is more common. Langhan’s type multinucleated giant cells, macrophages, heterophils, lymphocytes, and plasma cells can all be present in varying combinations. The number of acid-fast organisms present can vary tremendously. Standard methods of culture and molecular characterization of organisms are appropriate. Mycobacterium avium is the most commonly identified species; however, a single case of M. bovis was identified in an ostrich.

### Mycoplasma struthionis sp. nov.

M. struthionis sp. nov. and Mycoplasma sp. Ms02 are closely related to M. synoviae. Mycoplasmosis has a significant economic impact on feedlot birds, and with exacerbation by extreme weather and possibly other stressors. Considerable research has been undertaken towards molecular characterization of these mycoplasmas and to develop an effective vaccine.

In 2004, three novel species of mycoplasma were identified from ostriches in South Africa. M. nasistruthionis sp. nov. and Mycoplasma sp. Ms02 are closely related to M. synoviae. Mycoplasmosis has a significant economic impact on ostrich industry with losses resulting from upper respiratory (rhinostrachitis) and air sac infections, particularly in feedlot birds, and with exacerbation by extreme weather and possibly other stressors. Considerable research has been undertaken towards molecular characterization of these mycoplasmas and to develop an effective vaccine.

Information on, and particularly pathogenicity of, other mycoplasmal infections in ratites is sporadic and frequently without detailed characterization of any organisms identified. Initial, non-molecular characterization of some isolates may not have been accurate. M. synoviae was identified by PCR from lung samples taken from slaughter ostriches in Iran. M. cloacae has been identified from tracheal, or air sac swabs from ratites, a mycoplasma that reacted with M. synoviae antibody was present in the joint of an emu with polyarthritis, M. synoviae was isolated from tracheal samples from juvenile common rheas with upper respiratory infection and sinusitis, and colonization of the trachea can occur in young ostriches experimentally infected with M. gallisepticum. Disease as a result of the major poultry mycoplasmoses has not been diagnosed in ratites.

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Welsh, R.D., Vanhooser, S.L., Dye, L.B., Niemann, R.W., 1997. Salmonella infection in ratites: diagnosis, epidemiology, and clinical significance. Vet. Med. 92(2), 193–198.
| Parasite | Location in Host | Species Affected and Geographic Location | Disease Characteristics |
|----------|------------------|------------------------------------------|-------------------------|
| **Proventricular/Ventricular Nematodes** | | | |
| *Libyostrongylus douglassii* (Trichostrongyloidea) | Proventriculus and gizzard | Ostrich (likely southern African origin, widespread distribution with animal movement) | Blood feeding nematodes that burrow into the proventricular glands and under the koilin layer causing inflammation, which can be diphtheric. Red worms may be visible on necropsy. *L. douglassii* (4–6 mm in length) is found on the mucosal surface, below the koilin layer. *L. dentatus* (6–12 mm in length) is most frequently within the koilin layer, but can also be found underneath it. Strongylid-type eggs are passed in feces. Wasting, anorexia, gastric stasis, proventricular impaction, fermentation of proventricular contents, and anemia may result. Mortality rates in chicks can be over 50%. In North and South America combined infections with *L. dentatus* occur, thus attribution of relative pathogenicity is impossible and many reports of disease cause by *L. douglassii* may in fact reflect mixed infections. Morphologic features of adults or infective larvae, and sequences of ITS1 and ITS2 regions can be used for separation of *Libyostrongylus* spp. from each other as well as from *C. struthionis*. |
| *Libyostrongylus magnus* (Trichostrongyloidea) | Proventriculus and gizzard | Ostrich (identified in specimens from Ethiopia and possibly Sudan) | Unlikely to be widely distributed as not identified in recent literature. Pathogenicity not known. |
| *Sicarius uncinipenis* (Spiruroidea) | Proventriculus and ventriculus | Common rhea (Brazil) | Reddish hematophagous spirurid nematodes 18–30 mm in length located primarily below and within the koilin layer, may also be found in proventricular glands and proximal duodenum. Ulceration and detachment of koilin, ventricular mucosal thickening, congestion, hemorrhagic foci, and nematode migration tracts are present. On histologic examination, mild to moderate lymphocytic, histiocytic and heterophilic infiltrate in the mucosa, particularly surrounding parasites or free eggs. Increased parasite numbers and severity of lesions occurs in high density captive situations. |
| *Sicarius waltoni* (Spiruroidea) | Venticulus | Common rhea (Brazil) | Spirurid, 20–25 mm length. |
| *Spurura (Vaznema) zschokkei* (Spiruroidea) | Proventriculus | Common rhea | Spirurid, 16–25 mm in length. Present in the submucosa. |
| *Odontospirura cetiopenis* (Spiruroidea) | Proventriculus and ventriculus | Rhea (South America) | Spirurid, 15–23 mm in length. |
| **Intestinal Nematodes** | | | |
| *Delectrocephalus dimidiatus* | Distal small and proximal large intestines (*D. dimidiatus*, *D. minor*); cecum, large intestine, rectum (*D. minor*); large intestine and rectum (*D. cesarpintoi*) | *Rheea americana* (*D. dimidiatus*—South America, North America, UK; *D. cesarpintoi*—South America, *P. minor*—South America), *Rheea pennata* (*D. dimidiatus*—USA, *P. minor*—South America) | Most reports concern *D. dimidiatus*, other species are likely similar in behavior. Filiform white or pale yellow worms 11–24 mm in length. Blood feeder resulting in anemia, weakness, diarrhea in chicks; can cause high mortality in heavy infestations. Hemorrhage may be present in mucosa. Strongyle-type eggs. Both *D. minor* and *D. dimidiatus* are also described from free-living birds. |

