Avian reovirus infections

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Summary
Avian reoviruses are ubiquitous among poultry flocks. Although infection is usually present without disease, reoviruses may occasionally be involved in several disease syndromes of which viral arthritis/tenosynovitis in chickens is the most important, particularly in broiler breeds. While reoviruses have been isolated from turkeys and several other species of birds with various conditions, the presence of the virus has been conclusively linked with disease in relatively few instances. In chickens in particular, avian reoviruses with a wide spectrum of pathogenic capability have been isolated and several antigenic types exist. Diagnosis is dependent on the detection of the virus in clinical samples, although the presence of the virus does not necessarily confirm that this is the cause of the disease, except where reoviruses are detected in affected joints. Serological tests are usually difficult to interpret in view of widespread and frequently harmless reovirus infection. The principal approach to control of viral arthritis/tenosynovitis is by vaccination using attenuated vaccines in young birds, followed by inactivated preparations for breeders intended to protect chicks by maternal antibodies. Many vaccines are based on the S1133 strain isolated in the United States of America, but these may not be effective against antigenic variants.

Keywords
Avian reovirus – Chickens – Lameness – Malabsorption syndrome – Stunting syndrome – Tenosynovitis – Vaccines – Viral arthritis.

Introduction

Reovirus infections of poultry are widespread and all commercial poultry flocks probably become infected at some time during the life of the flock. An estimated 85%-90% of reoviruses isolated are non-pathogenic. However, pathogenic strains of virus exist which have been associated with a number of disease syndromes, although in many instances, the virus cannot be proved to be the cause of the disease. Viral arthritis, otherwise known as tenosynovitis, is the exception to this. Reoviruses have been implicated in the stunting/malabsorption syndrome, although current evidence does not suggest that these viruses are the main cause. The importance of reovirus infections throughout the world varies widely from region to region. The reasons for this are unclear, but probably relate to the density of broiler-type chickens, relative isolation of the stock geographically and the prevalence of pathogenic strains of reovirus.

Description of the diseases

Reoviruses are involved in a variety of disease conditions in domestic poultry of which the most important is viral arthritis/tenosynovitis in chickens, where the cause-and-effect relationship is well established (42, 55, 84). Viral arthritis/tenosynovitis is predominantly a disease of meat-type chickens (broilers) and is an important cause of leg weakness. The main lesion is a swelling of one or both hock (tibiotarsal-tarsometatarsal) joints, the main load-bearing joint in the bird, causing acute lameness. The condition is rare in birds of less than four to five weeks of age and is commonly seen up to sixteen weeks of age, with a peak incidence at approximately seven weeks. Occasionally, broiler breeders at peak production are affected. Morbidity is variable but usually below 10% and mortality is low. Affected joints are swollen and inflamed and in the most severe cases, rupture of the gastrocnemius tendon and erosion of the articular cartilage

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occur. Where both joints are severely affected, the bird is immobilised. Occasionally, one or more digital flexor tendons are ruptured. Rupture of the gastrocnemius is accompanied by haemorrhage which in turn causes green discolouration of the skin at the joint.

Economic losses from viral arthritis/tenosynovitis are due to poor growth and feed conversion, mainly through inability of lame birds to reach feed, deaths through trampling by healthy birds and downgrading of carcasses at slaughter due to the unsightly appearance of affected hock joints.

Avian reoviruses have also been associated with other disease conditions in chickens where the role of the virus is less clear and indeed sometimes tenuous. These include enteric problems such as cloacal pasting and mortality (13), ulcerative enteritis (46), enteric disease (13), respiratory disease (16, 76), inclusion body hepatitis (53), increased mortality and heart lesions in young broilers (7), sudden deaths in young broilers associated with lesions in the heart, kidney and liver (6) and the variously named runting/malabsorption/brittle bone disease in young broilers (20, 70, 72, 96, 99). Recently, sudden deaths have been reported in young broilers in Poland. The disease was characterised by liver lesions, from which a reovirus was isolated which could reproduce the disease experimentally (Z. Minta, personal communication).

