Avian Reovirus

Zeinab M. S. Amin Girh[1], Nagwa S. Rabie[1] and Mona S. Zaki[2]

[1]Department of Poultry Diseases, National Research Centre, Dokki, Giza, Egypt.
[2]Hydrobiology Department, National Research Centre, Dokki, Giza Egypt.
Dr_mona_zaki@yahoo.co.uk

Abstract: Reovirus infections are actually related to a lot of disease conditions with different clinicalmanifestations. Reoviruses have been isolated from a variety of tissues in poultry, suffering from differentdisease conditions including viral arthritis/tenosynovitis, stunting syndrome, respiratory disease, entericdisease, immunosuppression and malabsorption syndrome. Economic losses related to reoviral infections arefrequently associated with increased mortality, viral arthritis/tenosynovitis and general lack of performance, including diminished weight gains, high feed conversionsuneven growth rates and reduced marketability ofthe affected birds. The resistance ofthe virus could be one of the reasons for such a high prevalence. This high prevalence put emphasis on thevaccination of the breeder flocks and shows the necessity of more studies on aspects of Reovirus.
[Zeinab M. S. Amin Girh, Nagwa S. Rabie and Mona S. Zak. **Avian Reovirus.** *Stem Cell* 2019;10(2):1-4]. ISSN: 1945-4570 (print); ISSN: 1945-4732 (online). http://www.sciencepub.net/stem. 1. doi:10.7537/marsscj100219.01.

Keywords: Avian Reovirus

Introduction

Reoviruses (a name derived from "respiratoryenteric orphan" or REO) are members of the genus*orthoreovirus*in the Reoviridae family **(Rosenberger,2003).** Found to be ubiquitous among poultry flocks, Avian Reoviruses (ARV) have been isolated frequentlyfrom the gastrointestinal and respiratory tracts of chickens affected.

By several pathological conditions, including viral arthritis or tenosynovitis, stunting syndrome, respiratory disease, enteric disease, immunosuppression, malabsorption syndrome or even inapparentinfections **(Jones, 2013).** Originally, the REO abbreviation was used toidentify virus groups that were not associated with any known disease **(Jones, 2013)**. Viral arthritis is an Viral arthritis is aneconomically important disease of chickens thatcanbe caused by different serotypes and pathotypes of ARV **(Rosenberger, 2003).** Tenosynovitis, defined bythe changes in the tendons and their sheaths, can beconsidered different from the condition caused by *Msynoviae*. Somereoviruses have an arthrotropic characteristicthat includes ruptured gastrocnemius tendons, pericarditis, myocarditis, hydropericardium, unevengrowth and mortality **(Jones, 2013).**Viral arthritis or tenosynovitis in poultry is oneof the pathological manifestations of ARV infection **(Rosenberger, 2003).** The reoviruses can act aloneas pathogenic agents or in combination with one ormore other aetiological agents, such as *M. synoviae*or *Staphylococcus* spp., and this situation can lead tovaried clinical pictures of arthritis or tenosynovitis **(Rosenberger, 2003).** The reoviruses can be isolated from birds without any signs of disease, but they arealso associated with a variety of problems includingviral arthritis/tenosynovitis, enteric disease and malabsorptionsyndrome **(Jones, 2013)**. Reoviruses have been isolated worldwide fromchickens affected by various disease conditions, predominantly including viral arthritis/tenosynovitis, stunting syndrome, enteric disease, malabsorptionsyndrome and immunosuppression **(Glass *et al.*, 1973; Goodwin *et al.*, 1993a, b; McNulty, 1993; Jones, 2003; Rosenberger, 2003).** Economic lossescausedbyreovirus infections are frequently the result of elevated mortality, increased slaughterhouse condemnationsand poor performance, including diminished weightgains and high feed conversions **(Dobson and Glisson, 1992; De Herdt*et al.*, 1999; Rosenberger, 2003).** Forthis reason, vaccination of chickens against reovirusispracticed in most parts of the world and has beenproven efficacious. Vaccination of breeders can protect young broilers through the transfer of maternal antibodies In 1998, however, reovirus was held responsible for major disease outbreaks in broiler flocks in Poland, notwithstanding the fact that their parents had been well vaccinated **(van Loon *et al.*, 2001).** Infectedbroiler flocks suffered from high mortality and signs of malabsorption. At necropsylesions including hydro -pericardium, enlarged livers with multiple necrotic foci and swollen spleens were found. The signs andlesions seen under field conditionswereexpe - rimentally reproduced in SPF chicks throughintramuscular and oral challenge with a reovirusstrain that was isolated from the broilers affected under fieldcircumstances. Recently, central nervous disorders were also ascribed to this type of reovirus infection inchickens **(Van De Zande *and* Kühn2007).** Reoviruses can be classified into