(Continued)
Table e6. Select Helminth Infections and Disease in Large Ratites (Cont.)

| Parasite                          | Location in Host | Species Affected and Geographic Location | Disease Characteristics |
|-----------------------------------|------------------|------------------------------------------|-------------------------|
| Codistomum struthionis (Strongyloidea) | Cecum, large intestine | Ostrich (likely southern African origin and widespread distribution with animal movement) | Family Strongylidae. Large nematode of the cecum, found primarily in the distal third, 13–17 mm in length. Considered by some to be apathogenic but has been associated with mucosal edema and thickening, nodules filled with “purulent mucus,” petechiae and small ulcers. On histology, villar atrophy, cryptal epithelial flattening and squamous metaplasia, and increased mononuclear cells in the submucosa may be present. Pathogenicity is not well determined; anemia or impaired growth may result. Morphologic keys are available to distinguish from the trichostrongylid nematodes. Eggs are indistinguishable from those of Libyostongylus spp. |
| Ascaridia orthocerca, Ascaridia sp. | Small intestine | Common rhea (Brazil, USA) | Worms 30–40 mm length. |
| Dromaeostrongylus bicuspid (Strongyloidea) | Small intestine | Emu Australia, Russia, (USA) | Moderate load of Dromaeostrongylus sp. was seen as an incidental finding in a farmed emu in Australia. Also seen in an emu in the Moscow zoo. Eggs similar to those of Trichostrongylus tenuis. Worms 6–8 mm in length. |
| Trichostrongylus tenuis (Trichostrongyloidea) | Cecum | Emu (USA) | Incidental finding, worms 8–10 mm in length. Diagnosis was made by feeding eggs to chickens and identifying adult worms in their intestines. |
| Tracheal/pulmonary nematodes | | | |
| Cyathostoma variegatum, Cyathostoma sp. (Strongyloidea) | Trachea and bronchi | Emu (USA) | Infection can be subclinical or result in respiratory disease; reported from young and juvenile birds. Infection assumed to result from exposure to waterfowl. |
| Syngamus trachea (Strongyloidea) | Trachea | Emu, rhea, ostrich (USA); emu (Germany) | Especially problematic in young birds, clinical signs include gasping, head shaking, and expulsion of froth and blood from trachea. |
| Filarial Nematodes (rare) | | | |
| Paronchocerca struthionis (Filarioidea) | Lungs, pulmonary arteries | Ostrich (West Africa) | Single description from nematode specimens, adults 31–45 mm in length. Microfilaria 100–123 mm in length. |
| Dicheilonema spicularia (Filarioidea) | Peritoneal cavity, air sacs, retroperitoneal | Ostrich (Southern Africa) | Adult worms not described, microfilaria not identified from blood. |
| Struthiofilaria megaloccephala | Peritoneal cavity, air sacs | Ostrich (Japan—imported) | Identified as an incidental finding in the body cavity of an adult ostrich in Japan that had been imported from South Africa. Numerous worms were present, and ranged from 35 to 75.5 mm in length. |
| Versternema struthionis | Peritoneal cavity, air sacs | Ostrich (Southern Africa) | Adult worms 32.5–55 mm in length, microfilaria not identified from blood. |
| Dicheilonema rheae (Filarioidea) | Peritoneal cavity, air sacs, between fascial planes including legs | Rhea (Tadzikstan, Ukraine), common rhea (Brazil, USA, Uruguay) | Worms can be 65 cm or more in length. Mortality reported but large numbers can be present in healthy birds. |
| Contortospiculum rhea | Air sacs, abdominal cavity, subserosal surfaces | Rhea | Worms can be 80 cm in length. Mortality can occur with massive infestations; seen in birds newly imported into German zoos. |
| Abberant Nematode Migration | | | |
| Chandlerella quiscali (Filarioidea) | Spinal cord and brain | Emu (southeastern USA) | Parasite migration through the brain and spinal cord results in severe neurological signs and high mortality in chicks. Disease outbreaks associated with increased presence of vector Culicoides crepuscularis (and possibly others). Passerine birds are the usual definitive hosts. |