Reoviruses have also been isolated from turkeys with tenosynovitis (48, 71), although the relationship between the virus and the disease seems less clear in this case. Al-Afaleq and Jones could find no evidence that reoviruses isolated from joints in chicks and poults caused tenosynovitis in poults although all the viruses caused tenosynovitis in chicks (2). Other isolations of reovirus from the intestinal tract of turkeys have produced inconclusive evidence that these viruses are primary causes of disease (71).

Muscovy ducks (*Cairina moschata*) may be affected by reoviruses which cause high morbidity and mortality, with necrotic foci in the liver, spleen and kidneys (56). Other avian species from which reoviruses have been isolated include African green parrots (*Psittacus erithacus*) with subcutaneous haemorrhages, necrotic lesions in the liver, bone marrow, airsacculitis and epicarditis (23), normal mallards (*Anas platyrhynchos*) (52), pigeons with diarrhoea and other exotic species (21), and American woodcocks (*Scolopax minor*) in which mortality was associated with a generalised infection and emaciation (12). The relationship of these isolates with the disease conditions is unknown. A strain isolated from a wedge-tailed eagle (*Aquila audax*) in a zoo, and others from ducks were found to produce histological changes in the joints of specific-pathogen-free (SPF) chicks (33). Although avian reoviruses may be transmissible between avian species, the importance of wild birds as reservoirs of infection has never been demonstrated.

Aetiological agent

A recent review of the molecular virology of avian reoviruses can be found in Kaleta and Heffels-Redmann (39). Avian reoviruses belong to the genus *Orthoreovirus*, in the family *Reoviridae*. Virus particles measure 70 nm to 80 nm, are non-enveloped and have icosahedral symmetry with a double-shelled arrangement of surface protein. The virus contains double-stranded ribonucleic acid which has ten segments. The genome can be separated into three size classes, namely: L (large), M (medium) and S (small). Similarly, proteins encoded by the genome also fall into three size classes, as follows: λ (large), μ (medium) or σ (small). Of eleven proteins, nine are structural ($\lambda1$, $\lambda2$, $\lambda3$, $\mu1$, $\mu2/\mu2C$, $\sigma1$, $\sigma2$ and $\sigma3$) and two nonstructural (μNS and σNS). Protein coding assignments of all ten genome segments of strain S1133 have been determined (98).

In common with mammalian reoviruses, the electrophoretic migration patterns of the genomic segments of individual avian reovirus isolates exhibit considerable polymorphism. Despite the similarities, avian reoviruses differ from mammalian counterparts in the lack of haemagglutinating activity, the ability to induce cell fusion and in the ability to induce pathological conditions in chickens (80).

Strains of avian reoviruses have been differentiated by cross neutralisation tests conducted in eggs or cell culture (40). The strain S1133 isolated in the USA is the basis of many commercial vaccines and appears widespread throughout the world, although many regional variants exist. Recently, strains have been differentiated using the polymerase chain reaction (PCR) and restriction fragment length polymorphism (RFLP).

Avian reoviruses are stable between pH 3.0 and pH 9.0. Ambient temperatures favour the survival of these viruses which are inactivated at 56°C in less than one hour. A study of the survivability of avian reoviruses on common materials found that the virus can survive for up to ten days on feathers, wood shavings, glass, rubber and galvanised metal, and for ten weeks in water, with limited effect on infectivity (C.E. Savage and R.C. Jones, unpublished findings). Earlier work reported that avian reoviruses were resistant to proteolytic enzymes, however Al-Afaleq and Jones described a strain from a turkey joint which was sensitive to trypsin (4), and other strains have also been demonstrated to have this property (38).

Avian reoviruses are relatively resistant to certain disinfectants. For example, one strain survived 2% formaldehyde at 4°C (62), another was only partially inactivated by 2% phenol after 24 h at room temperature, but 100% ethyl alcohol was effective (76).

