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Forms of avian reovirus in poultry production: An overview

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ABSTRACT

This review article focuses on avian reovirus (ARV) regarding the virus characters, susceptibility and transmission, the different clinical forms, laboratory diagnosis, and preventive measures. Despite most of ARV strains are abundant and innocuous, they are responsible for many diseases conditions in poultry industry. The pathogenic ARV strains induce great economic losses including growth retardation, increasing culling rate, high mortality rate, immunosuppression, and increasing the carcass rejection rate at processing. Strains of ARV belong to the family *Reoviridae* and genus *Orthoreovirus* are non-enveloped and double-stranded RNA. Almost all of avian species are susceptible to infection especially at young ages. The virus rapidly spreads among flocks via the horizontal, vertical, and mechanical routes. The infection with ARV is mainly associated with arthritis/ tenosynovitis and runting stunting syndrome. However, other clinical pictures such as gastroenteritis, hepatitis, myocarditis, and respiratory disease are also related to ARV infections. Laboratory diagnosis is based on isolation and characterization of the virus using conventional methods of detection. Nevertheless, recent molecular techniques are also regarded as suitable for the efficient diagnosis. Serological detection of specific ARV antibodies have been also applied. Adoption of hygienic measures and vaccination with live or inactivated vaccines are the most suitable methods for the prevention of field ARV infections.

Introduction

The term "reovirus" is an abbreviation for "respiratory, enteric, orphan virus". The virus was first isolated from the lungs and intestines of humans without clinical manifestations (Jones, 2000). The avian reovirus (ARV) strains seem to be virtually ubiquitous among commercial poultry flocks because they have been detected in apparently healthy birds (Rosenberger *et al*., 2003). About 85-90% of the isolated ARV strains were non-pathogenic (Pitcovski and Goyal, 2020). However, pathogenic ARV strains are usually associated with different clinical pictures including viral arthritis (VA)/ tenosynovitis (Levisohn *et al*., 1980; Page *et al*., 1982a), runting stunting syndrome (RSS)/malabsorption syndrome (MAS)/ brittle bone disease/femoral head necrosis (Vertommen *et al*., 1980; van der Heide *et al*., 1981; Page *et al*., 1982b; Pass *et al*., 1982; Goodwin *et al*., 1993), enteric disease (Dutta and Pomeroy, 1969), respiratory disease (Fahey and Crawley, 1954; Petek *et al*., 1967), cloacal pasting and mortality (Dutta and Pomeroy, 1969), ulcerative enteritis (Krauss and Ueberschar, 1966), inclusion body hepatitis (McFerran *et al*., 1976), and sudden deaths with lesions in the heart, kidney, and liver in broilers (Bains *et al*., 1974; Bagust and Westbury, 1975). Besides, high mortalities with splenic swelling and necrosis were seen in cases of Pekin ducklings have a novel ARV (Du *et al*., 2020; Xiao *et al*., 2020), and nephritis, hepatitis, and splenitis in goslings (Gouvea and Schnitzer, 1982). Replication of the ARV in the bursa of Fabricious (Pantin-Jackwood *et al*., 2007) and suppression of macrophages and T cells (Pertile *et al*., 1996) cause transient and possibly permanent immunosuppression.

Infections with ARV have been reported in many countries worldwide including United States of America (Goodwin *et al*., 1993; Pantin-Jackwood *et al*., 2008; Lu *et al*., 2015; Egaña-Labrin *et al*., 2019), France (Troxler *et al*., 2013), Poland (Sty´s-Fijoł *et al*., 2017; Czekaj *et al*., 2018; Nowak *et al*., 2022), Canada (Ayalew *et al*., 2017; Palomino-Tapia *et al*., 2022), Brazil (Souza *et al*., 2018; De Carli *et al*., 2020), Germany (Farkas *et al*., 2018), China (Chen *et al*., 2012a,b; Zhong *et al*., 2016; Cao *et al*., 2019; Chen *et al*., 2019; Zhang *et al*., 2019; Huang *et al*., 2023), Japan (Yamaguchi *et al*., 2022), Hungary (Palya *et al*., 2003), India (Awandkar *et al*., 2012, 2017), Egypt (Madbouly *et al*., 1997a,b,c; Madbouly *et al*., 2001; Zaher and Mohamed, 2009; Abd El-Samie, 2015; Mansour *et al*., 2018), Sudan (Elmubarak *et al*., 1990), Iran (Khodashenas and Aghakhan, 1992; Bokaie *et al*., 2008; Hedayati *et al*., 2013, 2016; Mirbagheri *et al*., 2020), and Iraq (Al-Baroodi, 2020).

