Ostrich diseases

D.J. Verwoerd

Onderstepoort Veterinary Institute, Private Bag X5, 0110 Onderstepoort, Republic of South Africa

The terms describing serovars of *Salmonella enterica* subsp. *enterica* are presented as follows: *Salmonella Enteritidis*, *S. Gallinarum*, *S. Pullorum*, *S. Typhimurium*, etc.

Summary
Scientific knowledge of ostrich diseases is incomplete and very fragmented, with specific details on technical aspects of diagnostic and/or screening tests completely absent in most cases. *Salmonella Typhimurium* is common in multispecies collections and causes mortality in chicks younger than three months on commercial farms, but is rarely found in chicks older than six months, or slaughter birds of twelve to fourteen months in southern Africa. *Campylobacter jejuni* and *Chlamydia psittaci* are occasionally reported, mainly in young ostriches, but both remain a diagnostic challenge. Crimean-Congo haemorrhagic fever is transmitted to domestic animals including ostriches, principally by ticks of the genus *Hyalomma*. In the ostrich, the disease causes no clinical symptoms during a viraemia of approximately four days. Spongiform encephalopathy has not been reliably reported in ostriches, while anthrax has occurred rarely in modern times but was reportedly an important cause of death approximately 100 years ago in South Africa. *Salmonella Gallinarum* and *S. Pullorum* are unknown in ostriches. *Pasteurella multocida* occurs but is easily contained with antibiotics. *Mycoplasma* spp. are regularly found in an upper respiratory disease syndrome complicated by opportunistic bacterial pathogens. Ostriches of all ages are susceptible to challenge by velogenic Newcastle disease virus (NDV), but standard inactivated La Sota poultry vaccines can stimulate protective immunity lasting over six months. The viraemic period in vaccinated slaughter ostriches is between nine and eleven days and there are no indications of a carrier state or presence of the virus in the meat or any other tissues after this period, with peak immunoglobulin G response reached on day fourteen post infection. Haemagglutination inhibition tests are significantly less sensitive and less specific than enzyme-linked immunosorbent assays. Cloacal and choanal swabs used for direct virological screening in clinically affected cases (field and experimental) could not detect NDV. All avian influenza isolates reported from ostriches have been non-pathogenic to poultry, even the H5 and H7 subtypes. Some of the latter have been associated with mortality of ostrich chicks in localised outbreaks during periods of inclement weather and with significant wild bird (waterfowl) contact. Borna disease causes a nervous syndrome in ostrich chicks, but to date, has only been reported in Israel. Eastern and Western equine encephalomyelitides cause fatal disease in ostriches and other ratites, with mortality ranging from less than 20% to over 80% in affected flocks. These diseases are present in North, Central and South America where the associated ornithophilic mosquito vectors occur. Equine and human vaccines are apparently safe and efficacious in ratites. Wesselsbron disease, infectious bursal disease (type 2), adenovirus and coronavirus infections have been reported from ostriches but the significance of these diseases is unclear. Due to the paucity of data regarding ostrich diseases and the unvalidated state of most poultry tests in this unique group of birds, strict observation of a
Introduction

Current scientific knowledge of diseases of ratite birds (ostriches, emus and rheas) is incomplete, fragmented and in most cases superficial or limited to anecdotal reports. The domestic ostrich (Struthio camelus domesticus) is the result of more than 100 years of selective breeding in the arid regions of South Africa for improved reproductive traits (eggs produced per breeding season), feather quality and improved docility, and is the most prominent member of this family in terms of international trade (live ostriches, fertile eggs and ostrich meat, leather and feathers). However, since the 1980s, significant ostrich industries based on indigenous, unselected birds have developed utilising several subspecies in other countries of Africa, namely: Botswana, Namibia and Zimbabwe (different phenotypes of S. c. australis) and Kenya and Tanzania (S. c. massaicus and S. c. molybophanes). Live ostriches and/or fertile eggs are exported from these countries to destinations world-wide, with notable industries established in Israel, southern Europe, North America, the People’s Republic of China, South-East Asia and Australia, while small numbers of ostriches are also established in Arabia, South America and northern Europe.

The globalisation of trade in live ratites and products has led to particular difficulties in drawing up sensible, biologically correct import protocols, as the information necessary is, in most cases, simply unknown. These birds are also physiologically so different from galliform poultry species (chickens, turkeys) or waterfowl (ducks, geese) that mere extrapolations are extremely inaccurate in most instances.

This paper is designed to present a compilation of available data on the diseases reported in ostriches which are of particular concern to the poultry industries. Also included, where relevant, is information pertinent to public health (e.g. Crimean-Congo haemorrhagic fever [CCHF]) or other animal industries (e.g. Eastern/Western equine encephalomyelitides). To allow a more complete interpretation, information available on a particular disease is not limited to ostriches, but also includes emus and rheas. The relevant papers in this special issue should be studied in conjunction with these sections, to allow comparisons with the situations in poultry. Where technical details on tests and vaccines for ostriches or ratites are not mentioned, this information is simply not available.

Public health concerns

Salmonellosis (Salmonella Typhimurium and Salmonella Enteritidis)

These non-host specific members of the genus Salmonella cause disease in most warm-blooded animals (including birds) and are thus of concern in terms of both the economical production of food animals and the possible transmission to human consumers, resulting in enteritis and/or septicemia. Salmonella Typhimurium and S. Enteritidis have been associated with clinical conditions in young, stressed (immuno-compromised) ostriches, similar to the situation in other host groups (70, 71, 72, 152; Onderstepoort Veterinary Institute [OVI], unpublished case reports 1993-1998). In particular, these agents have been implicated in mortalities of ostriches and emus from multispecies collections, animal dealers and similar ‘zoo type’ situations which are predisposed to cross-species transmission of pathogens under highly stressful conditions (147).

Pathology in all peracute and acute cases shows ecchymotic to suffusive haemorrhages in the serosa of the gastrointestinal tract (GIT) and parenchymatous organs. The mucosa of the small intestine (jejunum, ileum) and/or colon is reddened with patchy ulcerations and adherent fibrinous exudate. The spleen is constantly enlarged and dark red/purple in colour (congestion) while the liver is also congested and swollen with focally disseminated necrosis (white spots) (72, 147).

Outbreaks amongst young ostrich chicks on large ostrich farms in southern Africa are usually associated with rodent-contaminated feed or raw materials, exposure to free-flying feral pigeons, doves, sparrows etc. in the chick runs, or the use of open water reservoirs or direct piping from surface water/contaminated bore holes (ground water contaminated by primitive, long-drop type latrines) (74, 145; OVI, unpublished case reports 1993-1998). Effective antibiotics, as determined through antibiograms combined with complex probiotics in a so-called ‘tandem programme’ (morning/evening alternation), followed by constant exposure to probiotics for a period of one week, have been successful in most of these outbreaks. Prevention has been achieved through the use of complex carbohydrates (e.g. mannose oligosaccharides), at an inclusion rate of 2 kg/tonne, during the critical first few weeks of life, in conjunction with strict attention to disinfection and biosecurity issues, especially pre-slaughter quarantine of thirty days is strongly advised, whilst live exports and fertile eggs should be screened through the additional use of sentinel chickens and/or young ostriches.

Keywords

those related to feed and water management (72, 153). Older animals are typically able to carry and intermittently shed salmonellae for an extended period (22, 75).

Pre-export testing of more than a thousand live ostriches from three farms in Namibia destined for Europe, aged seven to nine months, revealed the following (152):

In total, 2,005 cloacal swabs were cultured for Salmonella using selective media over a period of seven days. Thirteen Salmonella isolations were made: four of S. Typhimurium; three of S. Anatum; two of S. Reading and one each of S. Tanzania, S. Lamberhurst, S. Muenchen and S. Enteritidis (untypable). Of the 2,573 serum samples tested, nineteen were positive for S. Enteritidis (0.7%), ten for S. Gallinarum/Pullorum (0.38%), and two for S. Hadar (0.07%).

The isolates of Salmonella detected by the ostrich diseases diagnostic programme at the Onderstepoort Veterinary Institute (OVI), South Africa, during investigations into clinical cases from 1996 to 1997, are shown in Table I (152).

Results of the analysis of all the ostrich Salmonella isolates sent to the OVI Salmonella laboratory for serotyping between 1988 and 1996 are listed in Table II (146). The primary isolations of these samples were made by the OVI, and by various regional veterinary and other laboratories in South Africa. The

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td>1988</td>
<td>5</td>
</tr>
<tr>
<td>1989</td>
<td>1</td>
</tr>
<tr>
<td>1990</td>
<td>4</td>
</tr>
<tr>
<td>1991</td>
<td>2</td>
</tr>
<tr>
<td>1992</td>
<td>12</td>
</tr>
<tr>
<td>1993</td>
<td>29</td>
</tr>
<tr>
<td>1994</td>
<td>18</td>
</tr>
<tr>
<td>1995</td>
<td>13</td>
</tr>
<tr>
<td>1996</td>
<td>36</td>
</tr>
<tr>
<td>Total</td>
<td>120</td>
</tr>
</tbody>
</table>

Salmonella isolates were sent to the OVI Salmonella laboratory for serotyping. The majority of the isolates were obtained from clinical samples. The period mentioned coincided with a tremendous increase in the number of ostrich farms in South Africa and thus represents samples that originated from a wide range of farms, both new and very well established (100 years or more), as well as operations varying from a few hundred birds to large flocks of 10,000 birds or more.

