Diseases of farmed crocodiles and ostriches

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Summary

Crocodiles and ostriches are very sensitive to stress, and the ideal conditions for intensive rearing have not yet been established. Consequently, mortality is often directly linked to conditions on the farm. Crocodile and caiman pox, adenoviral hepatitis, mycoplasmosis, chlamydiosis and coccidiosis are crocodile-specific infections with reservoirs in wild populations and adult wild-caught breeding stock. Other important conditions are salmonellosis, non-specific septicaemia, trichinellosis, the nutritional diseases osteomalacia, fat necrosis and gout, as well as winter sores.

The only ostrich-specific transmissible disease is libyostrongylosis. Other important conditions are Newcastle disease, avian influenza, fading chick syndrome, tibiotarsal rotation and enteritis. No cases of coccidiosis in ostriches have ever been confirmed.

Keywords

Alligators – Animal husbandry – Caiman – Crocodiles – Diseases – Libyostrongylosis – Ostriches – Wildlife.

Introduction

Commercial ostrich farming began approximately 150 years ago, initially for the feathers only, much later for the leather as well and only relatively recently for the meat. Recent years have seen a world-wide expansion of ostrich production. As a result of a long history of domestication, farmed ostriches are classified as domestic stock in South Africa. In comparison, the commercial production of crocodiles for skins and meat originated much more recently. If based on the collection of eggs or hatchlings from the wild, with the release of a certain percentage of grown stock back into the wild, the production is referred to as 'ranching', whereas the production from captive breeding stock is termed 'farming'. This distinction does not apply to the title or text of this paper, and in this paper the term 'crocodiles' includes all crocodilian species, including caimans and alligators.

There are no ostrich-specific infectious or contagious diseases, the only specific pathogen being *Libyostrongylus*, the wireworm. A few crocodile-specific infectious agents exist, but none of these could be regarded as a primary pathogen. However, ostriches and crocodiles share an extreme sensitivity to stress, which under the widely practised intensive rearing conditions can trigger disastrous outbreaks of disease and mortality.

Crocodiles

Thermoregulation and stress

Although poikilotherms, crocodiles like to maintain body temperature within a narrow range of 28°C-33°C by using the thermogradient in their natural environment consisting of sunshine and shade, warm surface and cold deep water, as well as burrows. Inability to thermoregulate or exposure to low or fluctuating temperatures and particularly to overheating are common sources of stress in farming situations. For these tropical animals, body temperatures above 36°C are lethal, while lowered body temperatures very severely reduce the activity of the immune system.

Hatchlings and juvenile crocodiles also fear exposure to the open sky; even indoors, the animals like to hide, and for this purpose hide boards should be provided (Fig. 1). Inability to hide, handling, capture and any sudden noise or movement in the environment are further common sources of stress.

Sources and reservoirs of infection

Crocodile-specific infectious agents are carried and shed by wild crocodiles and can contaminate crocodile inhabited waters, such as rivers or lakes, from which water often is drawn is for use on farms. Wild-caught captive breeding stock often also carry these infectious agents and can act as a source for annual disease outbreaks for many years.

Non-specific pathogens such as salmonellae and mycobacteria can be introduced into the rearing unit through meat from poultry, pigs or cattle which have died on the farm, as such meat is commonly used for feeding crocodiles. Flies, rats and other animals attracted to the scraps also are frequent carriers of crocodile-non-specific infectious agents. Given the high population densities in intensive rearing systems, the frequent cleaning and disinfection of pens is of utmost importance (Fig. 2).

Crocodile pox

Crocodile pox is caused by a parapoxvirus (27) which infects hatchling and juvenile crocodiles and produces brown crusty lesions in the oral cavity (Fig. 3), on the head and on the ventral and lateral surfaces of body and tail (Fig. 4). Outbreaks have been reported from farmed Nile crocodiles (*Crocodylus niloticus*) (15, 22, 31, 37, 61) and individual cases have been noted in *Crocodylus porosus* and *C. johnsonii* from Australia (11, 14).

