Avian rhinotracheitis

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Summary

Turkey rhinotracheitis, now commonly termed avian pneumovirus (APV) infection, is associated with serious welfare and economic problems in susceptible populations of turkeys and probably also of chickens. The infection principally affects the upper respiratory tract, although egg-laying performance may also be affected in breeding turkeys. Secondary infections exacerbate the effects of the primary virus infection. The virus persists for only a short time both in the host and in the environment and is not known to be transmitted via the egg. Highly effective vaccines are available to control APV infections, and hence good biosecurity and careful use of these vaccines should enable infection to be controlled and spread restricted. Diagnosis and surveillance are normally performed serologically using enzyme-linked immunosorbent assays (ELISAs). Several different ELISA kits are available commercially, but these give variable results and are not wholly satisfactory since interpretation of results is difficult.

Keywords


Introduction

Avian rhinotracheitis, initially named turkey rhinotracheitis (TRT), is now more frequently referred to as avian pneumovirus (APV) infection. The disease is an upper respiratory tract infection of turkeys, caused by a pneumovirus. The initial virus infection is commonly complicated by secondary bacterial infection. This results in high morbidity and variable, but often high, mortality. In turkey hens, the virus can also induce substantial reductions in egg production. Infection of susceptible birds of any age can result in serious economic losses (79, 90).

The same virus can also infect chickens, but in this species, the role of the virus as a primary pathogen is less clearly defined, although it is commonly associated with swollen head syndrome (SHS) in breeder chickens (73).

The disease

Turkeys

In growing turkeys, infection of the respiratory tract with APV can occur from a very young age. The disease is characterised by sneezing, tracheal rales, nasal and ocular discharge and swollen infraorbital sinuses (9). Conjunctivitis has also been reported (90). The nasal discharge, whilst initially clear and watery, often becomes thicker and mucopurulent. The ocular discharge is frequently frothy. Coughing and head shaking are commonly observed. Onset of clinical signs is rapid and the infection may spread through a house of turkeys within 12 h to 24 h (90). In an uncomplicated infection, recovery is rapid and the birds will appear normal again within ten to fourteen days (90). In fully susceptible flocks, mortality may reach 15% (79). In situations where farm management is poor, or where secondary infection with bacteria (22) or Mycoplasma gallisepticum (68) occurs, airsacculitis, pericarditis, pneumonia and pericarditis may be prolonged, with associated morbidity and mortality. Dual infection with bacteria which increases the severity of clinical signs may not influence the duration or the extent of virus replication (22). However, under experimental conditions, the presence of APV can exacerbate an M. gallisepticum infection, rather than the reverse (68). Similarly, field observations have suggested that APV may enhance Ornithobacterium rhinotracheale infection (39).

In vivo replication

Replication of APV in growing turkeys appears to be limited to the respiratory tract (particularly the upper respiratory tract), and to be of short duration. Both attenuated and virulent strains of APV replicate to highest titre in upper respiratory tract tissues of young turkeys, but only for approximately ten days post inoculation (dpi) (22, 106). Some replication also
caused by avian pneumovirus

Protection of laying turkeys against a decline in egg production

Fig. 1

nine, one hen had a folded shell membrane in the magnum
with occasional deposits of solid yolk in the abdomen. On day
regression, with follicles showing some shrinkage, were
usually normal in appearance, early stage ovarian
scattered throughout the oviduct between two and twelve dpi,
in experimentally-infected thirty-week-old female turkeys
poor, as a result of an increased incidence of thin-shelled eggs.

In laying turkeys, the respiratory infection is normally much
less severe. However, APV infection may result in a substantial
reduction in egg production (Fig. 1). This has been
demonstrated experimentally (26) and under field conditions,
where reductions in egg production of up to 40% have been
reported (1, 90). Up to three weeks are required for recovery,
and the quality of the eggs laid during this period may be
poor, as a result of an increased incidence of thin-shelled eggs.

In a sequential study performed with a virulent strain of TRT
in experimentally-infected thirty-week-old female turkeys
(47), whitish masses of inspissated albumen were found
in the trachea, but other tissues, including conjunctiva and
lung were virtually normal. These findings led to the
suggestion that turbinate pathology may be a suitable marker
for the evaluation of APV infection (62).