Over this period, 120 Salmonella isolates were received by the OVI Salmonella laboratory. For the first four years of the survey period (1988-1991), very few isolates were received (a total of twelve), but from 1992 onwards, twelve to thirty-six isolates were identified per year. Serovar Typhimurium was the serovar most frequently isolated (25/120), followed by Muenchen (11/120) and Brancaster (8/120). An unexpectedly high number of Salmonella II isolates was detected (15/120). However, these were all different isolates, and do not represent the prevalence of a single serovar. Salmonella II serovars are not regarded as primary pathogens, and their presence in these ostriches is not indicative of a disease. In some of these cases (4/15), other salmonellae were also isolated which may have been of greater importance.

Typhimurium was the most common Salmonella in all types of samples, and isolation of this serovar from clinical material was always of pathological importance. The age of the birds affected ranged from young chicks to three-month-old ostriches. The pathological conditions observed were septicaemia with lesions, especially in the liver, spleen and lung, as well as enteritis.

From a public health perspective, the Salmonella status of slaughter ostriches (typically twelve to fourteen months old) is of much greater importance than that of chicks or clinical cases. Huchzermeyer mentions the possibility of carcass contamination during slaughter and notes that unlike mammals, birds (including ostriches) and reptiles have no mesenteric lymph nodes, and thus enteric Salmonella infections could, in theory, rapidly become septicaemic and be present in internal organs as a result of severe stress (71,

Table I
Isolations of Salmonella from clinical investigations by the Onderstepoort Veterinary Institute Ostrich Diagnostic Programme from 1 June 1996 to 25 April 1997

<table>
<thead>
<tr>
<th>Serotype</th>
<th>Number of isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. Typhimurium</td>
<td>13 + 1 (water)</td>
</tr>
<tr>
<td>S. Muenchen</td>
<td>8</td>
</tr>
<tr>
<td>S. Tsenie</td>
<td>3</td>
</tr>
<tr>
<td>S. Enteritidis</td>
<td>3</td>
</tr>
<tr>
<td>S. Riggil</td>
<td>1</td>
</tr>
<tr>
<td>S. Georgia</td>
<td>1</td>
</tr>
<tr>
<td>S. Malidiguri</td>
<td>1 + 1 (feed)</td>
</tr>
<tr>
<td>S. Thompson</td>
<td>1</td>
</tr>
<tr>
<td>S. Chester</td>
<td>2</td>
</tr>
<tr>
<td>S. Lamberhurst</td>
<td>1</td>
</tr>
<tr>
<td>S. Papuana</td>
<td>1</td>
</tr>
<tr>
<td>S. Djakarta</td>
<td>1</td>
</tr>
<tr>
<td>S. Isangani</td>
<td>1</td>
</tr>
<tr>
<td>S. Stonefoyer</td>
<td>1 (feed)</td>
</tr>
<tr>
<td>S. type II</td>
<td>2</td>
</tr>
<tr>
<td>Citrobacter</td>
<td>2 (a)</td>
</tr>
<tr>
<td>Total</td>
<td>45 (b)</td>
</tr>
</tbody>
</table>

(a) suspected Salmonella on preliminary bacteriological investigation
(b) includes two isolates from feed samples, one water sample and two Citrobacter isolates
Total cases with suspected bacteriological antecility investigated during this period = 280
Thus Salmonella isolates per bacteriological cases seen = 13.6%
Pathogenic Salmonella = 25/45 = 56%; 25/280 = 8.9% of total
Ongoing surveillance of the Salmonella status of slaughter ostriches (partially sponsored by the International Atomic Energy Agency) is conducted in South Africa. Preliminary results collected from 311 ostriches from all regions, over a nine-month period, are shown in Table III (132).

Table III
Salmonella isolations and Salmonella serology agar gel immunodiffusion (AGID) from the ongoing Meat Hygiene Surveillance Programme conducted in South Africa on export slaughter ostriches (preliminary data: 1998/1999)

<table>
<thead>
<tr>
<th>Salmonella serotype</th>
<th>Bacteriology</th>
<th>Serology</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. Typhimurium</td>
<td>4.2%</td>
<td>4.5%</td>
</tr>
<tr>
<td>S. Enteritidis</td>
<td>0%</td>
<td>2%</td>
</tr>
<tr>
<td>S. Hadar</td>
<td>0%</td>
<td>0.3%</td>
</tr>
<tr>
<td>S. Muenchen</td>
<td>0.3%</td>
<td>ND</td>
</tr>
<tr>
<td>Other*</td>
<td>1.2%</td>
<td>ND</td>
</tr>
<tr>
<td>Total</td>
<td>5.7%</td>
<td>6.8%</td>
</tr>
</tbody>
</table>

* Included S. Lamberhurst, S. Salamae tsevie and S. Sondiego, each at approximately 0.4% prevalence
ND: no data

Testing of exotic birds in the United States of America (USA) revealed that one out of seventeen ostriches (5.8%) were positive by the slide agglutination test for S. Typhimurium, while all seven emus tested were negative (58). Salmonella isolation attempts from ratites in 1993 in Oklahoma were positive in forty-six out of 248 ostriches, thirty-four out of ninety-nine emus, and sixteen out of sixty rheas. The total incidence was approximately 23% (96/407). In contrast, fifteen out of 181 attempts were positive in 1992. Serotypes isolated from ratites are shown in Table IV.

Table IV
Serotypes of Salmonella isolated from ratites during 1993 in Oklahoma, United States of America

<table>
<thead>
<tr>
<th>Serotype</th>
<th>Ostrich</th>
<th>Emu</th>
<th>Rhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. Typhimurium</td>
<td>20</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>S. Typhimurum (Copenhagen)</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>S. Muenchen</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>S. Panama</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>S. Enteritidis (phagetype B)</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>S. Newport</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>S. Rubislaw</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>S. Livingston</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>S. Anatum</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>S. Montevideo</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>S. Godseberg</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Salmonella (Group B)</td>
<td>3</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>Salmonella (Group C)</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Salmonella (Group C2)</td>
<td>6</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Salmonella (Group E)</td>
<td>1</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Untypable</td>
<td>2</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>46</td>
<td>34</td>
<td>16</td>
</tr>
</tbody>
</table>

Live birds should be screened through culture of cloacal swabs and possibly the use of sentinel ostriches or chickens.

Where approved, an alternative approach is the irradiation of meat to ensure freedom from pathogens, including Salmonella. Samples of meat from bison, ostrich, alligator and caiman (similar in proximate analysis) which were experimentally inoculated with several species of Salmonella and Staphylococcus aureus, were effectively sterilised by gamma irradiation. Based on the average gamma radiation D values obtained in this study and the minimum dose currently approved for the irradiation of poultry (1.5 kGy), gamma irradiation would eliminate 2.8 and 4.1 log units of Salmonella and S. aureus, respectively (139). The resistance of Salmonella to gamma radiation on these 'exotic' meats was less than that reported for beef, lamb and turkey (138). However, due to the very low prevalence of salmonellae in slaughter ostriches reported above, such an all-inclusive approach to treatment is probably unnecessary.

Campylobacteriosis

Campylobacter jejuni has been reported in young ostriches from several parts of the world. In Israel, two-week- to four-month-old ostrich chicks showed depression, anorexia, dehydration and green urine. Mortality reached 40% and the pathology resembled vibrionic hepatitis in poultry. Campylobacter jejuni serotype 8 was isolated from the livers of affected birds. Treatment with furaltadone (250 mg/l drinking water) in the younger birds, and with norfloxacin (30 mg/kg live weight) in the older birds, reduced mortality (106).
Campylobacter was isolated from the intestines of ostriches, emus and rheas in the USA, with some isolates from emus displaying uncharacteristic properties (101, 102).

In South Africa, C. jejuni was found to be associated with outbreaks of enteritis and hepatitis in ostrich chicks ranging from three weeks to two months old. Affected chicks showed depression, anorexia and diarrhoea, while the pathological lesions were mainly a pronounced typhlocolitis and extensive multifocally disseminated micro-abscesses in the liver. In all cases, the history involved severe stress (e.g. long transport distances, cold temperatures, overcrowding, mycotoxicoses).

Effective treatment consisted of antibiotics (danofloxacin at 5 mg/kg) coupled with complex probiotics in the food. A particular contract-chick feeding operation experienced repeated outbreaks despite changes in management. An experimental autogenous inactivated Campylobacter bacterin vaccine in an oil emulsion adjuvant enriched with vitamin E, given to all chicks on arrival, seemed to assist in preventing further outbreaks in this specific operation. In most instances, Campylobacter-associated mortalities or poor growth were sporadic and were resolved with the treatment described above and prevented by changing management-related stressful procedures (OVI, unpublished case reports 1993-1998).

Campylobacter jejuni has also been isolated from rheas in the USA, where a three-month-old rhea chick was presented with typical vibronic hepatitis lesions in one liver lobe. An in-contact ostrich chick died from yolk sac infection and C. jejuni was also isolated from the yolk (112). In another incident, C. jejuni was isolated from a single rhea where a number of the flock had died from spirochaete-associated typhlocolitis (60).

Campylobacter is known to be a part of the natural flora of the intestinal tract of many species of birds (101), as well as many domestic mammals, with average carrier rates of 60% for broiler chickens, 20% for cattle and sheep and 80% (C. coli) for pigs reported from surveys in several countries (127). No such data are available for slaughter age ostriches, and as mass mechanised processing is blamed for the high levels of cross-contamination associated with poultry meat, individual slaughter and evisceration procedures during ostrich slaughter should prevent carcass contamination.