different serotypesusing the plaque reduction assay. In this test, reovirusstrains isolated from diseased broilers in Poland couldnot be neutralized by antisera against knownreoviruses **(van Loon *et al.*, 2001).** Furthermore, characterization of the strains with a panel ofmonoclonal antibodies revealed a reaction pattern that was different from the reovirus strains described in theliterature **(Johnson, 1972; van der Heide*et al.*, 1974; Hieronymus, 1983; Rosenberger *et al.*, 1989; vanLoon, 2001)**. Therefore it was concluded that thereovirus strains from Poland belonged to a newserotype. They were subsequently called Enteric Reovirus Strains (ERS). Screening in the field duringthe following years demonstrated that strains of ERStypereovirus are prevalent in many Europeancountries, the USA, Argentina, the United Arabic Emirates, South Africa, the Philippines and Indonesia **(Van De Zande and Lin, 2005)** *Aetiology and economic important* Reoviruses have a worldwide distribution in chickensbut are more related to meat-type birds **(Van der Heide, 1977).** They are commonly found in the digestiveand respiratory tracts of clinically normal chickensand turkeys. It is estimated that most of the reovirusesisolated from chickensare non-pathogenic. Severalstudies performed over the last years have revealedunique properties for ARV e, displayed bydifferent fromby mammal viruses. **(Jones, 2013)**

ARV, which replicatein the cytoplasm, arenonenvelopedwith an icosahedral symmetry and a doubleshelled capsid and are one of the fewnon-envelopedviruses that cause cell to fuse **(Xu and Coombsa, 2009).** This specific genome segments responsible for proteincoding have been identified for the S1133strain of ARV and differentiates them phylogeneticallyfrommost other animal reoviruses **(Day, 2009).** Another interest characteristic of the ARV is that they are knownto induce apoptosis in infected cells **(Benavente and Martínez-Costas, 2007).** Avian reoviruses infections are of economic importanceto the poultry industry **(Savage and Jones, 2003)**. In meat-type chickens, economic losses are frequentlyassociated with *reovirus*infections. Increased mortality, viral arthritis/tenosynovitis and a general lack ofperformance are among the observed problems **(Jones,2013).** Breeder flocks that develop viral arthritis just prior to the onset of or during egg production may, inaddition to lameness, be affected by increased mortality, decreasedegg production, suboptimal hatchability/fertility and vertical transmission of the virusto progeny. Infectious viral arthritis is currently the best definedand most readily diagnosed reovirosis. **(Rosenberger,2003)**

Epidemiology and pathogenesis

Reoviruses can be classified using serologic proceduresor grouped according to their virulence. There are five serotypes of reoviruses from 77 isolates fromintestines, respiratory tract and synovial isolates **(Day2009).** They are antigenically similar viruses and demonstrateclear strain differences based on virulenceand virus persistence. There are considerable crossneutralization between heterologous serotypes (Islam et al., 1988). The ARV genome consists of 10 segmentsof double-stranded RNA: three large (L1, L2, L3), three medium (M1, M2, M3) and four small (S1, S2, S3, S4) (Jones, 2013).