Outbreaks of crocodile pox occur in hatchling and juvenile Nile crocodiles. Lesions on the eyelids may cause blindness, and lesions on the head may cause shrinking of the skin, leading to deformities. Intracytoplasmic Bollinger and Borrel inclusion bodies may be found on histopathological examination of the lesions (Fig. 5). Although morbidity is high, mortality usually remains low, as long as the disease does not become complicated by opportunistic bacterial and fungal infections. Spontaneous recovery normally occurs within a few weeks or months.

No specific treatment is recommended and no vaccine is available. Prevention is based on hygiene, particularly on avoiding the use of surface water in the rearing units.

Caiman pox

Caiman pox is caused by a parapoxvirus (27) which infects hatchling and juvenile spectacled caimans (*Caiman crocodilus*), producing grey or greyish-white lesions in the mouth and on the dorsal skin of head, body and legs (Fig. 6). The colour and distribution of the lesions, in addition to the apparent limitation of the disease to a single species indicate that this is a separate disease entity. Outbreaks have been reported from the United States of America (USA) (44), South Africa (62), Brazil (20, 53, 54) and Colombia (67).

During an outbreak, a large proportion of the animals in a rearing unit may be affected. Under good rearing conditions, recovery is spontaneous but may take six weeks or longer. No specific treatment is available; prevention is based on strict hygiene and rearing in a stress-free environment.

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Adenoviral hepatitis

Adenoviral hepatitis has been reported from Nile crocodiles under five months of age in Zimbabwe (22, 45). The virus has also been found in South Africa (40). The intestines, pancreas and lungs may occasionally be affected as well as the liver. Some outbreaks cause high mortality, particularly during the winter months, while a chronic adenoviral hepatitis is seen as a major cause of runting (24). The clinical signs are lethargy and anorexia. Post-mortem findings may include a slight icterus, a markedly swollen and pale liver (Fig. 7) and pale yellow bile (23). The diagnosis is confirmed by the histopathological demonstration of the typical intranuclear inclusion bodies in the hepatocytes.

No treatment or vaccine is available. Prevention should be based on avoiding horizontal spread of virus via contaminated water, as well as avoiding stress, particularly thermal stress caused by fluctuating temperatures in open-air pens in winter.

Mycoplasmosis

Polyarthritis and pneumonia due to *Mycoplasma crocodyli* infection have occurred in one- to three-year-old crocodiles on several farms in Zimbabwe (48, 56), and a *Mycoplasma* sp. was isolated from lungs and synovial fluid of adult American alligators (*Alligator mississippiensis*) (18). Mycoplasma were also found by electron microscopy in the faeces of farmed Nile crocodiles in South Africa (Fig. 8) (40).

Affected animals have swollen joints and are unable to move. Both vertical and horizontal transmission are suspected. Severe or repeated stress may precipitate the manifestation of the disease. Treatment of cases with tetracycline by injection and in the feed led to a reduction in clinical signs, but did not prevent relapses (57). A vaccine produced using *M. crocodyli* gave a certain degree of protection (57).

Chlamydiosis

Two forms of chlamydiosis occur in farmed Nile crocodiles, usually under one year of age, namely: an acute hepatitis (found in Zimbabwe, often together with adenoviral hepatitis) and a chronic bilateral conjunctivitis (Fig. 9) (24, 39). While closely related to *Chlamydia psittaci*, the agent could be a distinct crocodile-specific species. The mode of transmission has not yet been elucidated, but contamination of surface water by wild carrier crocodiles is suspected.

In outbreaks of the acute form, the hatchlings die without showing any clinical signs. The liver is enlarged, pale and mottled and the spleen slightly enlarged. Mild ascites and severe hydropericardium are present. The diagnosis is based on the demonstration of the agent either microscopically (numerous colonies of intracytoplasmic organisms in the hepatocytes) (Fig. 10) or by culture. In the ocular form, an accumulation of fibrin may occur under the third eyelid, causing blindness. In these cases, the diagnosis can be confirmed by culture only, as colonies of the organism are rarely seen in histopathological preparations of conjunctival tissue.