The ARV belongs to family Ortho*Reoviridae* and genus *Orthoreovirus* and it is non-enveloped, segmented, and double stranded RNA. Almost all domestic poultry species could be infected with the virus (Shehata *et al*., 2021; Kovács *et al*., 2022; Huang *et al*., 2023). Infections with ARV induced adverse economic impacts in terms of poor performance parameters, reduced marketability, increased culling rate, and high mortality rate of broiler, layer, and breeder chicken flocks (Jones, 2013; Nham *et al*., 2017). In addition, unsightly appearance of affected hock joints may result in increasing the incidence of carcass rejection at slaughter (Souza *et al*., 2018; Reck *et al*., 2019). The molecular identification of the virus strains can identify the species-specific types in turkeys, ducks, goose, chickens (Jones *et al*., 1989). Young birds, especially those without maternal antibodies, are highly susceptible to ARV infection (van der Heide, 2000). Low pathogenic strains of ARV mostly induced sub-clinical or latent asymptomatic, however, virulent strains, particularly in immunosuppressed birds, are often associated with VA and/or MAS (Jones, 2013). Moreover, the latent infection may become active following secondary bacterial or viral infection. Therefore, the virus' virulence and dose, the

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route of infection, the age of birds, the existence of maternal antibodies, and the immune status of birds are key factors for determination of ARV infection course. It is important to mention that ARV has no public health importance.

Application of management practices and strict biosecurity measures as well as effective vaccination programs are crucial for the prevention of ARV infection. The first line of defense against ARV infection in young ages is the maternal immunity from vaccinated breeder pullets (Giambrone *et al*., 1992; Cookson *et al*., 2005; Madbouly *et al*., 2009). Thus, vaccination of chicken and turkey breeders' flocks with inactivated ARV vaccines could decrease the possibility of vertical transmission and afford progeny with specific protective maternal antibodies against the field virus strains (Sellers, 2017). Moreover, apthogenic and modified live ARV vaccines show variable protective results (Petrone-Garcia *et al*., 2021).

Accordingly, the objectives of this article were demonstration of the ARV characters, susceptibility and transmission, different clinical forms, laboratory diagnosis, and preventive measures.

The virus

ARV belongs to the family *Reoviridae* and genus *Orthoreovirus* (Robertson and Wilcox, 1986). The virus size is 70-80 nanometer, non-enveloped, and double-stranded RNA with a unique icosahedral inner and outer capsid shell (Benavente and Martinez-Costas, 2007). According to the molecular size on electrophoresis, the linear segments (n=10) of ARV genome are classified into small (S1, S2, S3, S4), medium (M1, M2, M3), and large (L1, L2, L3) segment. Moreover, the ARV genome has 8 structural and 4 non-structural proteins (Bodelon *et al*., 2001). These proteins are λ, μ, and σ encoded by these segments, respectively. Likewise, proteins encoded by the genome fall into 3 size classes: X (large), p (medium), or a (small). The segment M1 is the most conserved target region (95% similarity) that could be detected molecularly (Tang *et al*., 2016). However, segments S1 and M2 are the most variable regions in the whole ARV genome (Su *et al*., 2006). The ARV could be classified into different clusters and genotypes based on a minor viral-cell attachment capsid protein (Sigma C and S1) (Schnitzer, 1985; Kant *et al*., 2002; Day, 2009). The S1 protein contains the most hypervariable regions of the virus that provoke specific neutralizing antibodies against the field infections (Liu *et al*., 2003; Guardado Calvo *et al*., 2005; Jones, 2013), and the other conserved sequences within this protein could also be detected (Goldenberg *et al*., 2010). Moreover, ARV could be re-classified into 6 lineages: I to VI according to S1 segment (Lu *et al*., 2015; Ayalew *et al*., 2017). The protein coding assignments of the whole genome of S 11 133 strain have been determined (Varela and Benavente, 1994; Martínez-Costas *et al*., 1997). The complete genome of ARV consists of 23,420 nucleotide base pairs (bp), including segments ranging from 1191 bp (S4) to 3959 bp (L1) (Dandár *et al*., 2014). The fusogenic strains of reoviruses can affect mammals, birds, and reptiles to form multinucleated syncytia, while non-fusogenic viruses mainly infect mammals (Day *et al*., 2007).