| Baylisascaris spp. (Ascaridoidea)<sup>4</sup> | Brain > spinal cord | Emu, ostrich > rhea (North America) | Migration of *B. procyonis* (natural host, raccoon) or, less likely, *B. columnaris* (natural host, skunk) larvae resulting in severe neurological signs. Nonsuppurative meningoencephalitis characterized by lymphoplasmacytic perivascular cuffing, myelin degeneration, foci malacia (parasitic tracts). Cerebellar white matter often particularly affected. Definitive diagnosis requires identification of nematode larvae in section but they are often not found, and are not necessarily adjacent to lesions. |
| --- | --- | --- | --- |
| **Cestodes** |  |  |  |
| *Houttuynia struthionis* (Davaineidae)<sup>3</sup><sup>4</sup> | Small intestine | Ostrich (South Africa, has radiated with species), common rhea (Brazil, free ranging) | Large, white, flat, segmented cestode, 30 cm–1 m in length. Can cause ill-thrift, mild diarrhea, enteritis, and possibly intestinal obstruction in ostrich chicks. |
| *Houttuynia struthionis* var neogaeae<sup>3</sup><sup>4</sup> | Intestine | Rhea (South America) | Described from a single historical sample. |
| *Cittotaenia rhea*<sup>3</sup> | Intestine | Rhea, (Brazil) | 5–9 cm in length |
| **Intestine** |  |  |  |
| *Cotugnia collini*<sup>3</sup> |  | Emu (Australia) | 5–7 cm in length |
| *Raillietina australis*, *Raillietina beveridgei*, *Raillietina chiltoni*, *Raillietina dromaius*, *Raillietina mitchelli*<sup>3</sup><sup>4</sup><sup>5</sup><sup>6</sup> | Small intestine | Emu (Australia) | *R. australis* - 40 cm in length, *R. beveridgei* - up to 600 cm (relaxed), *R. chiltoni* - up to 90 cm (relaxed), *R. dromaius* - up to 200 cm (relaxed), *R. mitchelli* - up to 120 cm (relaxed). Units of cm are assumed, not specified in source. Prevalence varies regionally with very high burdens possible. No identified effect on host. |
| *Raillietina casuarii*, *Raillietina infrequens*, *Raillietina geraldscmidti*<sup>3</sup><sup>4</sup><sup>5</sup><sup>6</sup> | Intestine | Southern cassowary (R. casuarii, R. infrequens - Australia, New Guinea, R. geraldscmidti - Australia); Dwarf cassowary (R. casuarii, R. infrequens - New Guinea) | *R. casuarii* - up to 35 cm in length, *R. infrequens* - up to 8 cm in length, R. geraldscmidti - up to 4 cm in length. |
| *Chapmania tauricollis*<sup>3</sup> |  | Rhea (Brazil) | No additional description provided. |
| **Trematodes** |  |  |  |
| *Philophthalmus gralli* (oriental eye fluke), *Philophthalmus aweerensis* (Philophthmidae)<sup>3</sup><sup>4</sup><sup>5</sup><sup>6</sup> | Conjunctival sac | Ostrich (P. gralli; Brazil, USA, Zimbabwe), rhea (P. aweerensis; UAE) | Ratites are an unusual host. Ingested metacercaria excyst, migrate up the esophagus to the pharynx or directly enter the nasal passages and lacrimal ducts where they develop. Adults attach to the outside of the nictitating membrane, resulting in severe conjunctivitis and secondary loss of body condition. Blood tinged fluid, miracidia, and parasite eggs will be present. A freshwater snail is the intermediate host. |
| *Fasciola hepatica* (presumptive)<sup>3</sup> | Liver | Emu (Australia) | Discovered incidentally in two emus on pasture in an area where the parasite was endemic. Gross and histologic lesions were consistent with subacute to chronic fascioliasis, and included linear hemorrhagic and necrotic tracks, nonsuppurative portal hepatitis and fibrosis with cholangiolar hyperplasia, and the presence of dense brown/black pigment. Parasites and operculated eggs were seen in bile ducts on one animal. Three of 10 other emus in the flock passed trematode eggs resembling those of *Fasciola hepatica*. |
| **Acanthocephala** |  |  |  |
| *Prosthiorhynchus rhea*<sup>3</sup> | Intestine | Rhea (South America) | Life cycle unknown, may cause local inflammation and possibly peritonitis. |
| *Echinorhynchus reticulatus*<sup>3</sup> | Intestine | Rhea (South America) | Adult worms 1–1.7 cm in length, from a single sample. |