Fig. 1 Hide boards in a crocodile rearing unit



Fig. 2

Frequent and thorough cleaning of the crocodile rearing pens is of the utmost importance



Fig. 3 Crusty pox lesions on the palate of a Nile crocodile hatchling



Fig. 4 Pox lesions on the ventral skin of a Nile crocodile hatchling



Fig. 5

Intracytoplasmatic inclusion bodies in a crocodile pox lesion



White crusty lesions of caiman pox on the back of a spectacled caiman hatchling



Fig. 7 Swollen and pale liver in a Nile crocodile hatchling with adenoviral hepatitis



Fig. 8 Transmission electromicrogram of a mycoplasm in negativelystained faeces of a Nile crocodile



Fig. 9

Juvenile Nile crocodile with chlamydial keratoconjunctivitis



Fig. 10 Intracytoplasmatic chlamydial colonies in the hepatocytes of a Nile crocodile hatchling

Both forms respond to treatment with tetracycline. Prophylaxis depends on strict hygiene measures.

Dermatophilosis

A *Dermatophilus* sp. was isolated from skin lesions referred to as brown spot' in farmed American alligators in Louisiana (10). However, attempts to isolate the agent from similar 'brown spot' lesions in Florida were unsuccessful (6). In Australia, a *Dermatophilus* sp. was also isolated in farmed crocodiles and the disease was transmitted by the means of broth cultures (12, 13, 14). The lesions begin as small spots of discoloration between the scales of the abdominal skin, and progress to erosion of the epidermis and ulceration. The diagnosis is based on the histopathological demonstration of the filamentous organisms and their culture. No treatment is available.

A similar condition, winter sores, occurs in crocodiles kept at suboptimal conditions in winter (see below).

Mycobacteriosis

Mycobacteriosis in farmed crocodiles is caused by environmental mycobacteria and facultative pathogens. The specific pathogens *Mycobacterium tuberculosis* and *M. bovis* are unlikely to be able to infect crocodiles because of the specific temperature requirements of these pathogens (42). Generalised granulomatous lesions were caused in *C. johnsonii* by *M. ulcerans* (5) and in *C. niloticus* by *M. avium* of porcine origin (42). A granulomatous dermatitis caused by an unidentified mycobacterium has been described from *C. porosus* (14).

Not all granulomatous lesions are caused by mycobacteria. For a diagnosis, the presence of mycobacteria in the tissues must be demonstrated microscopically or by culture in a specialised laboratory. Note that these mycobacteria do not grow on the media and at the temperatures used in medical laboratories. No treatment is available. Prevention is based on strict hygiene measures and avoiding the use of raw meat from suspect sources.

Salmonellosis

Salmonellosis is caused by many different serovars of *Salmonella* (65) and manifests itself either as enteritis, particularly in hatchlings, or as septicaemia. The enteritis usually is exudative, exudation being a characteristic feature of reptilian inflammation (41), and leads to intestinal occlusion and the slow death of the affected hatchlings. A haemorrhagic enteritis due to an infection with S. Choleraesuis has also been described (60). Septicaemia often is precipitated by severe stress and usually associated with anorexia. The causative bacteria are frequently introduced through meat fed to the crocodiles (obtained from animals that have died on the farm).

Diagnosis is based on the isolation and identification of the causative agent and a possible treatment on the antibiogram. However, in cases of intestinal occlusion and anorexia, the oral administration of an antibiotic will not be possible. In such cases, a flock (or herd) treatment would be of prophylactic value, protecting those animals in the group which are not yet in an advanced stage of the disease. Prevention is by the application of strict hygienic measures, such as the boiling of meat to be fed or feeding dry pelleted feeds. The reported use of a calf paratyphoid vaccine in an outbreak probably was of limited value (43). The aspect of possible contamination of crocodile meat for human consumption must also be considered (34, 55).

Non-specific septicaemia

Crocodiles are very sensitive to stress. Under conditions of severe stress, intestinal bacteria appear to be able to penetrate the mucosal barrier of the intestine and cause septicaemia in a manner similar to shock septicaemia in human trauma and burn patients (21). If the stressful event is repeated or associated with low temperatures (e.g. transport in winter), the immune system may not be able to eliminate the bacteria, and fatal disease may develop. In some cases, the bacteria become localised in the joints, causing a polyarthritis (36).

Initially, the affected animals are depressed, and later with the developing arthritis, become unable to move. Since the gut

translocation is a matter of chance, different animals within an affected group may be infected with different species of bacteria. In the early stages, an antibacterial treatment may be successful. Avoidance of stressful procedures at any time, but particularly during winter is one of the most important preventive measures.