The strains of ARV are relatively resistant outside the host as it can survive for up to 10 days on feathers, glass, wood shavings, rubber, and galvanised metal, and for 10 weeks in water. The virus is stable at pH 3.0-9.0. The ambient temperature favors the virus viability, but 56°C inactivates it within an hour. Though most of ARV strains are resistant to the proteolytic enzymes, sensitivity to trypsin was detected in turkeys with VA (Al-Afaleq and Jones, 1991; Jones *et al*., 1996). The ARV showed variable sensitivities to the different disinfectants. For instance, the virus may remain viable in 2% formaldehyde at 4°C (Meulemanns and Halen, 1982), but it can be inactivated by 2% phenol and 100% ethyl alcohol (Petek *et al*., 1967). It is important to note that ARV strains are non-haemagglutinating and fusional to the host cells, while mammalian strains are not (Robertson and Wilcox, 1986).

Susceptibility

Host

Chickens and turkeys are the most susceptible hosts to ARV infection (Madbouly and El-Sawah, 1999; Jones, 2013; Tang *et al*., 2015). Other avian species such as ducks (Malkinson *et al*., 1981; Farkas *et al*., 2018; Cao *et al*., 2019; Zhang *et al*., 2019), geese (Palya *et al*., 2003; Zhang *et al*., 2006; Yun *et al*., 2012; Dandár *et al*., 2014; Nowak *et al*., 2022), psittacine birds (Sánchez-Cordón *et al*., 2002), African green parrots (Graham, 1987), pigeons (Gough *et al*., 1988), American woodcocks (Docherty *et al*., 1994), and wild exotic birds (Natalia and Hanna, 2017) could be infected with the virus. Some differences including antigenicity, hosts, and pathogenic features have been reported between chicken and duck reovirus origins (Yun *et al*., 2013). The complete genomic sequences of reovirus strains have been performed in chickens (Dandár *et al*., 2014), turkeys (Tang *et al*., 2015), Muscovy ducks (Wang *et al*., 2013), and geese (Yun *et al*., 2012; Niu *et al*., 2018). Despite the high similarity sequence of the S1 segment among reovirus strains of duck and geese origin, they should not be classified as one species (Bányai *et al*., 2005). Heavy or meat type chicken breeds are more susceptible to VA than light egg types breeds (Jones and Kibenge, 1984).

Age

The resistance to ARV infection in chickens is obviously age-linked (Montgomery *et al*., 1986; Roessler and Rosenberger, 1989). Under experimental infection, day-old chicks showed higher intestinal virus load and more severe joint lesions than 2-week-old chickens (Jones and Georgiou, 1984). The disease picture in older chickens is usually less severe with a longer incubation period than in younger's (Jones and Georgiou, 1984).

Infection and transmission

The main route of ARV infection in poultry flocks is the ingestion or inhalation of infected materials (Ni and Kemp, 1995). Mechanical virus infection via injured skin or litter is also possible and the virus could establish in the hock joints (Al-Afaleq and Jones, 1990). The ARV could remain viable in the oviduct of hens for at least 258 days (Kerr and Olson, 1969), thus vertical infection is possible. The vertical transmission of the virus may be accompanied by embryos death with decreasing hatchability (Al-Muffarej *et al*., 1996) and the infected progeny show adverse losses. However, this route probably occurs at a very low rate (Menendez *et al*., 1975; Al-Muffarej *et al*., 1996). Under experimental conditions, vertical transmission of ARV was proved, and the inoculated virus persisted for long time in the ceacal tonsils and hock joints (van der Heide and Kalbac, 1975; Jones and Georgiou, 1984). Therefore, hatched chicks from infected breeders could act as reservoir carriers or nucleuses for the virus transmission to the non-infected contact birds and the surrounding environment (Menendez *et al*., 1975; Al-Muffarej *et al*., 1996). Free-living wild birds could carry virulent ARV strains (genetically related to chicken origin), so such birds are regarded as reservoirs for the commercial poultry flocks (Lawson *et al*., 2015).