There are no indications for the inclusion of special Campylobacter culture testing as part of the screening of either live ostriches, eggs or ostrich products for international trade.

Chlamydiosis

The world-wide distribution of strains of Chlamydia psittaci in a large number of avian species (in 1971, 139 species representing fourteen orders and approximately thirty families [28]) and the observation that feral birds could be a source of infection for domestic poultry, implies that the goal of total control is unrealistic (59). An outbreak in young ostriches, resulting in high mortality and the infection of human contacts, has been reported from a game park in France (107). Chlamydiosis has also been reported from an ostrich chick in North America (74), while the agent was isolated in Texas from a cloacal swab of an adult ostrich hen that had a low positive titre and had laid infertile eggs. Isolates from eleven ratites submitted to diagnostic laboratories in Texas and California were serotyped using serovar-specific monoclonal antibodies and characterised by polymerase chain reaction (PCR) restriction fragment length polymorphism of the major outer membrane protein genome. All were determined to be serovar E. This serovar is known to have existed in the USA for more than sixty years, while a number of isolates obtained from ducks in England, during outbreaks of high mortalities, were also found to be this serovar, in retrospective studies (11).

In another monoclonal antibody study in Israel, conjunctival, cloacal and faecal smears from seventy-four ostriches revealed the presence of C. psittaci antigen in 23% of the birds (88). An outbreak of chlamydiosis in three- to five-month-old ostriches in Namibia resulted in 37% mortality in a flock of 160 birds (84). After translocation from another farm, the birds became depressed, developed dyspnoea and a 'penguin-like' gait, and died acutely. Macroscopic pathology included fibrinopurulent tracheitis, pneumonia, pericarditis and perhepatitis. Out of several thousand investigations of mortality in ostrich chicks from 1993 to 1998, principally from ostrich farms located in the central (summer-rainfall) region of South Africa, clinical chlamydiosis was found in a single case (72; OVI, unpublished case reports 1993-1998). These chicks displayed a severe necrotising hepatitis and splenitis, with chlamydial colonies prominent in both organs on histological examination.

The organism was isolated from a single case of unilateral conjunctivitis in a juvenile ostrich from Germany (85), while conjunctivitis or sudden death was also the main disease sign in rhea in several cases from Texas (57). A further three cases in rhea ranging in age from two months to three years, with prominent splenomegaly were reported from Louisiana (33).

These reports clearly indicate that ostriches and other ratites typically reared in open-air camps which allow regular direct and indirect contact with wild birds (including feral pigeons) are susceptible to Chlamydia infections (comparable to the situation in turkeys). This is contrary to suggestions of only 'anecdotal evidence of neonatal susceptibility' (145).

Historically, the pandemic of human chlamydial infections of 1929-1930 focused on contact with psittacine species as the main risk factor. However, shortly afterwards, non-psittacine
birds, particularly turkeys and ducks, were recognised as sources of human infection, and inapparent/latent infections or carrier/intermittent shedding were demonstrated to be typical of most avian species (121).

Chemoprophylaxis with medicated feed (chlorotetracycline) during the standard thirty-day quarantine has been regarded as an effective, practical and economically feasible method to provide adequate safeguards for quarantine station employees (e.g. in the USA), against chlamydial transmission from psittacines. However, as the recommended treatment for chlamydiosis is constant tetracycline treatment for a period of forty-five days, even an official thirty-day treatment would fail to prevent the introduction of chlamydiae into a country via the exotic bird trade (49, 116, 121). Recently-developed antibiotics (quinolones) could possibly offer a solution (86).

Demonstration or identification of C. psittaci in live birds during periods of non-shedding remain extremely difficult. Tests used include culture, serological testing, immunoassays and molecular (PCR) based tests. These tests are detailed in the Office International des Epizooties (OIE) Manual of Standards for Diagnostic Tests and Vaccines (99), but comparison studies concluded that any live bird assay may fail to detect chlamydial infection because of detection limits (sensitivity thresholds), cross-reactions and non-shedding.

The most recent techniques (i.e. PCR-based technology) have not been compared with these other tests in a variety of situations in domestic and exotic birds (51). Although several reports have described and indicated the advantages in terms of sensitivity and ease of sampling of the techniques used in PCR amplification of chlamydial nucleic acid sequences (62, 77), limited comparisons with ELISA showed severe discrepancies (in both directions), indicating that caution is still required when interpreting results (47). In particular, the situation regarding chronically infected birds that harbour mainly intracellular chlamydiae in target organs, remains a challenge for diagnosis and screening.

Due to a paucity of information regarding the biology of C. psittaci in ostriches and in light of the above discussion, the most that can be concluded in relation to international trade and human health aspects of ostriches is the following:

a) ostriches are susceptible to C. psittaci infections and probably display the same outcomes following infection as most other groups of birds (ranging from inapparent infection to acute mortality)

b) the sensitivity and specificity of serological tests or immunoassays for C. psittaci in ostriches is unknown, but PCR technology would probably be as useful in the testing of this species as in any other

c) due to the very wide host range and the presence of C. psittaci in almost all avian groups, the organism can be regarded as ubiquitous, and before being implemented, the value of testing or screening live ostrich imports should be evaluated against the background risk posed by migratory birds (particularly waterfowl). The testing or treatment of clinically affected birds or flocks coupled with high levels of disinfection during quarantine is probably more sensible than reliance on the screening of healthy birds or the treatment of sero-negative birds (53).

**Crimean-Congo haemorrhagic fever**

Crimean-Congo haemorrhagic fever is a zoonotic disease caused by a tick-borne ribonucleic acid (RNA) virus of the family Bunyaviridae, genus Nairovirus, which is present in Africa, Asia and Eastern Europe.

The virus causes only a mild fever and viraemia for up to one week in cattle, sheep and small mammals (especially hares), while clinical disease occurs only in humans, resulting in approximately 25%-30% mortality (134). Infection can be caused by contact with body fluids from viraemic hosts but is mainly associated with tick bites. Although the virus has been isolated from thirty species of ticks, trans-stadial and transovarian transmission has only been reported for Hyalomma marginatum, Rhipicephalus sanguineus and Dermacentor marginatus, and the distribution of the disease coincides closely with that of these ticks (66), strongly suggesting that they are the most important vectors of CCHF virus.

Limited observations in passerine birds and domestic chickens have found these birds to be refractory to the virus, while experimentally infected guinea-fowl developed a transient viremia of very low intensity and an antibody response of only a few weeks duration. Such birds are therefore unlikely to be able to infect ticks (125, 134). However, migratory birds carry immature ticks, and could thus serve to disseminate CCHF virus which has been transmitted transovarially in the ticks (67, 68). The relatively high prevalence and titres of antibody found in ostriches, which commonly carry the xerophilic Hyalomma ticks in the semi-arid regions where they are farmed on extensive ranches and riverside alfalfa grazing camps, indicate that ostriches may be more susceptible to infection than other birds (66, 125). Approximately fifteen to twenty-five cases of CCHF in humans are recorded every year in South Africa, mainly in adult men engaged in the livestock industry (farmers, labourers, abattoir-workers and veterinarians) in the semi-arid regions of the country (where Hyalomma commonly occurs), with a slight preponderance of cases from February to March and October to November, when adult Hyalomma tend to manifest peak questing activity (active searching by ticks for their vertebrate hosts) (115, 134).

World attention was again focused on CCHF and ostriches after seventeen abattoir workers at an ostrich abattoir in Oudtshoorn, South Africa, contracted the disease after contact with a group of ostriches with a particularly heavy tick infestation. One patient died, while all the others recovered fully. Due to concern about the safety of exported ostrich meat to consumers, the European Union (EU) temporarily suspended exports from South Africa (41). This prompted the
experimental infection of nine ostriches raised under tick-free conditions with CCHF virus to determine viraemia and the immune-response curve (34, 136). These results can be summarised as follows:

a) no clinical symptoms are associated with CCHF in ostriches
b) viraemia lasts approximately four days
c) virus can be isolated from blood and organs
d) virus was not isolated from muscle, but CCHF genetic material was detected by reverse transcriptase-PCR (RT-PCR)
e) seroconversion begins on day five post-infection and by day thirteen, antibodies could be detected in all surviving experimentally infected ostriches.

These results were the foundation for the Decision 97/138/EC, which revoked the ban under certain conditions (42). This decision included protection measures which were extended to all countries of Africa and Asia and consisted of treatment by pyrethroid-based acaricides, with ostriches kept free of ticks in rodent-controlled areas, for at least fourteen days prior to slaughter. These requirements apply to both slaughter birds and ostriches destined for live export. Although these requirements are practical, economically feasible and have been strictly applied since 1997 by the Veterinary Services of South Africa, the potential for the introduction of CCHF-viremic ostriches from countries in Eastern Europe, where the relevant ticks naturally occur and the virus causes regular clinical infections in humans (e.g. historically in the Crimean peninsula), was not considered by the EU authorities (34). In fact these safety measures should logically be extended to all domestic livestock and the movement of both live animals and meat (pre-slaughter measures) across borders (134, 136), as the risks regarding tick contact or viraemic periods in these different classes of animals are very similar.