Septicaemia caused by stressful preslaughter procedures (stress septicaemia) may seriously affect the quality of crocodile meat through *in vivo* contamination.

Coccidiosis

Although several coccidian species from crocodiles have been described (3, 51), those associated with outbreaks of coccidiosis have yet to be identified. It has been suggested that the organisms responsible may belong to the genus *Goussia* (26). The oocysts of the pathogenic coccidians are very fragile and usually only the sporocysts are found, often trapped in the mucosal crypts by exudate and also transported by lymph and blood to other organs and causing cases of generalised coccidiosis, which have been reported in Nile crocodiles (22, 59) in addition to *C. porosus* and *C. novaeguineae* (49, 50), including cases of transovarian transmission in spectacled caimans (67). The intra-intestinal sporulation is typical of this type of crocodilian coccidiosis. Generally, wild crocodiles in the vicinity of the farm as well as carriers among the breeding stock act as reservoirs for the infection.

Animals affected by coccidiosis become listless and may take a long time to die. On post-mortem examination, a fibrinous enteritis is usually seen, often occluding the intestine. Given the fragility of the oocysts, the infection cannot be diagnosed by means of faecal analyses or intestinal smears, but only by histopathological examination (Fig. 11). The most effective treatment consists of mixing sulphachlorpyrazine into the ration. Avoiding the use of surface water and similar hygiene measures are the basis of a prophylactic programme.



Fig. 11 Coccidial sporocysts in the crypt epithelium of a juvenile Nile crocodile

Trichinellosis

Larvae of a *Trichinella* sp. have been found in the muscles of farmed Nile crocodiles in Zimbabwe. These larvae were found to be infective to rats as well as to pigs (25, 58). However, in another study, *Caiman sclerops* was found to be refractory to experimental infection with a number of *Trichinella* spp. from different sources (47).

Nutritional diseases

Lack of bones in the meat diet causes crocodile hatchlings to develop osteomalacia with kyphoscoliosis of the vertebral column (Fig. 12), soft and flexible jaws ('rubber jaws') and diaphanous ('glassy') teeth (32). The affected animals are unable to move on land, but can still swim. Addition of bone meal or calcium diphosphate to the ration rapidly rectifies the deficiency, but does not straighten the vertebral column.

The lack of sufficiently high levels of vitamin E in crocodiles that are fed fish, particularly if the fish is not very fresh, can lead to fat necrosis and pansteatitis (Fig. 13) (24). This can be prevented by the antioxidant activity of vitamin E. The necrotic fat hardens and the hardened intermuscular fat in the tail reduces the motility of the affected crocodiles, which slowly waste away. The hardened fat in the tail can be clinically palpated in the live crocodile. No treatment is available.



Juvenile Nile crocodile with kyphoscoliosis caused by osteomalacia



Cross section of the tail of a Nile crocodile with pansteatitis Note the yellow, hardened fat between the long tail muscles

Gout commonly occurs when crocodiles in outside ponds are fed during winter. While the animals are still able to digest the food, they can no longer utilise the absorbed protein, and the kidneys are unable to excrete the excess uric acid, which is deposited in the joints (Fig. 14), rendering the affected crocodiles unable to move. With less frequent feeding in winter, the proportion of uric acid increases against ammonia produced by the kidneys (29), further aggravating the situation. Vitamin A deficiency may also play a role in this condition (4).



Fig. 14 Massive gout deposits in the knee and tarsal joints of a juvenile Nile crocodile

Winter sores

Under cold conditions, the blood supply to the skin is reduced and as the action of the immune system is largely impeded by the cold, small skin scratches do not heal and bacterial and fungal infections from the soiled environment persist, causing small erosions between scales which are covered with yellow or brown crusts (Fig. 15). These lesions are called 'winter sores' because of their prevalence in winter (33). Faecal bacteria and fungi are usually isolated from these lesions, which do not respond easily to antibacterial or antifungal treatment, particularly if cold conditions persist.



Fig. 15 'Winter sores' on the ventral tail surface of a juvenile Nile crocodile

Winter sores cause persisting damage and severely affect the grading of the skins. Therefore, if crocodiles must be kept outside during winter, it is important to keep the animals at low stocking rates, and to take particular care of the regular cleaning and disinfection of the rearing pens.