Tissue distribution

The nasal, tracheal, or oral experimental infection of specific pathogen free (SPF) hens revealed distribution of ARV to the respiratory, enteric, and reproductive organs as well as the hock joints (Menendez *et al*., 1975). The immunofluorescence, immuno-peroxidase, and electron microscopy proved that the small intestine and the bursa of Fabricious are the portals of the virus entry, followed by a potential dissemination of the virus to the other organs within 24-48 hrs of infection (Jones *et al*., 1989). The study of Kibenge *et al*. (1985) showed that oral experi-

mental infection of chicks with ARV induced replication in the digestive tract, followed by viraemia with the presence of the virus in the plasma, erythrocyte, and mononuclear cells within 30 hrs of infection, and finally distribution throughout the body organs 3 to 5 days post-infection. However, the main target organ of arthro-tropic ARV strains is the hock or the tibiotarsal-tarsometatarsal joint where the virus replicates, shows a long-term persistence, and induces a serious joint damage (Walker *et al*., 1972; Sahu and Olson, 1975; Jones and Kibenge, 1984). Some studies also considered the liver is the target organ for ARV as orally infected chicks showed deaths within 10 days post-infection due to severe hepatitis (Jones and Kibenge, 1984). As a result of ARV persistence in chicken's tissues for a long time, the virus could be recovered up to 285 days (Kerr and Olson, 1969) and 13 weeks (Jones and Onunkwo, 1978) from spleen and hock joint, respectively. The tropism of ARV to the different tissues was genetically determined as it could be related to mutations in the S1 segment of the genome (Meanger *et al*., 1999).

Clinical forms

Viral arthritis/ tenosynovitis

The VA/tenosynovitis associated with ARV infection causes adverse economic losses as a result from inability of lame birds to reach feed with a subsequent reduced growth rate, poor conversion ratio, or deaths. Moreover, during the carcass processing, the incidence of low grade carcasses and the rejection rate may increase due to the unsightly appearance of the affected hock joints. The prevalence of VA is rare in ages less than 4-5 weeks, but it is commonly detected at 16 weeks of age. Sometimes, broiler breeders at the peak of production could be affected. Heavy or meat-type broiler breeds chickens are more susceptible to VA than light hybrids or commercial White Leghorns (Jones and Kibenge, 1984). However, occasional cases of ARV associated VA outbreaks were found in lighter layers breeds (Schwartz *et al*., 1976). The pathogenicity of VA in chickens is sometimes influenced by presence of other co- infections such as *Mycoplasma synoviae* (*M. synoviae*) (Bradbury and Garuti, 1978; Reck *et al*., 2019), *Staphylococcus aureus* (*S. aureus*) (Kibenge and Wilcox, 1983), infectious bursal disease virus (Moradian *et al*., 1985), and chicken anaemia virus (McNeilly *et al*., 1995). However, no synergistic effect has been found between *M. synoviae* and ARV induced VA in turkey poults (Al-Afaleq *et al*., 1989).

Clinically, affected chickens with VA exhibited different degrees of uni and/or bilateral swelling of joints especially the hocks (tibiotarsal-tarsometatarsal), lameness, and difficulties in movement. When both joints are severely affected, the birds are completely immobilized. The morbidity rate is variable and may reach 10%, while the mortality rate is lesser (Judith *et al*., 2007). Affected breeder chicken flocks during egg production exhibit lameness, increased mortality, decline egg production, suboptimal hatchability or fertility, and vertical transmission of ARV to progeny (Jones and Georgiou, 1984).