No evidence has been obtained to suggest that CCHF virus constitutes a public health hazard in meat from any animal or bird processed and aged according to normal health regulations (66, 134, 135). The measures outlined above should be enacted as an additional safeguard, protecting principally those who would normally come into contact with fresh blood of potentially viremic animals (e.g. abattoir workers).

Serological screening can be performed using a competitive ELISA (C-ELISA) (29) or sandwich ELISA using conjugated antiserum to ostrich immunoglobulins (Ig) (136). The sandwich ELISA was slightly less sensitive than the former, but correlated very well over the fourteen day experimental period. Viral RNA can be detected by RT-PCR in infected material (30), while the virus can be isolated in Vero cells or by intracerebral inoculation of day-old mice (124). Due to the significant zoonotic risk to laboratory personnel, these isolations should only be undertaken in a maximum risk bio-secure laboratory facility.

**Spongiform encephalopathy**

Bovine spongiform encephalopathy (BSE), also known as 'mad cow disease' is a fatal disease thought to be caused by a prion (similar to scrapie in sheep) and is associated with variant Creutzfeldt-Jakob disease in man (36, 113). Experimental infections were successful in several animal species, including cattle, pigs, sheep, goats, marmosets and mice, and disease has been reported in wild captive animals in the United Kingdom (UK) (81). The disease causes central nervous symptoms and is diagnosed by histopathological examination of brain tissue and the demonstration of scrapie-associated fibrils (159). Spongiform brain lesions were reported in three adult ostriches from two zoos in Germany which had a history of central nervous system disease with ataxia, imbalance and unco-ordinated feeding. The material was not examined for scrapie-associated fibrils. The birds had been fed a proprietary poultry feed which included carcass meal. Allegedly, the birds had also been fed meat, some of which was from injured cattle slaughtered in cases of emergency. If this was indeed the case, it was certainly a strange and unnatural husbandry practice (71, 72). Due to the sensitivity over BSE and related conditions, ostrich rations should not contain carcass meal (81, 123). From these reports, it is not clear whether Newcastle disease (ND) was included in the differential diagnosis, but the authors concluded that a definitive diagnosis could not be made and that a toxic or nutritional aetiology could not be discounted.

A review of more than a thousand diagnostic ostrich cases submitted to the OVI during the period 1993-1998, as well as published reports of similar symptomatology in ratites (156) is presented below as a differential diagnosis list for 'nervous conditions' in ratites:

- Newcastle disease
- hypoglycaemia (e.g. impacted stomach)
- trauma
- pain (torticollis, head swaying; e.g. penetrating foreign objects in proventriculus/gizzard)
- Western equine encephalomyelitis
- Eastern equine encephalomyelitis
- Borna disease
- spongiform encephalopathy
- bacterial septicaemia (Clostridium perfringens, Pseudomonas aeruginosa, Salmonella spp. and Escherichia coli strains)
- Chandlerella quiscoli
- Balyascaris procyonis
- gangliosidosis
- hepatic encephalopathy (fatty degeneration of liver)
- lead poisoning (ingested battery plates)
- *Clostridium botulinum*
- neoplasia (cerebral haemangiosarcoma)
- mycotoxicoses (e.g. from suspected *Fusarium moniliforme* toxins).

In southern Africa, the first four entries above were by far the most common causes of reported ‘nervous signs’ in ostriches over six months of age, while bacterial septicaemia and hypoglycaemia were the most important in ostrich chicks.

Ostrich products or live ostriches of any age do not appear to pose any risk to an importing country regarding BSE.

**Anthrax**

Anthrax is a peracute disease characterised by septicaemia and sudden death with the exudation of tarry blood from the body orifices of the cadaver. The disease is caused by *Bacillus anthracis*, which forms highly resistant spores upon exposure to air, protracting the infectivity of a contaminated environment. The disease occurs world-wide, mainly in poorly drained alkaline soils and has been reviewed extensively in standard textbooks (25, 40).

Ostriches are the only birds known to be susceptible to anthrax, possibly because of a generally lower body temperature than other birds (72). Many field cases were reported in the early 1900s in South Africa (117), while experimental infections led to the description by Theiler of two syndromes, namely: sudden death and ‘anthrax fever’, both of which could occur simultaneously in a flock (140).

Sudden death was associated with petechial haemorrhages on the pleura and peritonium, congestion of the intestines, a normal or enlarged spleen with very dark pulp and typical *B. anthracis* bacilli in the blood, demonstrated by standard smears.

Anthrax fever caused anorexia and somnolence but ostriches recovered naturally or after treatment with penicillin. No bacilli could be found in the blood smears from such birds.

A recent outbreak of anthrax in a dairy herd in the Eastern Cape, South Africa, was associated with the feeding of infected bone meal, which was a common method of anthrax transmission in the UK in the pre-BSE era (25). The outbreak resulted in several bovine mortalities, but no ostriches on the same farm became ill or died, despite receiving the same feedstuffs (OVI, unpublished case reports 1993-1998).

The disease was controlled in cattle in the early part of this century in South Africa and most parts of the world with the advent of effective vaccines. These vaccines also successfully protect ostriches from the disease (140). Clinically healthy ostriches, fertile eggs and ostrich products therefore do not constitute any anthrax risk to an importing country.

**Poultry diseases**

**Fowl typhoid (*Salmonella Gallinarum*) and pullorum disease (*Salmonella Pullorum*)**

No field cases or experimental evidence have reported infection with either *S. Gallinarum* or *S. Pullorum* in ostriches (145; OVI, unpublished case reports 1993-1998). *Salmonella Pullorum* has been reported to have the potential to infect emus (144), but no cases have been reported from any rations.

Laboratory diagnosis should be performed according to requirements stated in the OIE *Manual of Standards for Diagnostic Tests and Vaccines* (99). The Manual states that the serum agglutination test is satisfactory for individual birds for establishing presence and prevalence within the flock in the case of poultry. However, due to the significant interference by non-specific agglutinins present in most ostrich sera in any agglutination-based test (see discussion of inactivation in section on Newcastle disease virus), this screening method has severe limitations when used for ostriches. Positive reactors should be confirmed as being infected by culture. The ELISA systems should be established as soon as possible to allow accurate serological screening or diagnosis of ostriches.

South Africa, which still dominates world trade in ostriches and ostrich products and where an estimated 60% or more of domestic ostriches are found, successfully eradicated both *S. Pullorum* and *S. Gallinarum* from poultry in the 1950s and has been officially free from these diseases since that time. They remain ‘controlled diseases’ under the South African Animal Diseases Act (Act 35 of 1984) and should any outbreaks occur, they will be stamped out. Similar certification from competent Veterinary Services should be all that is required from countries exporting ostriches and ostrich products.

**Avian mycoplasmosis (*Mycoplasma gallisepticum*)**

*Mycoplasma* has been isolated from ostrich chicks as well as feedlot and breeder birds, and their pathogenicity has been suspected. This group of organisms was isolated from the lungs and trachea of 32 out of 372 ostrich chicks in North America, unassociated with any pathology (126). Such agents have been isolated regularly during winter from feedlot ostriches in South Africa with respiratory symptoms, concomitant with a range of other opportunistic pathogens, including *E. coli*, *P. aeruginosa*, *Pasteurella* spp. and even occasionally *Haemophilus paragallinarum*. The role of mycoplasmas is unclear in such cases (72; OVI, unpublished case reports 1993-1998). *Mycoplasma synoviae* has also been isolated in several of these cases while mycoplasmas were demonstrated by electron microscopy in GIT contents from chicks with emeritis younger than four weeks. Clinical improvements were noted after treatment with tylosin at 300 ppm in the starter ration (72; OVI, unpublished case reports...
1993-1998). Positive serological reactions to the poultry pathogens *M. gallisepticum* and *M. synoviae* in ostriches have been reported from Italy (104). Experimental inoculation of six- to eight-week-old ostriches with *M. gallisepticum* and subsequent recovery through culturing and PCR, demonstrated that this agent can colonise the tracheas of young ostriches. A similar trial with *M. synoviae* gave inconclusive results (38).

A chronic severe sinusitis caused by *M. gallisepticum* was reported in rheas (109), while *M. synoviae* was isolated from another flock of rheas in North America with clinical signs of mycoplasmosis (162).

Given the apparent susceptibility of ostriches and rheas to poultry mycoplasmas and the lack of information concerning the role of these and other species of *Mycoplasma* in pathological conditions of this class of birds, ratite farms are recommended to be kept free from poultry (72). Ostriches destined for export should be clinically healthy, especially regarding upper respiratory infections (i.e. periorcular swelling of sinuses). Due to the limitations of agglutination-based tests in ratites, quarantine testing of live birds should utilise sentinels instead. Fertile eggs should originate from clinically healthy, closed breeding flocks. There are no indications that ostrich meat is associated with a risk of spreading *Mycoplasma*. 

**Pasteurellosis**

Haemorrhagic septicaemia caused by *Pasteurella multocida* has caused ostrich mortality in zoos during outbreaks that simultaneously affected many species of animals (95, 100). The cases varied from acute deaths to illness lasting several weeks.

Nine ostriches of ten to twelve months of age died at Frankfurt Zoo after several weeks of illness and recumbency, during which time the birds maintained an appetite. The disease commenced with lacrimation and conjunctivitis, while splenic abscesses were noted at necropsy and *P. multocida* was isolated from heart blood (95).

In Kano Zoo, Nigeria, an ostrich died acutely along with several game species, kangaroos and a bateleur eagle. The post-mortem examination revealed generalised congestion with petechiae and ecchymoses on the epicardium, endocardium, kidneys and intestines (100).