In general, ARV is associated with arthritis, butthey have also been identified as the etiological agents of other diseases. Some examples are malabsorptionsyndromeconditions, pericarditis, myocarditis, hydropericardium, enteritis, hepatitis, bursal and thymicatrophy, osteoporosis, and acute and chronic respiratory syndromes **(Rosenberger, 2003).** Although reoviruseshave been found in manyavian species, chickens and turkeys are the only recognized natural or experimentalhosts for reovirus-induced arthritis (Pertilem et al., 1996). Other bird species from which reovirusescanbe isolated are ducks, pigeons, geese and psittacine species **(Watier, 2010).** Initially, the ARV replicates in the villi of the smallintestine and in the bursa, and then spreads to other tissues. Generally, osmotic diarrhea appears due to villi blunting **(Rosenberger, 2003).**

When a bird is infectedbyreoviruses, these increase susceptibility to otherinfectious agents (Watier, 2010). This immunosuppressionis due to lymphoid depletion and compromise ofthe immune system. Some authors report age-relatedresistance to reovirus-induced arthritis (Jones and Georgian, 1984; Olson and Kerr, 1966). Again, this age-associated susceptibility may be related to theinability of young birds to develop an effective immune response **(Jones, 2013).** The virus can be spread laterally (horizontal transmission) but vertical and eggtransmissionare also possible **(Robertson and Wilcox,1986).** ARV may be excreted from the intestinalor respiratory tracts for at least 10 days post-inoculation. This fact suggests fecal contamination as a primarysource of contact **(Jones, 2013).** Viral persistence canlast for long periods, special in the caecal tonsils and hock joints (**Savage and Jones, 2003).** Birds that areinfectedata young age are potential sources of infection **(Rosenberger, 2003).** Whether or not the diseaseoccurs following infection with ARV, the incubation period ranges from 1 to 11 daysand is highly dependentupon the virus pathotype, age of the host androute of exposure (footpad inoculation, intramuscular, intravenous)**.** Very often, infections are unapparent and demonstrable only by serology or virus isolation **(Jones, 2013)**. The virus frequently locates in the flexor and extensortendons of the pelvic limb and is commonly seenin young birds (1-2 months). Mortality

is usually low, but morbidity can becan be as high as 100%. Avian reovirusespossess group-specific antigen and serotype-specificantigen. Host's humoral immunity (neutralizing antibodies) can be detected 7-10 days following infection. The presence of neutralizing antibodies and its importancein establishing protection is not-well defined yet. Birds may become persistently infected in the presenceof high levels of circulating antibodies. It is apparent, however, that maternal antibodies can afford a degreeof protection to day-old chickens against naturally occurring and experimental challenges. From severalstudies, the suppression of T-cell-mediated immunity bycyclosporin A resulted in increased mortality inreovirus-infected birds, but the relative severity of tendon lesions was not altered. Antibody protection isrelated to serotype homogeneity, virulence, host age and antibody titer **(Grande et al., 2002; Jones, 2013; Rosenberger, 2003).** For cell mediated immunity, the CD8+ T-cells may play a role in pathogenesis and/or*reovirus*clearance in the small intestine. Some authors have shown that challenging viruses are controlled inthe absence of actively produced antibodiesin B-cell immunosuppressed chicks **(Day, 2009).** This suggeststhat cellular immunity may be sufficient for broiler protection. **(Jones, 2013)**

Clinical signs and lesions

In an acute infection, lameness is generally presentand some chickens are atrophied (Crespo and Shivaprasad, 2011). In chronic infection, lameness iseven more pronounced, but the percentage of infectedchickens is small. Lameness in this type of lesions isdue to enlargement in the area of the gastrocnemiusor digital flexor tendons. In general, the rupture of thegastrocnemius tendon is noticeable (Figure 4). Theswelling of the digital flexor and metatarsal extensortendons is the more pronounced macroscopic lesion. Swelling of the foot pad and hock joint is less frequent, being marked be the edema of the tarsal and metatarsal tendon. Some petechialhemorrhages are frequent in the synovial membranes **(Jones, 2013; Rosenberger, 2003).** In chronic infection, inflammation of tendon areas progresses, tendon sheaths become hard and they fuse insome cases. In early infection, recovery is quick, butvery often the tendon rupture occurs at transfer **(Crespoand Shivaprasad, 2011)**. In terms of microscopic lesions, the basic pictureis edema, coagulation necrosis, accumulation of heterophilicmaterial and perivascular infiltration. Thereis also hypertrophy and hyperplasia of synovial cells, infiltration of lymphocytes and macrophages, anda proliferation of reticular cells (Hill et al., 1989). Lesions are strongly time-dependent and changeshave been found in the type and number of positivelystaining cells. The synovial membranes develop villousprocesses during the chronic phase and lymphoidnodules are present. When the process becomes furtherchronic, the inflammatory picture changes, theamount of fibrous connective tissue increases, and apronounced infiltration or proliferation of reticularcells, lymphocytes, macrophages and plasma cellscan also be seen. Irregular granulation tissues replacesome tendons, and large villi appear on the synovialmembranes. (Jones, 2013)