On post-mortem examination, excessive turbid synovial fluid could be observed around the synovial membranes and the surrounding tissues. In the progressive VA cases, petechial hemorrhages on the synovial membranes and erosions on the articular cartilage may be also seen (Ballal *et al*., 1998; Mansour *et al*., 2018). Swelling and adhesion of the digital flexor tendons, rupture, and fibrosis of the surrounding tissues could be observed in heavy chicken breeds (van der Heide, 1977; McNulty, 1993; Rosenberger and Olson, 1997). Rupture of tendons is usually associated with haemorrhage which in turn causes green discoloration of the skin over the joint. Organs rather than joints could be affected following natural or experimental infection with ARV strains induced VA (Kerr and Olson, 1969; Roessler and Rosenberger, 1989). For example, lesions in the liver, heart, spleen, and bursa of Fabricius could also be observed in chicken flocks with a typical arthritis (Kerr and Olson, 1969; Tang *et al*., 1987; Hill *et al*., 1989). Un-related condition such as feathers abnormalities was also described in a previous report of VA (Rosenberger *et al*., 1989).

Microscopically, the tendon of the joint in VA cases revealed infiltration with lymphocytes, plasma cells, and few heterophils (Mansour *et al*., 2018) as well as thickening due to oedema and hyperplasia of the synoviocytes. The synovial membranes showed proliferation of villi and infiltration with inflammatory cells. In advanced cases of VA, the loose connective tissue surrounding the tendon sheaths could be replaced by fibrous tissue. Arthritis is multifactorial-dependent as many other bacterial infections such as *M. synoviae* and *S. aureus* may cause a similar disease. The pathological difference is considered a matter of degree (Kibenge and Wilcox, 1983). Hill *et al*. (1989) showed that the histological change due to reovirus was diffuse lymphocytic inflammation, while that caused by *Staphylococci* was focal purulent.

VA is regarded as an auto-immune disease that could be used as a model for rheumatoid arthritis in humans (Marquardt *et al*., 1983), despite absence of rheumatoid factor. Moreover, anti-nuclear antibodies (Pradhan *et al*., 1987) and anti-collagen antibodies (Islam *et al*., 1990) have been demonstrated in infected chickens.

Runting stunting syndrome

The RSS or MAS affects the gastrointestinal of broilers causing adverse economic losses due nutrients malabsorption, poor feed conversion ratio, low weight gain, growth retardation, stunting, non-uniform flock, and downgraded carcass quality (Barnes *et al*., 2000). Chickens of all ages are susceptible to RSS infection (Kang *et al*., 2012), however, young broilers up to 3 weeks of age are highly susceptible (Rebel *et al*., 2006).

Despite reovirus is considered one of the most important virus causing RSS, other different enteric viruses such as picornavirus (Lima *et al*., 2019; de Oliveira *et al*., 2021), rotavirus (Otto *et al*., 2012), astrovirus (Kang *et al*., 2018), coronavirus (Hauck *et al*., 2016), parvovirus (Zsak *et al*., 2013; Kapgate *et al*., 2018), and others may accompanied with a such complex disease.

Affected birds with RSS show diarrhea containing undigested food particles resulting in wet litter, low body weight gain, retarded and uneven growth, abnormal or helicopter shape feathers, loss of pigments in the form of pale shank, beaks, combs, and wattles, bone abnormalities, distended abdomens, and high morbidity rate (Page *et al*., 1982b; Zavala and Sellers, 2005; Rebel *et al*., 2006; Mansour *et al*., 2018). The mortality associated with RSS is either due to disability of the affected birds to reach the feed and water supplies or due to the disruption of food digestion and absorption (Rosenberger *et al*., 1989; Songserm *et al*., 2003).

The intestine of RSS affected chickens revealed pale serosa and presence of poorly digested food admixed with watery-mucoid or foamy contents (Nili *et al*., 2007; de Oliveira *et al*., 2021). The proventriculus might be dilated with enlarged and hemorrhagic glands, while the gizzard decreased in size with the presence of un-digested food particles (Page *et al*., 1982b; Mansour *et al*., 2018). The pancreas could also show pancreatitis, fibrosis, atrophy, and necrosis (Davis *et al*., 2013; Nunez *et al*., 2016; Mansour *et al*., 2018). Inflamed and congested kidneys maybe also observed (Elmubarak *et al*., 1990; Awandkar *et al*., 2017). Atrophy of the bursa of Fabricius, thymus glands, and spleen could be another signs (Hieronymus *et al*., 1983; Kouwenhoven *et al*., 1983). Stunted chickens showed severe emaciation, prominent keel bone, and pale breast muscles (Tang *et al*., 1987; Awandkar *et al*., 2017).