Ostriches

Newcastle disease

Three important features distinguish Newcastle disease in ostriches from that in poultry. Firstly, there is no respiratory involvement and therefore no airborne transmission. Secondly, the only symptoms observed are the typical nervous symptoms, an inability to control head and neck posture and movements (Fig. 16). Thirdly, no pathognomonic macroscopic or histopathological lesions are present (35).



Fig. 16 Ostrich chick with Newcastle disease unable to lift its head off the ground

As a result of the lack of airborne transmission, only one or a few birds are clinically ill at any one time during an outbreak and the infection does not even cross a wire partition between groups. The haemagglutination inhibition test is not a reliable diagnostic tool, as both false negatives and false positives can be produced (1). However, the enzyme-linked immunosorbent assay (ELISA) gives reliable results (16, 69). Vaccination with LaSota eye drop followed by subcutaneous injection with a killed emulsified vaccine provides reliable protection.

When the survivors of experimentally-infected vaccinated and non-vaccinated slaughter ostriches were slaughtered two weeks after the last death, the virus could not be re-isolated from organs, meat, bone marrow or the gastrointestinal tract of these birds, indicating the apparent absence of a carrier status (66).

Avian influenza

The avian influenza virus is highly variable and is normally carried and spread by wild birds. The strains differ in pathogenicity to different avian species and are classified according to pathogenicity for poultry. Outbreaks in ostriches do not necessarily affect poultry. Of the recent outbreaks, only that in Italy involved a poultry-pathogenic strain (H7N1) (17). Other strains have been isolated from ostriches, namely: H7N1 (1991, 1992), H5N9 (1994) and H9N2 (1995) in South Africa and H5N2 (1995) in Zimbabwe. None of these strains was poultry pathogenic (52). An apathogenic strain (H5N2) was isolated from ostriches in Denmark (46).

In the case in Italy, clinical signs included green urine, as observed in previous outbreaks (2), as well as nervous signs similar to those seen in Newcastle disease, and haemorrhagic faeces (17). Necrosis of the liver and enteritis are common postmortem findings. No treatment is available and vaccination is not an option because of the variability of the strains. Wild birds attracted to the ostrich feed play a major role in the spread of avian influenza to ostrich flocks, and consequently, unconsumed feed should not be left lying in the troughs for any length of time.

Fading chick syndrome – gastric stasis

Fading chick syndrome embraces a major proportion of all ostrich chick mortality and is a widely discussed but ill-defined and poorly understood phenomenon. Clearly diagnosable conditions such as yolk sac infection and enteritis should not be classed as fading chick syndrome. This syndrome is typically characterised by a halt in growth and loss of weight (fade away) in some chicks, normally between two and six weeks of age, until the chicks die after complete exhaustion of their reserves. Postulated causes include poor nutrition, including parental nutrition, stress, heat and cold, as well as possible infectious agents yet to be identified. A specific complication is megabacterial gastritis (Fig. 17) (38) and fungi can also invade the weakened gastric mucosa.

Post-mortem lesions include ascites, depletion of fat in the coronary groove of the heart and an abnormally soft and folded koilin layer in the gizzard (koilin hypertrophy) (Fig. 18). Often the proventriculus is filled with food, but faecal matter is nearly completely absent from the intestines except for the distal parts of the colon. The soft and folded consistency of the koilin layer of the gizzard is interpreted as sign of complete stoppage of gastric contractions, i.e. gastric stasis (35). The lack of gastric contractions prevents the transport of food from the proventriculus into the intestine. Consequently, the bird starves to death, despite having a full stomach.

No guaranteed treatment is available, but dosing the birds with nutritious liquids, including vegetable oil, and stimulating gastric contractions by injections of metachlopramide (0.1 ml/kg live mass) may be helpful (63). Prevention must to be based on the exclusion of possible causative factors such as malnutrition, stress, cold, etc.