The viruses may be cultivated in embryonating chicken eggs, where inoculation into the yolk sac after six days of incubation

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causes death accompanied by haemorrhaging of the embryos and the appearance of yellowish-green foci on the liver (55). Several primary chick embryo or chicken cell cultures are susceptible to avian reoviruses, such as fibroblasts, lung, liver and kidney of chick embryo, and chick kidney cells. Of these, chick embryo liver cells have been found to be the most sensitive for primary isolation from clinical material (24, 52). The typical cytopathic effect of avian reoviruses is the production of syncytia.

Epidemiology

Both vertical and horizontal transmission of avian reoviruses are recognised. Egg transmission has been confirmed after experimental infection (5, 60, 92), but the rate of transmission is probably very low in nature. Congenitally infected chicks are thought to act as a nucleus of infection for the rest of the hatch, since most are likely to become infected via the faecal-oral route (30), although infection via the respiratory tract may also occur. In addition, reoviruses may enter broken skin of the feet of chicks from the litter and become established in the hock joints (3).

Avian reovirus has been found to persist in the tissues of chickens for many weeks. Kerr and Olson recovered virus from the spleen of chickens inoculated 285 days previously (41), while Jones and Onunkwo found that an arthrotropic virus was present in the hock joints for at least thirteen weeks after experimental infection (30). Whether virus which persists in the joints or elsewhere may be reactivated by sexual maturity or some other biological trigger has not been investigated, but this might explain the occasional reisolation of virus from affected joints of broiler breeders (29), despite evidence that older birds are normally resistant to infection (see below).

Although predominantly a disease of the heavy meat-type bird, reoviral arthritis has been reported occasionally in light egg layers (86). Jones and Kibenge provided experimental evidence that broiler chicks were more susceptible to reovirus arthritis than SPF light hybrids or commercial White Leghorns (34).

Resistance to reovirus infection in chickens is clearly age-linked. Jones and Georgiou demonstrated that chicks infected at day-old were more susceptible to experimentally-induced tenosynovitis than others infected at two weeks or older (32). In chicks infected at day-old, higher intestinal virus titres and more severe joint lesions developed than in those infected when older. Similar results were observed by others (64, 81, 82).

Infectious agents which enhance the effects of reovirus pathogenesis in the joints of the chicken include *Mycoplasma synoviae* (8), *Staphylococcus aureus* (42), infectious bursal disease virus (65) and chicken anaemia virus (54), although in the latter case, synergism may not occur with all reovirus strains. In turkey poults, no evidence of synergism was found after inoculation with *M. synoviae* and an arthrotropic reovirus (1).

Immunopathogenesis

Virus distribution

Although avian reoviruses have been associated with several disease conditions in poultry, most effort has been concentrated on the study of reovirus-associated arthritis, indicating the greater importance of this condition.

Vertical transmission of reoviruses usually occurs at a low rate (5, 60), and most chicks become infected at an early age via the oral or occasionally the respiratory route, from the small nucleus of congenitally infected hatch-mates or from the environment. Experimental infection of adult SPF hens via the nasal, tracheal or oesophageal routes, showed distribution of virus to all areas of the respiratory, enteric and reproductive tracts and the tendon of the hock joints (61). The importance of viraemia was confirmed by a study in young chicks (43), where following oral infection, virus was recovered from the plasma, erythrocyte and mononuclear cell fractions of blood within 30 h. By three to five days, virus had been distributed throughout the body. Despite this widespread tissue dissemination, the principal site of virus replication is the enteric tract (43).

A study of the early pathogenesis of an arthrotropic reovirus in day-old SPF chicks using virus isolation, immuno-fluorescence, immunoperoxidase and electron microscopy showed that the epithelial cells of the small intestine and the bursa of Fabricius are the main sites of primary infection and portal of entry of the virus which rapidly spreads to other organs within 24 h to 48 h of infection (37). The site where virus replication has the most serious consequences is the tibiotarsal-tarsometatarsal (hock) joint (34, 37, 85, 100). At this site, the virus replication and perhaps long-term persistence induce a series of processes which are poorly understood, leading to joint damage and in the most severe cases, tendon rupture.

Although some reports strongly suggest that most, if not all, avian reoviruses have arthritogenic potential for the hock joint tissues (33, 85), several experimental reports indicate a wide spectrum of ability among virus strains to cause pathological changes (11, 22, 33, 44, 82, 91).