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with occasional deposits of solid yolk in the abdomen. On day
nine, one hen had a folded shell membrane in the magnum
and a second bird had egg peritonitis. Although the ovaries
were usually normal in appearance, early stage ovarian
regression, with follicles showing some shrinkage, was
observed up to twelve dpi. Virus was isolated from the
magnum and vagina, but at nine dpi only, and then only after
three in vitro passages. Viral antigen was also detected by
indirect immunofluorescence in uterus epithelium at seven
dpi and throughout the oviduct at nine dpi. This finding was
confirmed by immunoperoxidase (IP) staining (75). However,
whether oviduct tissue is actually susceptible to APV infection
remains unclear. Using explants of oviduct, the intrinsic
susceptibility of oviduct epithelium has been demonstrated,
but in vivo, virus replication in the oviduct could not be
demonstrated, even after intravenous inoculation (52).

Chickens

In chickens, the role of APV as a primary pathogen is less well
established, although the virus is commonly associated with
SHS (3, 11, 38, 69, 73, 80). The same virus that causes disease
in turkeys is known to infect chickens and induce a specific
antibody response (101), although not always in association
with any disease (19). However, the virus has been isolated
from diseased chickens of all ages (11, 36, 48, 61, 81, 91).
Experimental infection of chickens with APV has also been
reported (13, 24, 46, 62). The dissemination of APV in the
chicken has been shown to be similar, and to be of equally
short duration, to that in growing turkeys. Using virus
isolation and immunochemical techniques, virus has been
detected only in respiratory tract tissues, with the largest
amounts observed in turbinate tissue (13, 46, 62). Attempts to
demonstrate APV antigen in the reproductive tract of adult
broiler breeders using immunochemical techniques were not
successful (62).

In broilers, APV may not be a primary pathogen, but may be
involved with other agents in a complex respiratory disease
syndrome. The benefit derived from using TRT vaccines in
broilers (see below) provides strong circumstantial evidence
for the involvement of this virus in disease in broilers. In
commercial layers, some evidence suggests that infection with
APVs may affect the quality or colour of eggs.

Swollen head syndrome

In breeders, strong evidence exists to suggest that APV may be
one of the aetiological agents of SHS. Furthermore, APV
antibodies have been found in chickens with SHS (101). The
syndrome is characterised by respiratory disease, apathy,
swelling of infraorbital sinuses and unilateral or bilateral facial
swelling, extending over the head. These signs are frequently
followed by cerebral disorientation, torticollis and
opisthotonos (38, 73, 80). Although mortality does not
usually exceed 1% to 2%, morbidity may reach 10%, and egg
production is frequently affected. However, APV is not the
only agent that has been associated with SHS in chickens. In
the first report of SHS, infectious bronchitis virus (IBV) was
isolated (66). More recently, IBV together with Escherichia
coli, was isolated from cases of SHS in California, United
States of America (USA) (29). Thus, bacteria of different
species, including E. coli, are also involved in SHS; E. coli

Fig. 1

Protection of laying turkeys against a decline in egg production
caused by avian pneumovirus
alone has been demonstrated to be capable of causing the condition under experimental conditions (67).

Aetiology

The causal agent of avian rhinotracheitis infection is a pneumovirus, the first APV to have been described. The virus has been identified both morphologically (10, 34, 59, 100) and by molecular sequencing (14). Sequence data for the F (72, 102), G (50, 55), L (85), M (84, 104), M2 (103), N (54), P (56) and SH (55) genes have been published. Avian pneumovirus has no non-structural NS1 and NS2 proteins (55) and the gene order (3’-N-P-M-F-M2-SH-G-L-5’) is different from that of mammalian pneumoviruses (3’-NS1-NS2-N-P-M-SH-G-F-M2-L-5’) (55, 104). Avian pneumovirus has therefore recently been classified as the type species for the new genus Metapneumovirus (82).

Strain variation

For many years only one serotype of APV was recognised, within which existed two subgroups, designated A and B. Both subgroups are found in the United Kingdom (UK) and mainland Europe (7, 70) and have been shown to coexist (70, 105). The two subgroups were differentiated on the basis of molecular sequencing of the G glycoprotein (50) and neutralisation tests using monoclonal antibodies (MAbs) that also recognise the G glycoprotein (17, 23). Complete neutralisation occurs between isolates of the two subgroups (23). Recently, an APV was isolated from turkeys in the States of Colorado and Minnesota, USA (89). This virus, designated the Colorado isolate, is morphologically similar to the viruses described previously. However, the Colorado isolate is neutralised by neither the above-mentioned MAbs (23) nor by monospecific antisera raised to A or B subgroup strains, nor does monospecific antiserum raised to the Colorado isolate neutralise A or B subgroup strains (27). Partial neutralisation of the Colorado isolate by a hyperimmune subgroup A antiserum has been reported (27). Sequence data for the matrix protein gene of the Colorado isolate show the gene to be distinct from the earlier APVs (88). No sequence data for other genes of the Colorado isolate have been published, and whether this virus represents the first isolate of a second serotype of APV remains to be established. Recent studies have suggested that an APV type, different from the A and B subgroups and also from the Colorado isolate, may exist in Northern Ireland of a virus related to, but different from, TRT virus (76), underline the need for awareness that new antigenic or genotypic types of APV may continue to emerge.