Fig. 17 Megabacteria in the koilin layer of an ostrich gizzard close to the epithelium of the superficial glands



Fig. 18 Koilin hypertrophy in the gizzard of an ostrich chick with gastric stasis

Tibiotarsal rotation

Tibiotarsal rotation is the rotation of the lower tibiotarsus outward along its longitudinal axis; it generally occurs in ostrich chicks at an age of three to twelve weeks (Fig. 19). Prevalence is not related to the sex of the bird, but rotation of the right limb is more prevalent than rotation of the left (8). The rotation occurs rapidly, over a few days, and most of the affected birds are unable to stand or walk. The mechanism involved in this rotation remains unclear and no single distinct cause has been identified. In affected birds, calcium and phosphorus levels in the bones were lower, and serum zinc levels were higher than those of normal birds (9). However, empirically, rotation appears to be related to frequent stumbling of chicks over obstacles such as low feed troughs. Poor nutrition, particularly mineral imbalance, malabsorption due to intestinal infections, genetics and lack of exercise are other causes that have been cited (35).

No successful surgical treatment has been reported. On farms with a high incidence of tibiotarsal rotation, the number of cases can be reduced by lowering the protein content of the



Fig. 19 Ostrich chick with tibiotarsal rotation in the right leg

ration and thereby slowing the rate of growth. Prevention should be based on the following:

– avoiding breeding from birds with a known familial incidence of leg rotation

- providing a well-balanced nutrition to both parents and chicks

- avoiding intestinal infections

- providing an injury-free environment (35).

Enteritis

Enteritis is one of the principal causes of mortality in ostrich chicks which are intensively reared on concrete floors. The problem is directly linked to an inability of the chicks to establish a normal intestinal flora as well as to the oral administration of antibiotics, which upsets the existing intestinal bacterial flora (35). Enteritis practically never occurs in chicks reared on pasture, except for clostridial enteritis which can occur after the sudden change from dry feed to lush pasture, which also upsets intestinal flora. Gram-negative bacteria and clostridia are the causative agents most commonly found in ostrich chick enteritis. Contributing factors are cold and stress (35).

If the enteritis is limited to the small intestine, diarrhoea may be absent. The affected chicks appear listless and dehydrated. The prominent post-mortem feature is a sero-mucous, serofibrinous or haemorrhagic enteritis, sometimes associated with small granulomatous lesions in the liver due to invasion by the causative bacteria (35).

Antibacterial treatment must be based on the results of bacterial isolation and sensitivity tests. The antibiotic of choice should be administered in daily alternation with a live probiotic. The most important preventive measure is to encourage the early establishment of a complete normal intestinal flora by dosing the chicks individually at hatch with live yoghurt, followed by provision of a live probiotic in the drinking water and then either exposure of the chicks to pasture, to clean garden soil (a handful scattered in the run) or to the faeces of domestic herbivore animals, e.g. rabbits, goats or sheep (35). Anticlostridial sheep vaccines can be used to vaccinate ostrich chicks against haemorrhagic enteritis caused by *C. perfringens* (35).

Respiratory disease

Upper respiratory disease is observed in feedlot ostriches under cold and dusty conditions in the form of conjunctivitis, rhinitis and tracheitis. If poultry pathogens such as *Mycoplasma* spp. become involved, the clinical signs may be very severe. Antibacterial treatment is usually ineffective if the detrimental environmental circumstances persist.

Respiratory mycosis of the air sacs is due to a build-up of contamination by fungal spores in the environment, particularly of *Aspergillus* spp. Affected birds become listless, but the air sac lesions produce no particular sound to enable detection by auscultation. Experiments suggest that fungal pneumonia is of enteric origin (68). Respiratory fungal infections can be treated successfully by fumigation or aerosol of the room with enilkonazole, in the presence of the birds (35).

Libyostrongylosis – gastric verminosis

The only true primary pathogen of the ostrich is the stomach worm Libyostrongylus douglasii Cobbolt, 1882, which lives under the koilin layer of gizzard and proventriculus (Fig. 20) as well as in the deep mucosal glands of the proventriculus (19). In severe infestations, the worm causes an intense irritation of the mucosa and a disruption of the koilin layer, exposing the mucosa to the action of gastric secreta and of bacteria and fungi, resulting in a rotten appearance of the inner surface of the stomach, which gave rise to the descriptive Afrikaans term for the disease, vrotmaag. Severe infestations cause gastric stasis (see above), anaemia and a slow death. The eggs closely resemble those of the only other roundworm parasite of the ostrich, Codiostomum struthionis, but can be differentiated in larval cultures (7). The parasite undergoes larval development within the egg in approximately 60 h and under dry conditions, can remain alive in the egg for more than a year (64). Levamisole, fenbendasole and ivermectin are commonly used to treat Libyostrongylus infections (35).