*Pasteurella multocida* has also been isolated from several cases of acute mortality in ostrich chicks from farms in South Africa. Post-mortem findings were non-specific and resembled the report from Nigeria. Outbreaks were easily contained with synthetic penicillins and prevented through improved biosecurity, especially regarding rodent control and contact with wild birds and surface water (OVI, unpublished case reports 1993-1998).

As no clinically ill ostrich should be accepted for export or slaughter, no specific testing against *P. multocida* in ostriches or ostrich products is needed, and certification in that regard should be the only requirement.

**Newcastle disease virus**

All avian species are considered susceptible to infection with Newcastle disease virus (NDV), although members of the families Anseriformes (waterfowl) and Psittaciformes (parrots) have been identified as subclinical carriers, intermittently shedding strains highly virulent for poultry and thus suggesting a higher innate resistance or threshold level than domestic poultry (2, 4, 56, 70, 163). This is of particular importance for the control of the disease in birds allowed free flight (e.g. racing pigeons and those birds reared in open-air camps, feedlots or on extensive grazing pastures, such as turkeys, backyard chickens and raptors). The control of ND in racing pigeons remains a world-wide challenge due to the nature of the sport (large numbers of birds from many different lofts are collected and transported together in confined spaces, with long flight distances, often across international borders, and many thousands of pigeons 'lost' annually which mix with feral populations) and the established relationship between NDV outbreaks in poultry and the pigeon-associated (P-paramyxovirus [PMV]-1) strain that occurred in the UK in 1984-1985 and again in 1997 (2, 5, 6, 16, 24, 44, 87).

This NDV serotype has been isolated from captive wild birds in South Africa (39), and pigeons and doves regularly visit ostrich feed containers on farms in this country, but P-PMV-1 has never been isolated from ostriches in South Africa.

Historically, ND in ostriches had been reported as isolated cases in zoos and circuses only (43, 79, 82, 110). In 1989, outbreaks occurred in farmed ostriches in Israel (105, 120), where the birds were reared in close proximity to commercial poultry. Approximately 30% of five- to nine-month-old birds died within three weeks. A velogenic NDV isolate ('Israel 67') caused approximately 80% mortality in experimentally infected three-month-old ostrich chicks within five to ten days and the virus was isolated from various organs from all dead birds.

Following a particularly severe outbreak of NDV in poultry after a twenty year absence, virulent NDV caused mortality in ostriches during several outbreaks on commercial ostrich farms in South Africa beginning in 1993 (14, 69, 70, 73, 149). As the epidemic spread to poultry in other countries in the region, outbreaks also occurred in ostriches in Namibia, Botswana and Zimbabwe.

The viruses isolated from these cases were identical to poultry isolates from this region obtained at the same time (93), and contact with or trade in backyard chickens and/or commercial poultry appeared to be the source of NDV in ostriches, rather than wild birds. Similar conclusions were reported in a recent
study conducted in Switzerland (24, 72, 122, 149) (Fig. 1). Field and experimental data indicate that the route and mechanism of infection have a major influence on the clinical severity of the disease in ostriches. Severe respiratory disease with rapid spread and high mortality is seen only in closed chick-rearing units, while ostriches kept outdoors usually contract the disease by the oral route from faeces or water, resulting in nervous signs and a very slow spread of infection (70, 72, 151, 154). Similar slow or limited spread of NDV has been reported in backyard chicken flocks in California (44).

These observations may be partly due to the sterilising effect of the ultraviolet rays of the sun in the semi-arid regions of southern Africa where ostriches are farmed (72). Several experimental challenge studies have been conducted in South Africa, investigating different aspects of the disease in different age groups of ostriches, including efficacy testing of inactivated aluminium hydroxide (AlOH) La Sota-strain vaccine. In all cases, challenge was with the field isolate of velogenic NDV, which has been active in southern Africa since 1993 (mean death time [MDT]: 42 h to 48 h; intracerebral pathogenicity index [ICPI]: 1.7) (8, 69, 150, 151, 154).

These results can be summarised as follows:

a) All ages investigated (two weeks to fourteen months old) were susceptible to velogenic NDV.

b) While clinical signs show a typical progression, no pathognomonic macro- or microscopical lesions are found in NDV-affected ostriches. Post-mortem lesions included subcutaneous head and neck oedema, splenomegaly, petechiae on serosal surfaces and occasionally paintbrush haemorrhages in the gizzard below the kollin layer, while perivascular lymphocytic cuffing was occasionally seen in the brain. The lesions have also been described from field cases (8, 72, 145).

c) Currently available La Sota vaccines (live plus inactivated) can stimulate protective immunity in ostriches which lasts more than six months. The immune response curves, after challenge with virulent NDV, from 142 slaughter ostriches divided into three groups that had been vaccinated approximately one to two months, two to four months and four to six months earlier, showed no significant differences between the groups. The response levelled at maximum titres on day fourteen post infection. This humoral immunity correlated very poorly with protection in the case of haemagglutination inhibition (HI) tests, and very well in the case of ELISA systems (154).

d) The period of viraemia in vaccinated slaughter ostriches (twelve to fourteen months old) was determined as nine to eleven days.

e) No evidence was found of a carrier state for velogenic NDV in slaughter ostriches.

f) The effective vaccination programme enforced by the Veterinary Services of South Africa, coupled with the pre-slaughter quarantine period necessary to comply with CCHF-control regulations, thus covered the determined viraemic period, and through statistical risk analysis methodology, it was determined that international trade in ostrich meat was not likely to spread NDV to non-endemic areas (e.g. the EU or USA).

g) Virological examination by cloacal or choanal swabs, using standard virological techniques (99), was unsuccessful even in clinical cases and therefore has no practical value as a screening tool (8, 151, 154). This is possibly due to the voluminous faeces of ostriches.
The serological testing of ostrich sera does not give such consistent results as in poultry. The HI test is regarded as unreliable, leading to both false positives and false negative results (8; OVI, unpublished case reports 1993-1998). Even heat treatment (56°C for 30 min) or absorption with kaolin does not remove all non-specific agglutinins. However, pre-treatment adsorption with 25 µl packed chicken red blood cells or ideally, ostrich chick red blood cells, will improve the accuracy of the HI assay (92; OVI, unpublished case reports 1993-1998). Positive titres are those equal to or greater than a 2^4 dilution using 4 haemagglutination units.

Stable conjugated antisera to ostrich Ig for indirect ELISAs have been developed (31, 161) and these are used as standard practice at the OVI. Recently, a monoclonal blocking ELISA which shows excellent sensitivity and specificity has become commercially available (45, 83), and this ELISA has the added benefit of not being limited to usage in one host species. These, or similar ELISAs should be the basis of serological screening of ostriches destined for export.

Cross-reactions with PMV-6 have also caused difficulties in the interpretation of NDV HI tests in quarantined, imported ostriches on two occasions in New Zealand (15, 37). The PMV-2 has been reported from ostrich chicks in the USA (126), while PMV-7 was isolated from two juvenile ostriches, one of them with impaction (164).

In all cases, the isolation of NDV is the ‘gold standard’ for diagnostic purposes (72, 99), with new molecular tests, e.g. RT-PCR, showing promise for the future, once validated in ostriches, to allow accurate interpretation of results from field samples or screening. This technology has already been included in the new OIE definition for NDV.

**Avian influenza**

Influenza A viruses of all fifteen known haemagglutinin subtypes have been isolated throughout the world from many domestic and wild species of bird (129). The highly pathogenic strains for chickens have all been of the subtypes H5 and H7, but viruses of these subtypes are not necessarily highly pathogenic (3). The considerable ability of influenza viruses to mutate to strains of higher virulence due to gene mutation or reassortment is well known (e.g. the H5N2 epizootics of 1983-1984 in Pennsylvania [48] and 1994-1995 in Mexico [151]), and this dictates close scrutiny of all H5 and H7 isolates from any species under any circumstances. The ability of influenza viruses of avian origin to cause epidemics of respiratory disease and even death in humans is of particular concern (1, 78, 133).

The epidemiology of avian influenza has been closely related to migrations and movements of wild waterfowl (3, 46, 64, 65, 130), and indirectly related via the use of untreated surface water, as the virus can persist in such environments for up to 200 days (131).

Domestic poultry that are kept or reared in open-air camps or utilise riverside grazing camps (e.g. turkeys or ratites), are particularly vulnerable to cross infection from waterfowl (3, 108, 111). Furthermore, evidence suggests interaction of influenza viruses of H1N1 subtype between turkeys and pigs (97).

The first reported outbreaks of avian influenza in ostriches occurred in 1991 and 1992 in the Eastern Cape Province and Oudtshoom district in South Africa, and were typed as H7N1, with no pathogenicity for chickens (7, 8, 9, 12, 13, 27). Clinically, the affected birds were depressed, had bright green discoloration of the urine, respiratory signs and ocular discharge, with mortality reaching 60% in some groups. The severity of symptoms and lesions depended on age and concurrent infection with *F. coli*, *P. aeruginosa*, *S. aureus* and *Aspergillus fumigatus* (air sacs). On post-mortem examination, the livers were enlarged, mottled and friable with coagulative necrosis surrounded by marked heterophil infiltrate and vasculitis histologically. Areas of necrosis were also present in the spleen and pancreas, and severe congestion occurred in the small intestine in addition to necrosis of the tips of the villi.