Strains of subgroups A and B are known to infect both turkeys and chickens. However, strains with different tropism for the two species may exist (11, 24). Experimental studies have shown that whilst an APV isolate derived from turkeys replicated equally well in the two species and caused clinical signs of similar severity, an isolate derived from chickens replicated to higher titre in chickens and caused more substantial clinical signs in that species (24). Further work is required to substantiate these findings. However, the suggestion that APV strains may have different species tropism might help to explain the difficulty in elucidating the pathogenesis of APV infections in the chicken and the apparent lack of spread of the Colorado isolate from turkeys to chickens in the USA (2).

Epidemiology

Given that APV is an enveloped ribonucleic acid virus, it is likely to be destroyed rapidly outside the host (45). The restriction of virus replication to the respiratory tract suggests that transmission is most likely to be airborne (32), although faecal shedding cannot be dismissed. The short persistence time of the virus in the bird suggests that virus is likely to be shed for only a few days after infection. Current evidence suggests that no carrier state exists and that APV is not latent in the bird. Although APV has been detected in the oviduct of laying turkeys (47), this was reported on one occasion only and circumstantial evidence from the field suggests that egg transmission of APV is unlikely to occur (G.P. Wilding, personal communication). However, the importance of rigorous farm hygiene and biosecurity in minimising the risk of APV transmission by, for example, personnel, vehicles and egg trays, must always be remembered (90).

The speed with which the virus spread within and between turkey flocks in eastern England following the initial outbreak in that country suggests that, although wire netting may delay the spread of infection between pens (90), the virus is highly infectious. This suggestion is supported by the fact that the disease spread throughout England within six months of the initial outbreak (79, 90). However, turkeys in Scotland, where the stocking density is much lower, remained free from evidence of infection for several years (6). Furthermore, data on the ability of APV to spread from infected to uninfected poults housed experimentally in the same air space are conflicting (22, 32). The ease of spread between birds housed in close proximity, together with probable poor or slow spread over greater distances is confirmed by the current situation in the USA. A high incidence of infection has been reported in the turkey population in Minnesota (2), but infection does not appear to have spread elsewhere in the USA. As with other pneumoviruses, direct contact between individuals is probably important for transmission.
Geographical distribution

The first report of APV infection was from South Africa in the late 1970s (9). Shortly afterwards, the disease was reported in France (32) and the UK, where the causal agent was first characterised (59, 97, 100). Subsequently, infection was soon reported in other countries of Europe (23, 40, 65). Frequently based on serological evidence alone, APV infections have now been reported in Israel (96), Morocco (30), Zimbabwe (12), Taipei China (58), Japan (78, 91, 92), Brazil (3), Central America (45) and the USA (89). This suggests an almost global distribution of the virus. Whilst no data are available from some geographical areas, the only two countries that have been reported to be free from infection are Australia (8) and Canada (43).

Susceptible species

With the exception of turkeys and chickens, the only avian species known to support the replication of APVs are pheasants, Muscovy ducks (Cairina moschata) (94) and guinea-fowl (35). In the latter species, natural clinical infection has been suggested in the Netherlands (57). Geese, pigeons and most ducks are apparently refractory to infection (35). Avian pneumovirus antibodies have been reported in ostrich in Zimbabwe (12) and in seagulls in the Baltic (44). Circumstantial evidence suggests a role for migratory birds in the global transmission of this virus, although no data are available to substantiate this. The initial spread of the virus from southern Africa to Europe, followed by an appearance on the east coast of the UK (90) could be explained by this hypothesis. Likewise, the sudden appearance of infection in Minnesota could be associated with the large number of migratory birds commonly found on the lakes in that State (2).