Microscopic examination of the small intestine (jejunum, duodenum, and ileum) of RSS affected broilers displayed cystic dilation of the crypt's lumen with flattening of the crypt's epithelium, reduced or atrophy of villous length, presence of inflammatory infiltrates, and decreased goblet cells (Qamar *et al*., 2013; de Oliveira *et al*., 2021). Moreover, degeneration, vacuolation, and fibrosis of pancreatic acinar cells, inflammation, and degeneration of proventricular glands and infiltration of macrophages and lymphocytes, as well as atrophy of bursa of Fabricius could be observed in some cases of RSS (Songserm *et al*., 2000; Qamar *et al*., 2013).

Diarrhea and excretion of essential nutrients in the droppings are the characteristic of RSS. This causes significant reduction in the serum components of such protein, albumin, globulin, iron, and calcium. Furthermore, affected birds couldn't adsorb dietary carotenoid pigments, vitamins, and other essential contents necessary for normal body growth and skin pigmentation. Impairment of enzymatic digestion prevents the release of pigments causing paleness of colour and this is termed as "pale bird syndrome" or "malabsorption syndrome". Moreover, this impairment may result in affection of the pancreas, intestinal tract, and proventriculus (Rebel *et al*., 2006). Moreover, feathering growth retardation and splitting of primary wings and tail feathers, resulting in loss of feathers with abnormal feathering pattern (Kouwenhoven *et al*., 1992). Fragile and brittle skeletons are also characteristics for RSS and this may be caused by decreasing in vitamin D3 absorption and exaggerated by the possibility of intestinal calcium being chelated to lipid and lost in the droppings (Khan *et al*., 1995). Reduction in the absorption of essential elements such as selenium may result in pancreatic fibrosis (Randall *et al*., 1981; Xu *et al*., 2017). Besides, depletion of selenium-dependent glutathione peroxidase enzyme in the pancreas and oxidative stress are predisposing factors to pancreatic atrophy (Denbow, 2015). Also, changes and atrophy of the lymphoid organs could be attributed to poor nutrient utilization (Khan *et al*., 1995).

New strains of ARV

In Muscovy ducks, ARV causes high morbidity and mortality and yellowish white necrotic foci on the liver with subcapsular hemorrhages, so the disease is known as "flower liver disease" (Yun *et al*., 2013; Zheng *et al*., 2016). Moreover, a new duck reovirus (NDRV) strain has been detected and caused "spleen necrosis disease of ducklings and goslings" which has been represented by haemorrhage and necrosis of the liver and spleen (Chen *et al*., 2012a,b; Bi *et al*., 2016). The first detection of NDRV was in China in 2005 (Pan *et al*., 2020). Cherry Valley duck, Shelduck, Muscovy duck, mule duck, duck, goose, and other waterfowl species can get the infection with NDRV (Wang *et al*., 2019). Young ducklings particularly at 5-25 days of age are highly susceptible with NDRV with morbidity rate 5-35% and mortality rate 2-20% (Pan *et al*., 2020). Intra-allantoic inoculation of 10-day-old embryonated chicken eggs with NDRV revealed delayed hatchability with necrosis of the liver and spleen of the embryos (Liu *et al*., 2016). Additionally, subcutaneous infection of 3-day-old chickens with NDRV induced loss of body weight, stunting, introfexion of claws, performing of splits, necrosis of the liver and spleen, and death (Yu *et al*., 2021). The virus can cause spleen necrosis, bursal atrophy, and immunosuppression with secondary bacterial infections.

Laboratory diagnosis

The signs and lesions induced by ARV infections are confusing, not diagnostic, and similar to many other bacterial or viral agents, therefore, laboratory isolation and identification are confirmative and considered the "gold standard" for diagnosis.