Subsequent isolations from these same farms or from Zimbabwe, were from cases with similar pathology or even from inapparent infections. The isolates were typed as follows: South Africa H7N1 (1991, 1992), H5N9 (1994), H9N2 (1995), H6N8 (1998), and Zimbabwe H5N2 (1995), but none of these isolates were pathogenic for poultry (93; OVI, unpublished case reports 1993-1998). However, some ratite isolates of avian influenza virus were found to have a realistic potential for interspecies transmission to poultry and for mutation to more pathogenic variants (137). An apathogenic virus, subtype H5N2, was isolated from juvenile ostriches in quarantine in Denmark together with an apathogenic PMV-1, the mortality associated with this case appeared to have been stress-related (e.g. impactions and enteritis) (76).

An inactivated emulsified H7N1 vaccine was successfully used to curb the outbreaks in South Africa from 1991 to 1992; the vaccine prevented morbidity and mortality, but did not prevent shedding of virus (7, 8). A similar approach has been used as a temporary measure in poultry, using the dominant strain associated with a particular outbreak (3, 21). Should the outbreak be caused by a highly pathogenic avian influenza (HPAI) virus, control should be achieved through stamping-out and quarantine measures in that vaccination with such viruses could prevent mortality, but still allow replication and shedding of the virus (3). Use of live virus vaccines should not be permitted due to the ability of this virus to spread, causing mixed infections in the same host species that will allow gene reassortments and possibly virulent mutant offspring (1, 3). Several new-generation vaccines, based on molecular technologies, are being developed and field tested. These include recombinant
between emus and chickens in relation to certain strains of waterfowl, and the crucial role of husbandry practices in the prevention of outbreaks, particularly ensuring that no contact was made with infected birds. This discussion reiterates the importance of surveillance for avian influenza (and ND) in wild birds, particularly waterfowl, and the crucial role of husbandry practices in the prevention of outbreaks, particularly ensuring that no contact occurs between farmed ratites and wild birds, which is more difficult to predict, compared to the situation in chickens and turkeys. Recent experimental studies have started to address some of these aspects: using a highly pathogenic strain (H5N1) from turkeys as well as a non-pathogenic isolate (H5N2) from ostriches. Manvell and co-workers infected ten three-week-old chickens and ten two-week-old ostriches with each virus (94). The ostriches infected by the highly pathogenic isolate failed to show any clinical signs when inoculated by either the intramuscular or intranasal route, while the infected chickens all died within 42 h. Seroconversion and isolation of virus from swabs confirmed that infection with this strain was established. The virus of low virulence also established infection in ostriches and produced a higher immune response than in chickens. These findings suggest a complex relationship between host species and virus strains, with the possibility of host-adaptations.

Strains of H7N1 and H5N2 subtypes were isolated from emus and rheas in Texas and North Carolina in the USA, while positive sera were found in eleven States. These contained humoral antibodies to all known H-subtypes, except H10, H13 and H14, and to all nine N-subtypes (103). Experimental infection of four- to twelve-month-old emus with a highly pathogenic strain, H7N7 (four birds), and a strain of low pathogenicity, H5N3, was conducted in Canada (61). The birds were susceptible, with virus shedding detectable in tracheal and cloacal swabs between three and ten days post infection. A brief period of mild clinical signs was noticed only in the groups infected with the highly pathogenic virus. Viruses recovered from both groups were similar in pathogenicity to the respective inoculums. All birds had seroconverted by ten days post infection, as determined by HI, agar gel immunodiffusion (AGID) and C-ELISA. This study indicates a significant threshold difference between emus and chickens in relation to certain strains of HPAI.

This discussion reiterates the importance of surveillance for avian influenza (and ND) in wild birds, particularly waterfowl, and the crucial role of husbandry practices in the prevention of outbreaks, particularly ensuring that no contact occurs between farmed ratites and wild birds, either directly or indirectly (through surface water). Serology based on ELISA should be validated as a matter of urgency in ratites, while vaccines have a limited, probably localised geographical role in this group of birds. At this stage of limited knowledge and validation of avian influenza testing in ostriches, the use of sentinel chickens should be considered during pre-export quarantine to provide satisfactory screening against avian influenza, with HI or ELISA serological screening of the in-contact chickens, or as soon as the technique is validated, ELISA serological screening of the ostriches themselves.

**Borna disease**

The causative agent of Borna disease is a single-stranded RNA virus of the family *Rhabdoviridae*. The disease is present in Central Europe, the USA, Japan, Sweden and the Near East, predominantly affecting horses and donkeys, and exceptionally a variety of other species, causing a range of behavioural disturbances with or without neurological signs, such as paresis and paralysis. The mode of transmission is unknown, but the presence of the virus in nasal secretions, saliva and urine suggests that dissemination occurs either by direct contact or through fomites and feed. The relatively small amounts of virus found in such secretions, and the low level of spread suggests that the disease is not highly contagious. No vertical transmission or free-living reservoirs have been identified, although rats are suspected to be involved. Arthropod vectors have also been suggested, but the situation is unclear (118, 142).

Borna disease in ostriches has only been reported from Israel in 1989-1994 where the disease produced a specific paresis and mortality in two- to six-week-old ostrich chicks. All affected birds died within four to eight days. The inoculation of serum from paretic ostriches to ostrich chicks at risk, followed by serum from normal adults, was effective in the prevention of further disease, demonstrating the protective action of antibodies (17, 18, 19, 158). Laboratory diagnosis is by virus isolation, the demonstration of viral protein by ELISA (unvalidated) (91) and histopathology. The latter consists of lesions of neuronal necrosis, satellitosis, neuronophagia and multifocal gliosis in the lumbar spinal cord (18, 19, 158).

Due to the short incubation period (less than 2 months) and the high case fatality rate, no special tests should be required for live ostriches destined for export. The flock of origin should have no history of Borna disease over the previous six months and the ostriches should undergo the normal thirty day pre-embarkation quarantine in addition to a thirty-day post-arrival quarantine. There are no data to suggest transmission of the virus via meat, eggs, leather or feathers.

**Equine encephalomyelitis (Eastern, Western)**

Eastern equine encephalomyelitis (EEE) and Western equine encephalomyelitis (WEE) are caused by members of the genus *Alphavirus* of the family *Togaviridae*. Both are restricted to North, Central and South America, where the viruses cycle...
between birds and specific ornithophilic mosquitoes. The viruses cause flu-like disease sporadically in horses and humans from mid-summer to late autumn, when infection builds up in the bird populations to a level which results in spillover to other species of mosquito which feed on both birds and mammals on the margins of swampy areas. Many infections in horses and humans are inapparent, and result in a viraemia lasting up to a week, followed by long-lasting immunity (141). Clinical disease is more common in young animals, with EEE frequently being fatal in horses and WEE typically causing a subclinical or mild disease with less than 30% mortality. The viruses have been reported to cause high mortality in domestic fowl, pheasants, chukars and quail, most often caused by EEE in the eastern coastal States of the USA. After introduction by mosquitoes, the horizontal transmission within flocks is primarily by feather picking and cannibalism (99) or, in turkeys and pheasants, by the faecal-oral route (26).

Both EEE and WEE viruses have caused fatal disease in ratites including ostriches (128), with EEE having a short incubation period (20 h to 25 h), acute haemorrhagic enterocolitis and a viraemia of up to two days. Birds aged from twenty to thirty-six months and juveniles are affected principally, with a morbidity rate of up to 70% and mortality of up to 87% (99, 143). Multifocal areas of coagulative necrosis in the liver have also been described in emus with EEE (148). Infection with WEE virus has been reported in emus, which showed depression, anorexia, leg weakness, ataxia, recumbency and paralysis. Flock morbidity ranged from 10% to 50% and mortality was below 20%. Recumbent birds died within 48 h, but mildly affected birds recovered within two weeks after supportive therapy (114). In another report describing similar clinical signs, emus aged from three months to three years old were diagnosed with WEE. Morbidity ranged from 15% to 50% and mortality was 8.8% (20). Ratites are not regarded as amplifiers of EEE or WEE viruses in endemic areas and are considered accidental hosts.

Vaccines for horses and humans are safe and stimulate effective immunity, and are also used in ratites with good results in endemic areas (99, 145).

The OIE Manual of Standards for Diagnostic Tests and Vaccines recommends the following tests: virus isolation or direct demonstration of antigen and demonstration of antibody by complement fixation, HI, ELISA and plaque reduction neutralisation tests. The detection of seropositive animals would indicate vaccination or recovery and in neither case would the birds be viraemic. Therefore, if import regulations stipulate serological testing, the author recommends that seronegative as well as serostable birds (tested during the thirty-day quarantine period) be accepted. No evidence suggests that eggs or products of ratite origin could serve as vehicles of transmission of WEE or EEE viruses.

**Wesselsbron disease**

The causative agent of Wesselsbron disease is an RNA virus of the family **Flaviviridae**, and the genus **Flavivirus**. No strain variation has been reported.

Wesselsbron disease occurs only in Africa and is an acute arthropod-borne infection of sheep, cattle and goats. The virus has been isolated from several species of mosquito, especially from floodwater-breeding *Aedes* spp., and from an ixodid tick. Mechanical transmission by biting flies during epidemics seems likely. The virus causes relatively high mortality in new-born lambs and kids, subclinical infection in adult animals, occasional abortions in ewes, and congenital malformations of the central nervous system accompanied by arthrogryposis of sheep and cattle foetuses. In humans, the virus causes a non-fatal, influenza-like illness (40).