Diagnosis

Clinical

In turkeys, although the clinical signs observed are similar to those caused by a number of different agents, diagnosis on the basis of clinical signs is believed to be possible (90). However, diagnosis of APV infections in chickens solely on the basis of clinical signs is not recommended, because of the uncertainty of the exact role of APV in disease in that species and because of the evidence that other viruses or bacteria may also cause SHS.

Virus isolation

Two substrates have been used successfully for the primary isolation of APV from field material, namely: embryonated eggs inoculated via the yolk sac (10, 27) and chicken or turkey embryo tracheal organ cultures (TOC) (59, 97, 100). In addition, the successful use of chick embryo rough cells has been reported (41). When inoculated onto TOCs, the A and B subgroup strains cause ciliostasis after approximately six to ten days, whilst peak virus titres are usually achieved approximately three to five dpi (22). For this reason, field material is commonly given up to four blind passages in TOCs at three- to four-day intervals, whilst a proportion of the cultures inoculated at each passage are incubated to allow examination for ciliary activity for up to ten days. For embryo passage, six-day-old embryos are inoculated via the yolk sac and the allantoic fluid and yolk sac membrane are harvested and homogenised for passage approximately eight days later (10, 27). After two or three passages, haemorrhages may be observed on the embryos and some embryo mortality may occur. Once embryo lesions are present, the egg fluid may be inoculated onto cell cultures. A cytopathic effect (CPE) will be seen after one or two additional passages. The CPE is characterised by scattered focal areas of cell rounding, leading to syncytial formation (10).

Regardless of the system used to isolate the virus, identity as APV may be confirmed by electron microscopy (10, 32, 59), IP (13, 75), immunofluorescence (IF) (47) or immunogold (77) techniques. However, the IP assay has been demonstrated to detect APV antigen rather poorly in chicken tissues (13), suggesting that this may not always be a sensitive alternative to virus isolation.

Once isolated in one of these systems, the virus can readily be adapted to grow on a range of cell types, including chick embryo fibroblasts (CEF) (37), chick embryo liver (CEL) cells (98), and Vero cells (10, 98). However, passage in cell culture rapidly leads to the attenuation of the virus (98). In contrast, the virus has been passaged at least 100 times in TOC without any loss of virulence (98). Although the initial isolate was obtained following embryo yolk sac passage (10), the most consistently successful system for the isolation of both APV subtypes from both turkeys and chickens, has been following passage in TOCs (69), and all APVs were believed to be ciliostatic. However, the Colorado isolate of APV is not ciliostatic (27), and therefore the exclusive use of TOCs for the primary isolation of APV can no longer be recommended. For primary isolation of APV from field material, passage via the yolk sac is to be preferred, possibly supported by the use of TOC.

A major problem confronting those attempting to isolate APV from field material is that the virus is present in the bird for only a very short time after initial infection. As a result, virus isolation must be attempted at the very first sign of clinical disease. Once clinical signs are obvious, virus isolation is not likely to be successful (Fig. 2). This probably explains the relatively small number of successful isolations of APV world-wide.
During virus isolation attempts, vaccine virus may be isolated in addition to virulent field virus. The vaccine strains are derived from virulent field viruses and differentiation has been difficult. However, in the future, molecular biological techniques may help to differentiate vaccine and field viruses. Differentiation of one of the subgroup B vaccine strains from its progenitor is claimed to be possible (15), but only on the basis of three amino acid changes in the genome (two in the G and one in the F gene). However, the stability of these amino acid changes is not yet known.

**Virus detection**

Recently, the polymerase chain reaction (PCR) has been used to diagnose APV infections (28, 53, 63, 70). Swabs taken from the oesophagus can be used as the starting material (53) and the technique may give more rapid results than virus isolation. Whilst PCR is unlikely to replace existing techniques, this should provide a valuable extra diagnostic tool, once fully validated.

**Serology**

Serology is the most common method of diagnosis of APV infections in both chickens and turkeys. A number of different assays, including IF (4) or serum neutralisation (SN), have been developed. However, detection of antibodies to APV by enzyme-linked immunosorbent assay (ELISA) is by far the most commonly used method (16, 31, 37, 74). A number of commercial kits are now available, each of which is suitable for testing both turkey and chicken serum. Some of the kits use the indirect method, whilst at least one is a blocking ELISA. However, these tests do show considerable variation in sensitivity. A comparison of three ELISAs for sensitivity and specificity revealed that whilst all three demonstrated 100% specificity, differences in sensitivity were present (64). Different kits may be suitable for different objectives (60). The situation is further complicated by discrepancies in the ability of different ELISAs to detect vaccinal antibody when different APV strains are used as coating antigens (31). This should be considered when selecting the appropriate kit, and the use of homologous antigen has been suggested if attempting to detect antibodies to live vaccines by ELISA (64, 93). However, whilst the ELISA kits currently available detect antibodies to both the A and B subgroups, these tests poorly detect antibodies to the Colorado isolate of APV. The use of the homologous strain as coating antigen when testing sera by ELISA for antibodies to that APV type is therefore strongly recommended (27) (Fig. 3).