Samples could be taken from the tendon sheath, synovial fluid, and articular cartilage in case of VA, or from the droppings, bursa of Fabricious, liver, spleen, trachea, lungs, and kidneys in case of systemic ARV infection. The virus could be inoculated in the yolk sac of 5-7 day-old embryonated chicken eggs to produce embryonic death and lesions within 5 to 6 days of inoculation (Guneratne *et al*., 1982). When the virus is present at low concentrations in the tissues, 2-3 passages in eggs are essential to induce death or lesions (McNulty, 1993). After the 1st passage, inoculated embryos showed oedema with abdominal distention, cutaneous congestion, and greenish discoloration of the liver and allantoic fluid, while the 2nd passage induced necrotic foci on the liver and heart (Mansour *et al*., 2018). In addition, ARV can grow on fibroblasts, lung, liver, and kidney primary cell lines of chick embryos or chickens (Chen *et al*., 2011). The virus produces typical cytopathic effects in the form of syncytium in the cell sheet and the affected cells lifted off into the medium after a few days (McFerran *et al*., 1976; Guneratne *et al*., 1982). Intra-nuclear eosinophilic inclusion bodies are diagnostic after staining of the infected cells with haematoxylin and eosin. Electron microscopy is also used for the detection of ARV in the affected tissues following negative staining or immuno-florescent staining (Walker *et al*., 1972).

Direct immunofluorescent (IF) and virus neutralization (VN) tests are used for diagnosis of ARV antigen (Jones and Onunkwo, 1978; Wickramasinghe *et al*., 1993). Monoclonal antibodies in immunoperoxidase staining method is also used to detect ARV in paraffin-embedded sections (Liu and Giambrone, 1997). Staining techniques may be useful for the early diagnosis of ARV infection. Cross neutralization tests have been applied for the differentiation between strains of ARV (Kawamura and Tsubahara, 1966). Besides, the virus can be detected by using rapid and sensitive molecular techniques such dot-blot hybridization (Liu and Giambrone, 1996; Yin and Lee, 1998), polymerase chain reaction (PCR) (Xie *et al*., 1997; Tang *et al*., 2016), and restriction fragment length polymorphism (Liu *et al*., 1997; Lee *et al*., 1998; Liu *et al*., 1999). The later test have been also used to differentiate between the vaccinal and field strains of ARV. Serum samples are routinely tested for the detection of ARV antibodies using some serological tests (Giambrone *et al*., 2007) such as agar gel immunodiffusion (AGID) (Olson, 1980), VN (Kawamura and Tsubahara, 1966; Giambrone, 1980), indirect (IIF) (Ide, 1982), and enzyme linked immuno-sorbent assay (ELISA) (Slaght *et al*., 1978; Islam and Jones, 1988; Petrone-Garcia *et al*., 2021). The VN is a type-specific antibody test that differentiates between antigenically different strains of virus, while AGID, IIF, and ELISA detect group antigens. Strains of ARV possess group- and serotype-specific antigens; therefore, their neutralizing antibodies can be detected 7-10 days post-infection. Chicks at hatching should have a 1:1.600 or higher neutralizing maternal derived antibody titer against to give a protection against ARV field infection during the first 3 weeks of age (Takase *et al*., 1996). Additionally, Western blot method has been also used in diagnosis (Endo-Munoz, 1990). A high correlation between the level of antibodies in the egg yolk of laying chicken flocks and those in the serum was detected (Silim and Venne, 1989). Unfortunately, ARV could be isolated from the healthy birds, so antibodies in serum are often detected in both diseased and healthy birds (Jones, 2000).

Prevention

Management practices

Keeping the farms free from ARV infection is difficult due to several factors including the relative resistance of the virus in the environment, ubiquitous nature of infection, the possibility of vertical transmission, lack of detectable specific antibodies, and absence of the virus in the cloacal swabs. However, a good biosecurity and management procedures should be strictly adopted to minimize or reduce ARV infection at young ages.