Wesselsbron disease has been reported in ratites on only one occasion. The virus was isolated from tissues of ostriches that died at four months of age. Although field data indicated very high mortality in the flock, experimental infection of six-month-old ostriches with the isolate failed to cause illness or death. Therefore, as none of the ostriches or offspring were affected, the role played by the virus in the deaths of these ostriches remains unexplained. A slaughterhouse survey of apparently healthy ostriches indicated that antibodies to Wesselsbron disease virus were found in approximately 50% of adult ostriches. The suspected mode of transmission of the virus was via arthropod vectors (10).

Laboratory diagnosis is performed by virus isolation (intracerebral inoculation of new-born mice, eight-day-old embryonated chicken eggs and cell culture) and serological tests (complement fixation, virus neutralisation and ELISA).

The virus does not appear to be vertically transmitted or disseminated via ostrich or ratite products. Live ratites exported to non-endemic areas should be seronegative during pre-export quarantine as the duration of viraemia of Wesselsbron virus in ostriches is currently unknown. Export should preferably be during the season of low vector activity (April to August in southern Africa).

**Infectious bursal disease**

The causative agent of infectious bursal disease is an RNA virus of the family **Birnaviridae**, genus **Avibirnavirus**. Two serotypes of the virus have been reported, designated serotype 1 and serotype 2.

All highly pathogenic strains belong to serotype 1. Chickens are the only known avian species to develop clinical disease. Although infectious bursal disease virus (IBDV) has been associated with a number of clinical conditions in turkeys, no isolates of pathogenic infectious bursal disease (IBD) strains have been obtained. Serotype 2 is widespread in turkeys and causes no clinical disease. This type has been isolated from...
chickens showing no clinical signs, and antibody to serotype 2 has been detected in some flocks of ducks. Antibodies to the IBDV group antigen have also been detected in sera of wild birds showing no clinical signs of the disease (89).

Isolation of an avibirnavirus said to be ‘identical to IBDV’ has been reported from immature ostriches in the USA in flocks experiencing high mortality (145).

Investigations into ostrich chick mortality in the UK (two cases of proventricular impaction and one case of enteritis) resulted in the isolation of IBD type 2 viruses (55). Similar birnavirus-like agents were detected on two occasions following direct electron microscopy (EM) examination of intestinal contents from 192 ostriches in the USA (126). The circumstances under which the three viruses were isolated and the prevalence of antibodies in farmed ostriches, poultry and wild birds world-wide suggest that IBD type 2 viruses are probably apathogenic for ostriches as well as for most avian species (55).

Antibodies to the IBDV group antigen have also been detected in the sera of ducks, turkeys and wild birds from various parts of the world (89), which suggests that these viruses are widely distributed in bird populations.

The IBD type 1 virus has not been reported in ostriches or ostrich products. Eggs should be sterilised by washing with virucidal disinfectant to prevent faecal contamination with NDV or other unknown viruses, and this should also inactivate any possible birnaviruses. Serological screening with the AGID test will show both type 1 and 2 antibodies and thus has limited value and should probably only be used in cases where both types are not present, e.g. in New Zealand where IBD type 2 is considered exotic (119). Once again it is stressed that no poultry should be permitted on ostrich or ratite farms, especially those with export status.

**Adenovirus infections**

The causative agent is a DNA virus of the family *Adenoviridae*, genus *Aviadenovirus*. Adenoviruses are widespread in poultry all over the world. In general, avian adenoviruses are divided into three groups. The first contains conventional group 1 fowl adenoviruses (FAV-1 to FAV-12) with FAV 1 as the type species. The second group includes adenoviruses associated with egg drop syndrome 1976 (EDS 76), with the virus ‘127’ as the type species. The third group comprises so-called type II fowl adenoviruses and includes turkey haemorrhagic enteritis (THE) virus, marble spleen disease virus of pheasants, and the avian adenosplenomegaly virus of chickens, with THE virus as the type species (75).

Adenoviruses have been implicated as the cause of wasting disease in ostriches at approximately two months of age in the USA. The clinical signs associated with these outbreaks included non-specific wasting, anorexia and depression (145).

An adenovirus was isolated from a four-month-old ostrich which died without showing any obvious disease signs. However, the precise role of the virus has not been established. Subsequent studies of this isolate showed the similarity to FAV-1 (35, 54).

The FAV-1 principally affects quail, causing a specific clinical syndrome known as quail bronchitis. The disease is highly contagious and morbidity and mortality may reach 80% to 100% (90).

Another study found six adenoviruses in young and adult ostriches, all belonging to group I. One isolate (FAV-2) was from a six-week-old ostrich with gross lesions of pancreatitis. The other five isolates (FAV-8) were either from young ostriches showing signs of diarrhoea or from apparently healthy breeding female ostriches producing eggs with shell aberrations (119). Other FAV-8 isolates have been detected in poultry in association with inclusion body hepatitis and respiratory disease in broiler chickens (75).

The FAV serotype 8 is, among other adenovirus group I serotypes, said to be implicated in natural outbreaks of inclusion body hepatitis in chickens. The disease is characterised by a sudden onset. Affected chickens die within 48 h. Morbidity is low, while mortality may reach 10% to 30% (32).

An adenovirus was isolated from ostrich chicks with liver lesions in outbreaks on ten farms in the USA (128) and was also demonstrated by EM in the liver of one ostrich chick (126). Raines and co-workers in Oklahoma reported further cases in ostrich chicks younger than two months with liver pathology (72). Clinical signs of ‘chick fading syndrome’ were produced when the isolates were inoculated orally into five test ostrich chicks (three negative controls). However, two adenovirus isolates from ostriches examined at the University of Georgia were found to be apathogenic for experimentally infected ostrich chicks (98).

Adenovirus and rotavirus particles have been found in the intestines of an emu chick with enteritis and *E. coli* septicaemia (63). Adenoviruses associated with EDS 76 or adenoviruses belonging to group II have not been isolated from ratites.

Laboratory diagnosis is based on virus isolation (host specific cell lines), immunofluorescence, haematoxylin and eosin staining and virus neutralisation tests to identify the serotype.

Adenoviruses are ubiquitous and the role of these viruses as pathogens in ostriches and other ratites has not been defined.
None of the available data suggest that live ostriches, ostrich meat or other products pose a risk of transmitting diseases caused by adenoviruses. Special control measures are therefore not indicated.

**Coronavirus infections of ostriches**

The causative agent is an RNA virus of the family *Coronaviridae* and the genus *Coronavirus*. A coronavirus has been isolated, along with a wide range of bacteria and other viruses, from young ostriches affected with an enteritis. However, the coronavirus may not have been the primary cause of the syndrome. A coronavirus-like agent has also been detected in a six-week-old rhea chick experiencing weakness, ataxia and death. The ostrich coronavirus has been suggested as a new species (50, 80, 145).

Coronaviral enteritis has also been diagnosed in ostriches in South Africa, associated with a chronic 'fading' syndrome or explosive outbreaks. The virus could not be isolated with conventional techniques, but was identified using EM (8; OVI, unpublished case reports 1993-1998).

Coronaviruses can also occur in domestic chickens (infectious bronchitis), turkeys, pheasants and pigeons, and are generally considered to be species-specific.

Only clinically healthy ostriches should qualify for export. No evidence has been found of transmission of coronaviruses via products.

**General comments**

The preceding discussions clearly indicate that in most instances, substantial knowledge concerning diseases of ostriches with international trade or public health significance is not currently available to allow national health screening for these diseases. Furthermore, most standard tests employed in the poultry industry are not validated in ostriches, and as illustrated in the case of HI tests, significant confounding effects occur in this species. Validated serological tests (e.g. ELISAs) are available as species-specific assays (e.g. indirect ELISAs against ND) or even non-species specific assays in exceptional cases (e.g. monoclonal blocking ELISA against ND). Even PCR-based investigations are limited, due to unknown pathogenesis and shedding patterns of most pathogens in ostriches. Lastly, the relative novelty of ostriches as a production species means that regulatory authorities are faced with an 'unknown factor' in terms of possible disease associations. The following suggestions should allow reasonable safety guarantees for consumers and importing countries:

a) Regulations regarding control of vegetation, rodents and ticks, designed as additional safety measures against CCHF for abattoir workers, as well as a thirty-day pre-slaughter residency ('quarantine'), should be enforced at all ostrich abattoirs in the areas were CCHF is endemic (Africa, Middle East, Asia, southern and eastern Europe).

b) Abattoirs approved for ostrich meat exports should have a functional hazard analysis critical control point (HACCP) action plan that is regularly evaluated by the Veterinary Services of the country concerned, with particular reference to control of Salmonella.

c) If importation of live ostriches is to be undertaken, pre-embarkation quarantine should allow the use of sentinel chickens held in cages with drinking water artificially contaminated with faeces from the ostriches in question. This is preferable to reliance on a battery of unvalidated poultry serological tests or virus isolation attempts. These tests should instead be used on the sentinel chickens, a species in which the tests are validated.

d) Fertile eggs should be disinfected with a recognised virucidal agent before packing for export.

e) Day-old chicks should originate from closed flocks registered for this purpose. As breeder ostriches do not tolerate regular disturbance or handling during the breeding season, sampling during the typical nine-month laying period would be impractical. Unvaccinated ostriches older than six months can be used as sentinels in breeder camps and the sera of these sentinels tested against viruses such as avian influenza or NDV on a three-monthly basis. Alternatively, or in addition, faecal material or drinking water from the breeder camps could be used to contaminate the drinking water of a flock of caged sentinel chickens on the same premises, these chickens could then be subjected to the required test protocol.