Experimental studies suggest that another target organ is the liver, since chicks given high doses of virus by the oral route die within ten days due to hepatitis (33).

The genetic determinants of avian reovirus pathogenesis have been investigated using reassortant analysis (68). Meanger *et al.* have recently asserted that the tissue tropism of avian reovirus is genetically determined and related to mutations in the S1 segment of the genome (59).

Immune responses

Kibenge *et al.* examined the effects of surgical and chemical immunosuppression on reovirus-induced reovirus tenosynovitis (45). Chicks infected with reovirus after thymectomy and bursal depletion by cyclophosphamide treatment showed a higher mortality rate, with longer virus persistence than those treated with cyclophosphamide only, or those bursectomised or thymectomised. The authors concluded that recovery from reovirus infection probably involves both B- and T-cell systems, with the B-cell system being more important in protection.

Humoral antibodies

Circulating antibodies can be demonstrated in the sera of birds infected with avian reoviruses by tests such as agar gel immunodiffusion (AGID) (69), virus neutralisation (VN) (17, 40), indirect immunofluorescence (IIF) (26) and enzyme-linked immunosorbent assay (ELISA) (89, 27). Agar gel immunodiffusion, IIF and ELISA all detect group antigens, while VN detects type-specific antibody which permits differentiation between antigenically different strains of virus. Shapouri *et al.* confirmed the importance of humoral immunity in immunisation-challenge experiments by using an *Escherichia coli*-expressed sigma-3 protein (87).

Maternal antibodies

Maternal antibodies to avian reoviruses are effective in protecting chicks infected at day-old from developing microscopic lesions of tenosynovitis after homologous virus challenge (93). The protective effect conferred by maternal antibodies is the basis of breeder vaccination (as discussed below).

Local antibodies

The effects of age at infection, route of infection and virus strain on the appearance of reovirus-specific immunoglobulin A (IgA) and IgG were investigated by Mukiibi-Muka and Jones (67). Intestinal IgA developed in the gut in chicks infected orally at seven and twenty-one days of age but not at day-old. This coincided with findings of reduced intestinal virus titres with increased age at infection. Following subcutaneous infection, only those birds infected at three weeks produced intestinal IgA. Immunoglobulin G in serum but not in the gut, was elicited similarly in all age groups inoculated by either route. A trypsin-sensitive reovirus, which failed to replicate in the gut, elicited substantial serum IgG but no intestinal IgA at any age. An immunisation-challenge study suggested a protective role for intestinal IgA (66). These results indicate that the age of chick, route of infection and trypsin sensitivity of the reovirus are all influential in local intestinal protection.

Cell-mediated immunity

Pertile *et al.* (75) used monoclonal antibodies specific for B and T lymphocytes and chicken Ia (a chicken class II major histocompatibility complex antigen) to study cellular infiltrates during the development of reovirus arthritis. T-lymphocytes and plasma cells were the predominant inflammatory cells in the synovium. In the acute phase, T-cells, mostly cluster of differentiation antigen 8 (CD8) were present in low numbers. Most activity was in the subacute phase with increased numbers of CD4 and CD8 lymphocytes. Aggregates of T-cells, IgM-positive B-cells and plasma cells were also present. The chronic stage was characterised by large numbers of primarily CD4 T-cells, with few IgM-positive B-cells. Lymphocytes in chronic arthritis stained positively for Ia. The authors concluded that the types, numbers and activation level of lymphocytes present in the tarsal joints are similar, but not identical to those seen in rheumatoid arthritis in humans.

Auto-immune disease

It has been suggested that avian reovirus arthritis is an auto-immune disease which could be a model for rheumatoid arthritis in humans (58, 100), although no rheumatoid factor has been demonstrated. Other indications that auto-immune processes may be implicated were provided by the demonstration of anti-nuclear antibodies in the sera of infected chickens (28, 77). Islam *et al.* also demonstrated the presence of anti-collagen antibodies in some birds (28).