(Continued)
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### Table e7. Select Intestinal Coccidia of Large Ratites

| Ratite Group | Coccidial Entity | Comments |
|--------------|------------------|----------|
| Ostrich<sup>a,b,c</sup> | *Isospora struthionis*, *Isospora* sp., enteric “coccidia” | *I. struthionis* n. sp. was identified from coccidial oocysts (sporulated—2 sporocysts/oocyst, each with four sporozoites) in the feces of an ostrich from a zoo in Turkmenistan. Sotiraki et al. (2001) later identified an *Eimeria* sp. coccidia (sporulated—4 sporocysts/oocyst) in the feces of ostriches in Greece. Coccidia are commonly described as being present in ostrich feces, and their presence was specifically evaluated by Mushi et al. (1998) in ostrich chicks in Botswana, but descriptions of organisms in histologic sections of intestine and substantiated evidence of clinical disease have not been presented. |
| Emu<sup>d</sup> | *Isospora dromaii*, enteric “coccidia” | *I. dromaii* n. sp. was described based on morphologic criteria, including observation of sporulated oocysts, from fecal samples from farmed emu in Brazil. Coccidial oocysts have been identified in the feces of emus less frequently than in ostriches. |
| Common rhea<sup>e</sup> | *Isospora* sp., enteric “coccidia” | Coccidial oocysts are infrequently identified in the feces of the common rhea. In one report these were further characterized as *Isospora* sp., but no descriptive data was provided. |

<sup>a</sup>Yakimoff, W.L., 1940. *Isospora struthionis* n. sp., coccidie de l’autruche africaine. Annales de la Société Belge de Médecine Tropicale, 20, 137–138.

<sup>b</sup>Mushi, E.Z., Isa, J.F.W., Chabo, R.G., Binta, M.G., 1998. Coccidia oocysts in the faeces of farmed ostrich (*Struthio camelus*) chicks in Botswana. Onderstepoort J. Vet. Res. 65(4), 281–284.

<sup>c</sup>Sotiraki, S.T., Georgiades, G., Antoniadou-Sotiriadou, K., Himonas, C.A., 2001. Gastrointestinal parasites in ostriches (*Struthio camelus*). Vet. Rec. 148(3), 84–86.

<sup>d</sup>dos Santos Teixeira, C., Gallo, S.S.M., Ederli, N.B., Berto, B.P., de Oliveira, F.C.R., 2014. *Isospora dromaii* n. sp. (Apicomplexa, Eimeriidae) isolated from emus, *Dromaius novaehollandiae* (Casuariiformes, Casuariidae). Parasitol. Res. 113(11), 3953–3955.