Finally, it is of utmost importance to stress that perceived risks related to trade in ostriches and ostrich products, other raptors or any other animals, should always be evaluated against the background risk situation already existing in the importing country.

In this particular case, those disease risks already present in domesticated or wild birds, racing pigeons and those associated with migrating birds should be considered. No country or region can claim zero risk against any pathogen over an extended period, and as one risk can only be compared to another, it is suggested that conflicting interpretations of data between trading partners be resolved with the aid of sophisticated computer programs developed for risk analysis in situations such as these.
Les maladies des autruches

D.J. Verwoerd

Résumé

Les connaissances scientifiques sur les maladies des autruches sont incomplètes et très fragmentaires, et dans la plupart des cas on ignore tout des aspects techniques des épreuves utilisables pour le diagnostic et/ou le dépistage de ces maladies. *Salmonella Typhimurium* est commune à plusieurs espèces d’autruches ; elle est à l’origine d’une mortalité chez les jeunes de moins de trois mois dans les élevages, mais elle est rarement décelée chez ceux âgés de plus de six mois ou chez les autruches de douze à quatorze mois destinées à l’abattage en Afrique australe. 

*Campylobacter jejuni* et *Chlamydia psittaci* sont occasionnellement observés, surtout chez les jeunes autruches, mais le diagnostic reste dans les deux cas difficile. La fièvre hémorragique de Crimée-Congo peut être transmise aux animaux domestiques, y compris aux autruches, principalement par des tiques appartenant au genre *Hyalomma*. Chez les autruches, l’infection est asymptomatique et la virémie persiste environ quatre jours. Aucun cas d’encéphalopathie spongiforme n’a été rapporté de façon certaine chez les autruches et les cas de fièvre charbonneuse ont été rares au cours des dernières décennies, alors que cette infection était, semble-t-il, une importante cause de mortalité il y a environ cent ans en Afrique du Sud. *Salmonella Gallinarum* et *S. Pullorum* n’ont pas été décelés chez les autruches, et l’infection due à *Pasteurella multocida* cède aisément à l’antibiothérapie. Quant aux *Mycoplasma spp.*, ils sont régulièrement associés à une infection de l’appareil respiratoire supérieur avec complications dues à des agents bactériens opportunistes. Les autruches de tous âges sont sensibles au virus vélogène de la maladie de Newcastle, mais des vaccins à virus inactif standard (souche La Sota) peuvent leur conférer une protection pendant six mois. La période de virémie chez les autruches vaccinées destinées à l’abattoir est de neuf à onze jours et aucun signe n’indique l’existence d’un portage ou la présence du virus dans les muscles ou dans d’autres tissus au-delà de cette période, la réponse maximale des immunoglobulines G étant atteinte le quatorzième jour après l’infection. La réaction d’inhibition de l’hémagglutination est nettement moins sensible et moins spécifique que les épreuves immuno-enzymatiques. Les écouvillonnages cloacaux et choanaux utilisés pour le dépistage sérologique direct chez des autruches cliniquement atteintes (infection naturelle et expérimentale) n’ont pas permis de déceler le virus de la maladie de Newcastle. 

Tous les isolats de virus influenza aviaire retrouvés chez des autruches se sont avérés non pathogènes pour les volailles, même les sous-types H5 et H7. Certaines souches de ces derniers sous-types ont été associées à la mortalité de jeunes autruches dans des foyers localisés, survenus dans des conditions climatiques difficiles et au contact d’oiseaux d’eau sauvages. La maladie de Borna provoque un syndrome nerveux chez les jeunes autruches, mais à ce jour, elle n’a été signalée qu’en Israël. L’encéphalomyélite équine de l’Est et de l’Ouest est mortelle chez les autruches et les autres ratites, le taux de mortalité variant de moins de 20 % à plus de 80 % dans les élevages atteints. Ces maladies sont présentes en Amérique du Nord, du Centre et du Sud, où elles sont transmises par des moustiques ornithophiles. Les vaccins destinés aux chevaux et à l’homme sont apparemment efficaces et sans danger pour les ratites. La maladie de Wesselsbron, la bursite infectieuse (type 2) et les infections dues à des adénovirus et à des *Coronavirus* ont été signalées chez les autruches, mais on ignore encore l’importance exacte de ces maladies.

En raison de l’insuffisance des données sur les maladies des autruches et du fait que la plupart des épreuves destinées aux volailles n’ont pas été validées pour ce groupe très particulier d’oiseaux, la stricte observation d’une quarantaine de trente jours avant l’abattage est fortement recommandée, tandis que les
autruches vivantes et les œufs fécondés destinés à l'exportation doivent être soumis à une surveillance basée sur la mise en place de poulets et/ou de jeunes autruches sentinelles.

**Mots-clés**

---

**Enfermedades del avestruz**

D.J. Verwoerd

**Resumen**

El conocimiento científico que se tiene de las enfermedades del avestruz es incompleto y muy fragmentario, con grandes y frecuentes lagunas sobre determinados aspectos técnicos de las pruebas para el diagnóstico y/o la detección masiva de estas enfermedades. En el África meridional, *Salmonella Typhimurium*, microorganismo asiduo de las instalaciones que reúnen a distintas especies, es causa de mortalidad sobre todo entre polluelos de menos de tres meses de explotaciones industriales, pero está mucho menos extendido en polluelos mayores de seis meses o ejemplares asaderos de entre doce y catorce meses de edad. Aunque ocasionalmente se detecta la presencia de *Campylobacter jejuni* y *Chlamydia psittaci*, principalmente en avestruces jóvenes, el diagnóstico de ambos microorganismos sigue planteando problemas. La fiebre hemorrágica de Crimea-Congo se transmite a los animales domésticos – entre ellos el avestruz – sobre todo a través de garrapatas del género *Hyalomma*. En el avestruz, esta enfermedad no induce ningún síntoma clínico durante la fase de viremia, que dura aproximadamente cuatro días. Por otra parte, no hay informes fidedignos que demuestren la presencia de encefalopatía espongiforme en el avestruz. En cuanto al carbunco bacteriano, y aunque en la era moderna se haya comunicado muy rara vez su presencia en el avestruz, esta enfermedad constituía, según los informes, una causa importante de muerte en Sudáfrica hace cerca de 100 años. *Salmonella Gallinarum* y *S. Pullorum* no se han detectado nunca en avestruces, y *Pasteurella multocida* es fácil de controlar con antibióticos. Regularmente se detectan especies de *Mycoplasma* asociadas a un síndrome de las vías respiratorias superiores complicado por infecciones oportunistas de patógenos bacterianos. Aunque el avestruz es susceptible a la cepa velogénica del virus de la enfermedad de Newcastle a cualquier edad, las vacunas inactivadas La Sota, de uso estandarizado en aves de corral, pueden inducir inmunidad protectora durante más de seis meses. En avestruces de engorde vacunadas, la fase vírica dura entre nueve y once días, y una vez transcurrido este periodo (con un pico de inmunoglobulina G a los catorce días de la infección) no queda en la carne ni en ningún otro tejido el menor indicio de la presencia del virus o de la condición de ejemplar portador. Las pruebas de inhibición de la hemoaglutinación presentan una sensibilidad y una especificidad significativamente menores que los ensayos inmunoenzimáticos. El estudio virológico directo de muestras cloacales y coanales de ejemplares afectados clínicamente (de forma tanto natural como experimental) no permitió detectar la presencia del virus de la enfermedad de Newcastle. Todas las cepas de influenza aviar aisladas en avestruces han resultado no patogénicas para las aves de corral, comprendidos los subtipos H5 y H7. Algunas cepas de este último subtipo parecen relacionadas con brotes de mortalidad de pollitos de avestruz, de carácter bastante localizado, sobrevenidos en épocas de mal tiempo y después
de un estrecho contacto con aves salvajes (anseriformes). La enfermedad de Borna provoca un síndrome nervioso en los pollitos de avestruz, aunque hasta la fecha sólo se ha comunicado su presencia en Israel. La encefalomieliitis equina (del Este o del Oeste) provoca una enfermedad fatal en avestruces y otras aves corredoras, con tasas de mortalidad que oscilan entre menos de un 20% y más de un 80% en las bandadas afectadas. Estas enfermedades están presentes en América del Norte, Central y del Sur, donde se encuentra el mosquito ornitofílico que ejerce de vector asociado a la enfermedad. La aplicación de las vacunas equinas y humanas a las aves corredoras parece segura y eficaz. Por último, también se han descrito en avestruces la enfermedad de Wesselsbron, la bursitis infecciosa (de tipo 2) y las infecciones por adenovirus y coronavirus, aunque no está claro hasta qué punto tienen una incidencia significativa. Teniendo en cuenta la escasez de datos sobre las enfermedades del avestruz y el hecho de que muchas de las pruebas aplicadas a las aves de corral no se han validado para este singular grupo de aves, resulta muy aconsejable observar una estricta cuarentena de treinta días antes del sacrificio, y utilizar además polluelos y/o avestruces jóvenes a modo de centinelas para controlar las exportaciones de ejemplares vivos y huevos fértils.

**Palabras clave**

**References**