Immunosuppression

Much speculation has arisen as to whether avian reoviruses are immunosuppressive, especially relating to the use of vaccines. Several reports have described field or experimental observations. Van der Heide *et al.* reported increased incidence of Marek's disease after simultaneous vaccination of day-old chicks with herpesvirus of turkeys (HVT) and reovirus vaccine (97). Further studies by Rinehart and Rosenberger observed condemnation rates due to Marek's disease to be four times higher after a similar vaccination protocol, compared to those given HVT alone (79). Experimental work showed that immunosuppression depended on the strain of reovirus used (79). In contrast, other workers found no evidence for immunosuppression (10, 63).

Recently, Pertile *et al.* demonstrated that macrophages in the spleen of reovirus infected chickens were present in a 'primed' state and produced increased levels of nitric oxide (73). The presence of macrophages correlated with depressed *in vitro* mitogenesis. Pertile *et al.* further showed that reovirus infection in chickens does not compromise the functional capabilities of T-cells, but induces suppressor macrophages that inhibit T-cell function (74).

Pathology

The gross and histological changes in reoviral arthritis have been reviewed by van der Heide (94), McNulty (55) and Rosenberger and Olson (84) and are briefly summarised below.

Early indications of effects on the joints include soft swelling of the joints which at necropsy are seen to involve synovial membranes and surrounding tissues, with excess clear fluid in the capsule which may be turbid if bacteria or mycoplasmas are also involved. As the disease progresses, petechiae may be seen in the synovial membranes, with the development of small erosions on the articular cartilage. Adhesions between the tendons and fibrosis of tissues prevent smooth movement and the shanks may be swollen when digital flexor tendons are affected. In older, heavier birds, the gastrocnemius tendon and occasionally the digital flexor tendons may rupture.

Histopathological changes include thickening of the tendon sheaths due to oedema, hypertrophy and hyperplasia of the synoviocytes, villous proliferation of the synovial membranes and invasion with inflammatory cells. Later, the loose connective tissue around the tendon sheaths is replaced by fibrous tissue.

The pathological lesions of reoviral arthritis are not pathognomonic and may resemble those caused by *S. aureus* and *M. synoviae*, both of which may be present with the reovirus. While Kibenge and Wilcox (42) considered the pathological differences to be a matter of degree, Hill *et al.* (25) showed that histological changes due to reovirus were characterised by diffuse lymphocytic inflammation, while those caused by staphylococci were a focal purulent synovitis.

Microscopic lesions in other tissues reported in association with natural or experimental reovirus tenosynovitis have been described in the liver, spleen and bursa (25, 41, 81, 91). Pericarditis and myocarditis have been consistently reported by some workers who suggested that these conditions might be diagnostic for viral arthritis (41, 91). Depending on the strain used, other changes unrelated to tenosynovitis have been described, such as feather abnormalities (83).

Diagnostic methods

While reovirus infection is widespread, these viruses are rarely the sole cause of a disease. In chickens, the most common manifestation of disease is lameness. The clinical signs of reovirus arthritis are not pathognomonic and may resemble those caused by other agents such as *M. synoviae* and *S. aureus,* both of which can sometimes be found together with reovirus in joint disease. The disease primarily affects meat-type birds but may be seen occasionally in light egg-laying breeds (86). Confirmation of reovirus infection requires laboratory examination and is best achieved by

demonstration of the virus, which hitherto has meant isolation, although more rapid methods, such as PCR, are being developed. Adenoviruses may commonly be isolated from affected joints but are probably of no importance (31). Routine testing of sera for reovirus antibodies is commonly performed by commercial broiler companies using ELISAs, but since reovirus infections are so common, interpretation of the results is difficult if not impossible.