Diagnosis, control, treatment, and prevention

A presumptive diagnosis of viral arthritis canbe made on the basis of signs and lesions. Primaryinvolvement of the metatarsal extensor and digitalflexor tendons, and heterophil infiltration in the heart, assist differentiatingthe infection from bacterialand mycoplasmalsynovitis **(Jones, 2013).** Differentdiagnostic methods are available: fluorescent antibodytechniques, virus isolation, typical physicochemicalcharacteristics and the presence of a group—specificantigen demonstrable with the agar gel precipitin test. Theimmunoperoxidase procedures can be used, butthey are not the first choice (Rosenberger, 2003). Serology for reoviruses is routinely used, being based on group-specific antibodies that can be detectedreadily by the agar gel precipitin test or by indirectfluorescent antibody test (IFAT). In more recent years, ELISA for detecting antibodies to avian reoviruses along with PCR has become more common **(Bruhn etal., 2005).** The ubiquitous nature of the avian reoviruses andtheir inherent stability, coupled with modern, highdensity Confinementrearing practices, suggests thatelimination of virus exposure may be difficult **(Jones, 2013).** Resistance to inactivation maybe frequentlycarried by mechanical means like brooding temperatures.

Commercially available disinfectants should bevalidated for efficacy before use, because of the avian *reovirus*group relative stability **(Rosenberger, 2003).** Vaccines and vaccination programshave evolved and can provide protection at 1 day of ageonwards. Active immunization can be achieved by vaccinationwith viable attenuated reoviruses, which areusually applied by the subcutaneous route **(Giambroneand Clay, 1986).** *Reovirus*vaccination of breedingstock can be carried out with live attenuated or inactivatedvaccines. The latter are more effectivewhen preceded by vaccination with a live vaccine. Ifa live vaccine is used, it should be administered priorto the onset of egg production, to prevent transovariantransmission of the vaccine virus **(Jones, 2000).** The advantages of this type of immunization programinclude immediate protection of the day-old progeny asprovided by maternal antibodies **(Jones, 2013).** Vaccinationof breeders is an effective method of controllingviral arthritis and other

pathogenic reoviruses, but itshould be recognizedthat protection is assured againsthomologous serotypes only. **(Rosenberger, 2003)**

References:

1. Rosenberger JK (2003) *Reovirus*infections. In: Diseases of Poultry13th ed, Iowa State University Press, Ames: pp 283-298.
2. Jones RC (2000) Avian*reovirus*infections. Diseases of poultry: world trade and public health implications. Rev Sci Tech 19: 614-625.
3. Jones RC (2013) *Reovirus*infections – Viral Arthritis. In: Diseases of Poultry 13th ed, Iowa State University Press, Ames: pp 352-361.
4. Van der Heide L (1977) Viral arthritis/tenosynovitis: a review. Avian Pathol 6: 271-284.
5. Xu W, Coombsa KM (2009) Conserved structure/function of the*orthoreovirus*major core proteins. Virus Res 144: 44-57.
6. Day JM (2009) The diversity of the *orthoreoviruses*: Moleculartaxonomy and phylogenetic divides. Infect Gen Evol 9: 390-400.
7. Benavente J, Martínez-Costas J (2007) Avian *Reovirus*: Structure andbiology. Virus Res 123: 105-119.
8. Savage CE, Jones RC (2003) The survival of avian reoviruses onmaterials associated with the poultry house environment. Avian Pathol 32: 417-423.
9. Islam MR, Jones RC, Kelly DF (1988) Pathogenesis of experimental*reovirus*tenosynovitis in chickens: influence of the route ofinfection. J Comp Pathol 98: 325-336.
10. Jones RC, Georgian K (1984) Reovirus-induced tenosynovitis inchickens: The influence of age at infection. Avian Pathol 13: 441-457.
11. Watier JM (2010) Une nouvelle souche de *Réovirus*pathogène. Filiéres Avicoles 735: 50-51.
12. Robertson M, Wilcox GE (1986) Avianreovirus. Vet Bull 56: 155-174.
13. Hill JE, Rowland GN, Glisson JR, Villegas P (1989) Comparativemicroscopic lesions in reoviral and staphylococcal tenosynovitis. Avian Dis 33: 401-410.
14. Giambrone JJ, Clay RP (1986) Evaluation of broiler breeder pulletvaccination programs for preventing clinical *reovirus*infections inthe progeny. Poultry Sci 65: 457-461.
15. Grande A, Costas C, Benavente J (2002) Subunit composition andconformational stability of the oligomeric form of the avian *reovirus*cell-attachment protein σC. J Gen Virol 83: 131-139.
16. Crespo R, Shivaprasad HL (2011) Rupture of gastrocnemius tendon in broiler breeder hens. Avian Dis 55: 495-498.
17. Bruhn S, Bruckner L, Ottiger HP (2005) Application of RT-PCR fordetection of avian *reovirus*contamination in avian viral vaccines. JVirol Meth 123: 179-186.
18. Glass S.E., Naqi S.A., Hall C.F., Kerr K.M. (1973). Isolationand characterization of a virus associated with arthritis ofchickens. *Avian Diseases 17*, 415-424.
19. Jones R.C. Reovirus infections: other reovirus infections. (2003). In: Saif Y.M, Barnes H.J., Glisson J.R., Fadly.
20. A.M., McDougald L.R., Swayne D.E. (editors). *Diseasesof Poultry*. 11th ed., Iowa State Press, Ames, IA., p. 293-298.
21. Goodwin M.A., Davis J.F., Player E.C. (1993a). Reovirusassociatedenteritis in Georgia broiler chicks. *Avian Diseases 37*, 229-233. Goodwin M.A., Davis J.F., McNulty M.A., Brown J., Player E.C. (1993b). Enteritis (so-called runting stuntingsyndrome) in Georgia broiler chicks. *Avian Diseases 37*,451-458.
22. McNulty M.S. (1993). Reovirus. In: McFerran J.B., McNulty M.S. (editors). *Virus Infections of Birds*. ElsevierScience Publishers, Amsterdam, London, New York, Tokyo, p. 181-198.
23. De Herdt P., Ducatelle R., Uyttebroek E., Hermans J., Sneep A., Torbeyns R. (1999). Reovirus serology in broiler parents and their progeny, and its correlation withperformance. *Avian Diseases 43*, 271-278.
24. Dobson K.N., Glisson J.R. (1992). Economic impact of adocumented case of reovirus infection in broiler breeders.*Avian Diseases 36*, 788-791.
25. van Loon A.A., Koopman H.C., Kosman W., Mumczur J., Szeleszcuk O., Karpinska E., Kosowska G, Lütticken D. (2001). Isolation of a new serotype of avian reovirusassociated with malabsorption syndrome in chickens. *Veterinary Quarterly 23*, 129-13.
26. Van De Zande S., Kühn E.-M. (2007). Central nervoussystem signs in chickens caused by a new reovirus strain: a pathogenesis study. *Veterinary Microbiology 120*, 42-49.
27. Johnson D.C. (1972) Diagnosis, pathology and etiology oftenosynovitis in broilers and broiler breeders. *Avian Diseases 16*, 1067-1072.
28. van der Heide L., Geissler J., Bryant E.S. (1974). Infectioustenosynovitis: serological and histopathological responseafter experimental infection with a Connecticut isolate. *Avian Diseases 18*, 289-296.
29. Hieronymus D.R.K., Villegas P., Kleven S.H. (1983). Identification and serological differentiation of severalreovirus strains isolated from chickens withmalabsorption syndrome. *Avian Diseases 27*, 246-254.
30. Rosenberger J.K., Sterner F.J., Botts S., Lee K.P., Margolin A. (1989). In vitro and in vivo characterization of avianreoviruses. I. Pathogenicity and antigenic relatedness ofseveral avian reovirus isolates. *Avian Diseases 33*, 535-544.
31. Van De Zande S., Lin F. (2005). Enteric reovirusstrain (ERS) infections: how to control the disease? *Proceedings 14th World Veterinary Poultry Congress*, Istanbul, Turkey. p. 325.