<sup>e</sup>Faust, B.S., Pappas, P.W., 1977. A survey of coccidia and helminth parasites of birds at the Columbus (Ohio) zoo. J. Zoo Anim. Med. 8(1), 18–23.
| Parasite Group                     | Host               | Species of Parasite                                      | Comment*                                                                 |
|-----------------------------------|--------------------|----------------------------------------------------------|--------------------------------------------------------------------------|
| Mallophaga (chewing lice)         | Ostrich            | Struthiolipeurus struthionis<sup>a,b</sup>               | Common. Feeds on the body scales and feather debris of ostriches and causes irritation and excessive grooming resulting in feather damage and loss. Adult lice are 3–4 mm long and readily visible, and eggs (nits) can be found on the underside of feathers attached to the barbs near the shaft. |
|                                   | Ostrich            | Struthiolipeurus rheae<sup>c</sup>                      | Identified from ostriches in Brazil.                                     |
|                                   | Ostrich            | Struthiolipeurus nandu<sup>a,d</sup>                    | Identified from an ostrich in a Spanish zoo.                             |
|                                   | Ostrich            | Struthiolipeurus rheae<sup>e</sup>                      | Heavy infestation can result in pruritis and feather damage or loss.    |
|                                   | Common rhea        | Struthiolipeurus nandu, Struthiolipeurus rheae, Struthiolipeurus renshi, Struthiolipeurus stresemanni, Meinertzhageniella (Lipeurus) lat<sup>a,e,i,b</sup> | Heavy infestation can result in pruritis and feather damage or loss.    |
|                                   | Lesser rhea        | Struthiolipeurus andinus, Meinertzhageniella schubarti<sup>a</sup> | Disease not described.                                                  |
|                                   | Emu                | Dahlembornia asymmetrica<sup>a</sup>                    | Disease not described.                                                  |
| Acari (feather mites)             | Ostrich            | Gabucinia sculptura (Struthiopterolichus sculpturatus), Gabucinia abbreviata<sup>a,c</sup> | Heavy infestation can result in feather damage.                         |
|                                   | Ostrich            | Gabucinia nouvel<sup>b</sup>                            | Identified from an ostrich in a French zoo that had been imported from central Africa, unusually, lesions were present on the skin rather than on the feathers. |
|                                   | Ostrich, common rhea | Gabucinia bicaudata (Struthiopterolichus bicaudatus<sup>b,h,e</sup>) | Heavy infestation can result in pruritis and feather damage or loss.    |
|                                   | Ostrich, common rhea | Paralges (Dermoglyphus) pachynemis<sup>e</sup>         | Disease not described.                                                  |
|                                   | Southern cassowary | Hexacaudalges casuariculus<sup>f</sup>                  | Single identification from a wild cassowary in Australia.               |

<sup>*Many reports simply identify the presence of the above ectoparasites, often in the absence of clinical signs or pathologic changes. It is supposed that pruritis and feather loss and damage can occur with sufficient intensity of infection of any of the above species.</sup>

<sup>a</sup>Huchzermeyer, F.W., 1998. Diseases of Ostriches and other Rattis. Agricultural Research Council, Onderstepoort Veterinary Institute, Onderstepoort, Republic of South Africa.

<sup>b</sup>Mohammed, B.R., Malang, S.K., 2015. Common ectoparasites of ostrich (Struthio camelus) and their control - A review. Research J. Anim. Vet. Fish. Sci. 3(10), 23–29.

<sup>c</sup>Pesenti, T.C., da Silva, D.S., Bertacco, L.L., Brum, J.G.W., Müller, G., 2009. Record of Struthiopterolichus sculpturatus and Struthiolipeurus rheae in Struthio camelus in the Rio Grande do Sul State, Brazil. Ciência Rural, 39(8), 2546–2549.

<sup>d</sup>Dominguez de Tena, M., Hernández Rodríguez, S., Bercera Martell, C., Calero Carretero, R., Moreno Montañez, T., Martínez Gómez, F., 1976. Struthiolipeurus nandu 1950 (Mallophaga: Philopteridae) parásito del avestruz (Struthio camelus) en el parque zoológico de Córdoba. Revista iberica de parasitología (36), 167–173.

<sup>e</sup>Ponce Gordo, F., Herrera, S., Castro, A.T., García Durán, B., Martínez Díaz, R.A., 2002. Parasites from farmed ostriches (Struthio camelus) and rheas (Rhea americana) in Europe. Vet. Parasitol. 107(1–2), 137–160.

<sup>f</sup>Weisbroth, S. H., Seelig, A. W., 1974. Struthiolipeurus rheae (Mallophaga: Philopteridae), an ectoparasite of the common rhea (Rhea americana). J. Parasitol. 60(5), 892–894.

<sup>g</sup>Sinkoc, A.L., Muller, G., Brum, J.G.W., Soares, M.P., Oliveira, L.T. and Gonçalves, I.P.D., 2005. Ocorrência de Struthiolipeurus rheae (Phthiraptera: Ischnocera, Philopteridae) em Rhea americana (Rheiformes: Rheidae) no Brasil. Arquivos do Instituto Biológico, 72(4), 535–538.

<sup>h</sup>André, M., 1960. Sarcoptides plumicoles parasites des autruches. Acarologia, 2, 556–567.

<sup>i</sup>Mironov, S.V., Proctor, H.C., 2005. A new feather mite genus of the family Psoroptoidea (Acari: Analgoidea) from cassowaries. J. Nat. Hist. 39(37), 3297–3304.
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Palaeognathae: Apterygiformes, Casuariiformes, Rheiformes, Struthioniformes; Tinamiformes

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