In cases where reovirus arthritis is suspected, since the number of birds clinically affected in a flock at any one time may be relatively small, and others may be developing the condition, examination of healthy as well as sick birds is advised. The birds should be brought to the laboratory so that the condition and gait can be appraised, and selected tissues can be collected without cross-contamination at necropsy. Alternatively, selected specimens collected aseptically can be sent to the laboratory in separate containers. The specimens could include faeces, trachea, liver, bursa, kidney and spleen. Where reovirus arthritis is suspected, the preferred samples are the hypotarsal sesamoid, including the tendons which pass through it, hock articular cartilage and synovial membrane (35). Swabbing of the joints, though simpler, may result in fewer recoveries than material from macerated tissue (35). Virus can frequently be recovered from joints with gross lesions, although isolation may not be possible in very advanced stages of joint degeneration. Specimens should be sent to the laboratory in transport medium, even though the virus is relatively resistant. If a delay occurs in processing, the specimens can be stored temporarily at 4°C, or for longer periods at –20°C or below.

Reovirus isolation is best achieved by inoculation of material into fertile chicken eggs or chick embryo cell cultures. Embryonating eggs, preferably from an SPF flock, are inoculated via the yolk sac after six days of incubation. Virulent reoviruses typically kill the embryos within five or six days of inoculation and embryos appear haemorrhagic with necrotic lesions on the liver. Inoculation of CEL cultures with reovirus results in syncytium formation in the cell sheet, with affected cells lifting off into the medium after a few days. Eosinophilic intranuclear inclusions can be seen if the cells are stained by haematoxylin and eosin. If virus is present in tissues at low titre, attempts at isolation in both systems may need two or three passages before effects are seen. The reovirus can be identified by electron microscopy after negative staining or immunofluorescence (IF) staining.

Isolation of reovirus from the joints may be considered diagnostic, but isolation from the faeces or gut tissue may be meaningless in view of the widespread nature of reovirus infection. Examination of faeces for virus is also probably of limited value in examining laying flocks for egg transmission. Al-Mufarrej *et al.* found that after experimental infection of hens with high titre virus, no virus was detected in cloacal swabs, even though tissues of chicks hatched from eggs laid at

that time were positive for virus (5). No markers exist for reovirus pathogenicity or tropism, therefore if information regarding these characteristics is needed, experimental infection of SPF chicks will be necessary.

Isolation and identification of reoviruses from the tissues is time-consuming, and other more rapid methods have been employed. Direct IF staining of cryostat sections of tendons has been used to detect the virus after experimental infection (30). More recently, Liu *et al.* used monoclonal antibodies in an immunoperoxidase staining method to detect reovirus in paraffin-embedded tissues (50). However, these methods are likely to be satisfactory only in the early stages of infection, perhaps before clinical signs of lameness are obvious. The value of these methods for field material has therefore yet to be confirmed.

Molecular approaches to identification of avian reoviruses in infected tissues have been described by several authors. These include dot-blot hybridisation (49, 102), PCR (101) and PCR combined with RFLP (47, 51). The latter enables the reovirus strain to be typed. Undoubtedly, these methods are relatively rapid and sensitive, but for routine use in examination of field material, they will need to be compared critically with virus isolation, which may be considered the 'gold standard' for avian reovirus diagnosis. In addition, isolation of the virus is necessary if it is to be studied further.

Several methods have been used to detect antibodies to avian reoviruses, including AGID, VN, IIF and ELISA. Additionally, a Western blot method has been described (15). Serological profiling for reovirus antibodies is frequently performed, but since infection is widespread the technique has limited diagnostic value, although it may be an indicator of immune status. Takase *et al.* considered that given the age-related resistance to reoviral arthritis and the half-life of maternal antibody, chicks should ideally have a 1:1,600 or higher neutralising maternal antibody titre at the time of hatching, to afford protection against oral infection until three weeks of age (90). A convenient method of testing laying flocks would be to test egg yolk, since Silim and Venne found high correlation between serum and egg yolk titres (88). Where ELISA results are equivocal, sera can be re-tested by Western blotting or IIF.

Public health implications

No public health implications are known to exist.

Prevention and control methods

Given the facts that avian reovirus infections are widespread, the viruses are relatively resistant outside the host, and vertical

transmission occurs, maintaining freedom from infection in commercial chicken flocks is virtually impossible. In addition, as indicated above, absence of detectable seroconversion and failure to detect virus in cloacal swabs are unreliable indicators of freedom from infection, or egg transmission. Thus, the main approach to reovirus control has been vaccination, using live and killed vaccines.