The SN test is also used to diagnose APV infections, but less commonly than the ELISA, although the two assays have similar sensitivity (5). The SN test may be performed in a variety of systems, including TOC, CEF, CEL or Vero cultures (5, 23, 32, 74, 87, 98).

**Disease surveillance**

The high specificity of the ELISA for APV antibodies makes this an ideal test for disease surveillance (64). However, the reported discrepancies in results obtained using different ELISA kits (31, 60, 64, 93), and the fact that homologous antigen is necessary when testing for antibodies to the Colorado isolate (27), indicate the wisdom of using more than one type of ELISA in situations where all known APV types must be detected with a very high sensitivity. The recent experience of the emergence of the Colorado isolate of APV in the USA (27, 89), in addition to data from France (7) indicate that further APV types, undetectable by existing assays, could emerge in the future.

**Treatment and control**

No treatment is available for APV infections, although secondary bacterial infections can be controlled by antibiotic therapy (42, 90). Good management practices and rigorous biosecurity can help prevent entry of infection in addition to minimising the effects of an APV infection.
Extensive evidence exists to demonstrate that APV infections can be prevented by vaccination (11, 20, 21, 33, 98, 99). Live attenuated vaccines are used to control APV infections in growing turkeys and broiler chickens and to prime future layers and breeders for the injection of inactivated vaccines prior to onset of lay. Care should be taken in the application of these vaccines to ensure that all birds receive an adequate dose. Maternally derived immunity does not appear to protect against infection, or prevent successful immunisation by vaccines (21, 71). The live attenuated vaccines stimulate both local immunity in the respiratory tract (51) and systemic immunity. Vaccinated birds without demonstrable antibody response (21) or turkeys which have been treated with cyclophosphamide to suppress T cell activity (49) may be protected against APV challenge, and this suggests that cell mediated immunity, rather than antibody, may be important in controlling infections with APV. The duration of immunity following vaccination has been demonstrated to be at least fourteen (21) to twenty-two weeks (99). However, reinfections may occur (105) and in many countries, revaccination of growing turkeys is common practice if the birds are reared beyond approximately ten to twelve weeks. Lifelong protection of broilers should be possible with a single vaccination. Whilst administration of an inactivated vaccine without live priming provides some protection against the effects of APV infection on egg production in breeding turkeys, the full programme of live priming followed by inactivated vaccine is necessary for complete protection of the bird (Fig. 1) (26).

A number of different vaccines produced by different companies are licensed in many countries (18). When administered carefully and correctly, these vaccines provide excellent protection and significantly reduce the prevalence of infection. Data show that excellent cross protection occurs between the A and B subgroup strains (25, 31, 93). In addition, vaccines developed against A and B strains protect not only against the Colorado isolate (Fig. 4) (27), but also against the non A, non B strains reported from France (95).

The F protein gene of APV, expressed in a fowl pox virus vector, can protect turkeys against virulent challenge (83), indicating that genetically engineered vaccines are a possibility for the future. Therefore, recombinant, or possibly deoxyribonucleic acid vaccines may conceivably be available to control APV infections, although such vaccines are not likely to be available in the near future.

Eradication

As the virus persists for a short time and spread to more remote areas appears to be slow (6), eradication of APV from an area is theoretically possible. However, the apparent highly infectious nature of the virus and the reported rapid spread between birds (90) suggest that this is likely to be difficult to achieve in practice. The only known example of eradication of APV is in the State of Colorado, USA. Infection of turkeys was reported in more than one production cycle. By the application of strict biosecurity and good management practices, the infection was eradicated from the entire State (R.K. Edson, personal communication).

Genetics

No genetic differences in susceptibility to APV infections are known to exist. However, very little work has been undertaken on this topic.

Public health implications

No evidence exists to suggest that APVs may be transmitted from poultry to humans. Therefore, no public health risks are thought to be associated with APV infections in poultry.