Fig. 20 Libyostrongyles attached to the ventricular mucosa after removal of the koilin layer

Two additional species of *Libyostrongylus* have been described, namely: *L. magnus* Gilbert, 1937, in the Ukraine found in ostriches originating from Ethiopia (28), and *L. dentatus* Hoberg, Lloyd and Omar, 1995, in the United States of America found in ostriches originating from Tanzania (30). However, the role and pathogenicity of these species remain to be determined.

Coccidiosis

No confirmed cases of coccidiosis have ever been reported from ostriches. The case of *Isospora struthionis* described from a zoo ostrich remains doubtful and probably consisted of sporulated oocysts from another avian host passaging passively through the ostrich gut (35, 70). Cases of haemorrhagic enteritis are caused by *Clostridium perfringens* and not by coccidia.

Conclusions

As in all other intensively farmed domestic species, most mortality in crocodiles and ostriches is closely related to rearing conditions. While both are also highly sensitive to stress, species-specific infectious diseases play only a minor role. A thorough knowledge of behavioural and physiological requirements is necessary to devise rearing conditions suitable for the particular species and to be able to diagnose mortality caused by unsuitable conditions. Extrapolations from one species to another, e.g. from poultry to ostriches, can lead to serious errors. Superficial knowledge can be misleading and dangerous. Due to its limited scope, this paper should only be used as starting point to more serious studies.

Maladies des crocodiles et des autruches d'élevage

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Résumé

Les crocodiles et les autruches sont très sensibles aux facteurs de stress et les conditions idéales pour un élevage intensif n'ont pas encore été bien définies. Aussi la mortalité de ces animaux est-elle souvent directement liée aux conditions de leur élevage. La variole des crocodiles et des caïmans, l'hépatite adénovirale, la mycoplasmose, la chlamydiose et la coccidiose sont des infections spécifiques des crocodiles, avec des réservoirs dans les populations sauvages et les reproducteurs adultes capturés dans la nature. Ces animaux souffrent également de salmonellose, de septicémie non spécifique, de trichinellose, de maladies nutritionnelles comme l'ostéomalacie, la stéatonécrose et la goutte, ainsi que de plaies hivernales.

La seule maladie transmissible et spécifique des autruches est la parasitose due à *Libyostrongylus* spp. Les autres affections importantes sont la maladie de Newcastle, la grippe aviaire, le syndrome de dépérissement du poussin, la rotation tibio-tarsienne et l'entérite. Aucun cas de coccidiose n'a jamais été confirmé chez l'autruche.

Mots-clés

Alligators – Autruches – Caïmans – Crocodiles – Élevage – Faune sauvage – Maladies – Parasitose due à Libyostrongylus spp.

Enfermedades de los cocodrilos y las avestruces de granja

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Resumen

El cocodrilo y el avestruz son dos animales muy sensibles al estrés, y todavía se desconocen las condiciones idóneas para su cría intensiva. De ahí que muy a menudo la mortalidad de esos animales guarde relación directa con las condiciones reinantes en la granja. La viruela del cocodrilo y el caimán, la hepatitis adenovírica, la micoplasmosis, la clamidiosis y la coccidiosis son

infecciones específicas del cocodrilo que tienen por reservorio las poblaciones en libertad y los adultos reproductores capturados en estado salvaje. Otras patologías importantes son la salmonelosis, la septicemia inespecífica, la triquinelosis y enfermedades de origen nutricional como la osteomalacia, la necrosis grasa o la gota, así como las úlceras invernales.

La única enfermedad transmisible específica del avestruz es la parasitosis por *Libyostrongylus* spp. Otras afecciones de importancia son la enfermedad de Newcastle, la influenza aviar, el síndrome debilitante del pollito, la rotación tibiotarsal y la enteritis. Nunca se ha confirmado caso alguno de coccidiosis en avestruces.

Palabras clave

Aligatores – Avestruces – Caimanes – Cocodrilos – Enfermedades – Fauna salvaje – Parasitosis por Libyostrongylus spp. – Producción animal.

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