Since chicks are most susceptible to avian reovirus infection immediately after hatching (32, 81), vaccine protocols are designed to protect these chicks during the early days of life. This has been accomplished by passive immunity from maternal antibody following vaccination of the breeder hens or by active immunity after early vaccination with a live vaccine.

Initial attempts to prevent early infection by simple immunisation were based on controlled exposure of one-day-old chicks to live virus (57). Later, passaged versions of the S1133 strain were used for vaccination of one-day-old chicks. However, in general, the use of live vaccines in chicks at one-day-old has not been very successful. This may be related to the poor intestinal immunity in very young chicks after immunisation at this stage (66).

Efforts were later directed towards administering live or inactivated vaccines to breeding stock to provide passive immunity to the progeny via the yolk (9, 93). Inactivated preparations from strain S1133 induced maternal antibody which was relatively short-lived (78, 94). Eidson *et al.* (14) and van der Heide and Page (95) used a preparation of S1133, attenuated after seventy-four embryo passages, to vaccinate broiler breeders at ten or fifteen weeks by drinking water. The progeny were subsequently found to be resistant to oral and subcutaneous challenge with homologous virus. However, an important drawback was that the vaccine did not protect the progeny against challenge with reoviruses of a different serotype (78).

Jones and Nwajei found that use of the above vaccine in laying hens reduced the incidence of lesions in the hock joints of progeny after challenge at one day old, but had little effect on the ability to reisolate virus from the joints (36). For the development and persistence of high levels of maternal antibody, Giambrone recommended the use of a live vaccine as a primer early in life, followed by an inactivated vaccine given at six weeks of age and again prior to lay (18). More recent developments have involved the use of coarse spray administration of a cell culture clone of strain S1133/66 (19). This preparation resulted in higher antibody levels than egg-passaged vaccine. Inactivated reovirus vaccines are frequently administered to breeder flocks in combination with other killed preparations against, for example, Newcastle disease and egg drop syndrome 1976.

Although maintaining commercial flocks free of reovirus infection is virtually impossible, good management and biosecurity procedures which minimise reovirus infection of very young chickens can be used in addition to vaccination to assist in the control of reovirus-associated disease.

Avian reoviruses and importation

Avian reoviruses are virtually ubiquitous among commercial poultry and can be transmitted via the egg. Thus theoretically, prevention of the introduction of reoviruses into a country may be difficult where chicks or eggs are imported, unless from flocks which have remained free from infection. However, apart from those kept in the most rigorous conditions of isolation, most breeder flocks are likely to have encountered reovirus infection and will have some residual immunity. Some breeders vaccinate parent flocks with killed vaccines before exporting eggs or chicks, so that good levels of maternal antibodies protect the chicks during the post-hatch period when the chicks are most susceptible. Early natural exposure of the parents, or vaccination with live vaccines prompts a better antibody response from killed vaccines. Serology for avian reovirus infections is of dubious value, but a rise in antibodies in a laying flock would suggest reactivation of virus and perhaps egg transmission, even though infection in the parents is asymptomatic.

Maternal antibodies generated by the conventional reovirus vaccines, mostly based on the S1133 strain from the USA, may not be protective against the antigenic variants which exist in some countries. Although reovirus infection is widespread and most strains appear to be harmless, a range of virulence and effects has been reported. Importation of stock or eggs is not advised from any region where a disease caused by a particularly virulent reovirus is very common.

Reoviruses are relatively resistant and survive well outside the host on egg shells, egg boxes and other fomites. Poultry products should normally be safe, unless contaminated with material from the gut.

Conclusions

Reovirus-associated diseases in poultry present many problems. While the viruses are ubiquitous and easy to grow in culture, disease is rare, and hence simple detection of virus in tissues, or demonstration of serum antibodies may not confirm that the reovirus is the cause of disease. Nonetheless, demonstration of reovirus in the hock joint tissue of affected chickens can be considered confirmatory for viral arthritis. More research is required to understand the underlying basis of pathogenicity of different strains of reoviruses and the triggers which may cause a reovirus to become pathogenic. In addition, recently developed molecular diagnostic methods such as PCR need full evaluation. The use of combined PCR-RFLP methodology appears to show promise for tracing the source of infections. Finally, the development of an improved reovirus vaccine awaits a better understanding of the immune responses of the chicken to the important immunogens of the reovirus.