Risks and consequences of introducing avian pneumovirus into a susceptible population

No evidence of egg transmission of APV exists, therefore, provided hatching eggs and related materials are correctly disinfected, risks of spread of infection via eggs should be minimal. No data are available on the survival time of APV on carcasses. However, the short persistence time in the live bird (13, 22) suggests that APV is unlikely to survive for long periods of time on the dead animal. Strict biosecurity measures, combined with careful disinfection of materials to be introduced into a non-infected area should greatly minimise the risk of importing the infection. The experience of the first appearance of APV infection in England suggests that if the infection is introduced into a fully susceptible
population, the consequences will be serious (79, 90). This is likely to be the case particularly if the stocking density is high. In this case, economic losses are likely to be serious, especially in turkeys (79), but possibly also in chicken flocks. However, the experience from the USA suggests that either eradication of infection from an area (R.K. Edson, personal communication) or containment within a particular area (as appears to have been achieved in the State of Minnesota) may be possible (2). Both poultry stocking densities and local geography may play an important role in determining the ability to contain an APV infection.

Import/export risks

Given that some countries remain free from APV infection, steps must be taken to avoid introduction of the virus into these countries. The short persistence time in both chickens and turkeys and the restricted tissue distribution of the virus help to minimise the risk of transmission. This applies to introduction both via live birds or hatching eggs and to possible contamination via carcasses or processed products.

If material for export derives from fully vaccinated flocks, and hatching eggs are quarantined on arrival, no risk of introduction of APV infection into clean areas should arise. The most difficult problem to overcome regarding APV monitoring in countries that are free from infection is the lack of an ideal ELISA for serological testing (see above). The use of at least two different APV ELISAs is therefore recommended to test sera from quarantined birds.

Conclusions

Avian pneumoviruses cause significant disease in both turkey and chicken flocks and the economic and welfare implications of infection in susceptible birds are usually serious. Spread of infection is generally rapid between birds housed in close proximity, but may be slow when population density is low or when susceptible populations are well separated geographically. The method of transmission over large distances is not understood, but wild birds have been implicated in transmission of the virus. Rigorous biosecurity is crucial for controlling infection and minimising spread, and highly effective vaccines are available for prophylaxis. Although different subtypes and possibly even different serotypes exist, good cross protection occurs between all the known types.

Diagnosis and surveillance are usually based on serology using ELISAs. However, the ELISA kits currently available give variable results and interpretation is difficult. Continued work is required in this area to develop more satisfactory assays.

Rhinotrachéite aviaire

J.K.A. Cook

Résumé

La rhinotrachéite de la dinde, désormais appelée infection à pneumovirus aviaire, est à l'origine de graves problèmes sanitaires et économiques chez les populations sensibles, à savoir les dindes et probablement aussi les poules. L'infection se localise essentiellement dans l'appareil respiratoire supérieur, mais elle peut également se traduire par une chute de ponte. Des infections secondaires viennent aggraver les effets de l'infection primaire. Le virus ne se maintient chez l'hôte et dans l'environnement que pendant une courte période et on ne connaît pas de cas de transmission par les œufs. Des vaccins très efficaces sont disponibles contre les infections à pneumovirus aviaires, de sorte qu'une bonne biosécurité et une vaccination rigoureuse devraient permettre de maîtriser l'infection et de restreindre la dissémination du virus. Le diagnostic et la surveillance sérologiques sont normalement réalisés à l'aide d'épreuves...
La rinotraqueítis del pavo, conocida hoy más comúnmente como infección por pneumovirus aviar, es fuente de grandes pérdidas económicas y responsable de graves problemas sanitarios entre las poblaciones susceptibles de pavos (y probablemente también pollos). La infección afecta principalmente al tracto respiratorio superior, aunque también puede mermar el rendimiento de pavas ponedoras. Por otro lado, los efectos de la infección vírica primaria se ven amplificados por infecciones secundarias. El virus persiste poco tiempo dentro del huésped o en su entorno, y no se ha descrito su transmisión a través del huevo. Para luchar contra las infecciones que provoca este virus existen vacunas de gran eficacia, lo que debería permitir, aplicando las oportunas medidas de seguridad biológica y utilizando con tino las vacunas, controlar la infección y detener su propagación. Para actividades de diagnóstico y vigilancia suele utilizarse el análisis serológico por métodos inmunoenzimáticos (ELISA). Varios kits de pruebas ELISA se encuentran disponibles en el comercio, pero presentan resultados variables y son poco satisfactorios debido a la dificultad de interpretar dichos resultados.

**Palabras clave**


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**References**