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Réoviroses aviaires

R.C. Jones

Résumé
Les réovirus aviaires sont ubiquistes dans les élevages avicoles. L'infection est habituellement présente sans signes apparents, mais les réovirus peuvent parfois être à l'origine de plusieurs syndromes chez les poulets, le plus important étant l'arthrite virale/ténosynovite, en particulier chez les sujets reproducteurs. Des réovirus ont été isolés chez des dindes et plusieurs autres espèces aviennes atteintes de maladies diverses, mais le lien entre la présence de ces virus et ces maladies n'a été catégoriquement établi que dans de rares cas. Chez les poulets, notamment, des réovirus aviaires présentant un pouvoir pathogène à large spectre ont été isolés et il existe plusieurs type d'antigènes. Le diagnostic se fonde sur la détection du virus dans des prélèvements cliniques, mais la présence du virus ne signifie pas nécessairement qu'il est l'agent responsable de la

maladie, sauf lorsque des réovirus sont décelés dans les articulations atteintes. Les épreuves sérologiques sont le plus souvent difficiles à interpréter, car les réoviroses sont très répandues et le plus souvent inoffensives. La prophylaxie de l'arthrite virale/ténosynovite repose essentiellement sur la primo-vaccination des jeunes volailles à l'aide de vaccins à virus atténué, puis à la vaccination des reproducteurs en utilisant des vaccins à virus inactivé, le but étant de protéger les poussins grâce aux anticorps maternels. De nombreux vaccins sont basés sur la souche S1133, isolée aux États-Unis d'Amérique, mais ils peuvent s'avérer inefficaces face à la diversité antigénique de ces virus.

Mots-clés
Arthrite virale – Boiterie – Poulets – Réovirus aviaires – Syndrome de malabsorption – Syndrome de retard de la croissance – Ténosynovite – Vaccins à virus inactivé – Vaccins à virus vivant.

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Infecciones aviares por reovirus

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Resumen
Los reovirus aviares son ubicuos entre las bandadas de aves de corral. Aunque en general la infección está presente sin causar ninguna enfermedad, los reovirus pueden estar implicados ocasionalmente en varios síndromes infecciosos, de los cuales el más importante es la artritis/tenosinovitis vírica del pollo, que afecta sobre todo a pollos asaderos. Aunque se han aislado reovirus en pavos y otras especies aviares afectadas de patologías varias, son relativamente escasos los episodios en que la presencia de esos virus ha podido relacionarse de forma concluyente con la patología en cuestión. Sobre todo en el pollo se han aislado diversos tipos antigénicos de reovirus aviares dotados de un poder patógeno de amplio espectro. El diagnóstico depende de la detección del agente etiológico en muestras clínicas. La presencia de reovirus, sin embargo, no confirma necesariamente que sean los causantes de la enfermedad, excepto cuando se encuentran en articulaciones afectadas. Habida cuenta de la distribución generalizada y del carácter a menudo inocuo de las infecciones por reovirus, las pruebas serológicas suelen resultar de difícil interpretación. El método principal de lucha contra la artritis/tenosinovitis vírica consiste en vacunar a las aves jóvenes con vacunas atenuadas y administrar después preparaciones inactivadas a los ejemplares reproductores, para que los anticuerpos maternos protejan a los polluelos. Muchas vacunas se preparan a partir de la cepa S1133, aislada en los Estados Unidos de América, aunque es posible que no sean eficaces contra todas las variantes antigénicas.

Palabras clave
Artritis viral – Cojera – Pollos – Reovirus aviares – Síndrome de mala absorción – Síndrome de retraso del crecimiento – Tenosinovitis – Vacunas inactivadas – Vacunas vivas.

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