Vaccination

Vaccination is regarded as the main and an important approach for the prevention of ARV infection. Apathogenic live and modified live vaccines as well as inactivated vaccines are available. Both apathogenic and inactivated vaccines are administrated subcutaneously, while a live modified vaccine is given orally. Vaccines of ARV are mainly used to prevent the vertical transmission, deliver maternal immunity to the progeny, and consequently prevent the infection of the young chicks (Sellers, 2017). It is recommended to vaccinate chicks using live ARV vaccines early as possible or immediately post hatching due to the high risk of early infection (Roessler and Rosenberger, 1989). Vaccination against ARV in broiler breeders is applied using live apathogenic vaccines (strain 2177), modified vaccines (strain S1133), and inactivated vaccines produced by patho-

genic reoviruses (S1133, 2408, SS412, and 1733 strains). Homologous autogenous ARV vaccine isolated from certain geographic region could be also used (Jones, 2000). Some studies showed that live vaccines failed to provide adequate protection against field virus challenge particularly when given at young age due to undeveloped poor intestinal immune response at this time (Chénier *et al*., 2014; Tang and Lu, 2015, 2016; Chen *et al*., 2019). The vaccinal strains are developed from lineage I, while the field VA/RSS strains are lineages from II to VI (Sellers, 2013, 2017). Therefore, there is no cross protection among these lineages (Tang and Lu, 2015). Additionally, vaccination with the same lineage could not offer sufficient protection to the flocks (Troxler *et al*., 2013).

The intestinal immunoglobulin (Ig) A develops in the gut of chicks at 7 and 21-day-old but not at day-old (Mukiibi-Muka and Jones, 1999). So, vaccination of adult breeders could be effective in providing young progeny with a sufficient passive or maternal immune response against ARV at day-old (van der Heide *et al*., 1976; Kibenge *et al*., 1987). Maternal immunity induced by ARV vaccination is primarily B-cell-mediated, while immune response following infection recovery is both B- and T-cell mediated. Rau *et al*. (1980) reported that an inactivated vaccine of ARV containing S11 33 strain induced a short life passive immunity. Thus, despite vaccination of broiler breeder chicken flocks against classical ARV, their progeny may show infection with the virus. The level of passive protection conferred by antibodies is correlated with serotype similarity, virus virulence, host' age, and antibody titer (Jones, 2000). CD8+ T cells have a major role in the intestinal clearance from ARV, while maternal immune cells do not play a significant role (Songserm *et al*., 2003).

Live attenuated vaccines of S1133 strain could be used to vaccinate broiler breeder chickens in the drinking water at 10 or 15 weeks of age (Eidson *et al*., 1979; van der Heide and Page, 1980). This vaccine could provide protection of the progeny chicks against homologous ARV strains only (Rau *et al*., 1980). Furthermore, vaccination of laying hens with the previous vaccine decreased the hock joints lesions in the challenged progeny at day-old of age (Jones and Nwajei, 1985). Priming with a live ARV vaccine at early stage of life followed by boostring with inactivated vaccine at 6-week-old and before egg production provoked high and persisted levels of maternal immunity (Giambrone, 1985). Nevertheless, the results of Petrone-Garcia *et al*. (2021) indicated that vaccination of broiler chickens having maternal antibodies with a live S1133 ARV strain resulting in pathological disruptions of the gastrointestinal integrity (proventriculous, intestine, and pancreas) and decreasing in performance parameters.

Giambrone and Hathcock (1991) demonstrated the efficacy of using a coarse-spray of a cell culture clone of strain S 1133/66 vaccine in providing higher antibody titers than egg-passaged vaccine. Bivalent or trivalent inactivated vaccine containing ARV, Newcastle disease virus, and egg drop syndrome 1976 virus have been also used for vaccination of breeders' flocks. Immunization-challenge experiment using *Escherichia coli*-expressed sigma-3 protein of ARV in chicks has been demonstrated. van der Heide *et al*. (1983) found that the incidence of Marek's disease has been increased following vaccination of day-old chicks with herpesvirus of turkeys (HVT) and ARV vaccine. In addition, chickens' condemnation rates were higher following vaccination with a combined HVT and ARV vaccine when compared to chickens given HVT vaccine alone (Rinehart and Rosenberger, 1983). It has been reported that presence of maternal derived reovirus antibodies in chicks derived from vaccinated breeder hens resulting in interference with active immunization against some other viral infections (Adriaan *et al*., 2003) such as Newcastle disease (Awandkar *et al*., 2017). Vaccination failure against ARV infection is common because infection with the variant field strains is refractory to the immunity induced by classical vaccine strains (Palomino-Tapia *et al*., 2022).

Conclusion

Since ARV is widely distributed and circulated in commercial poultry flocks without clinical manifestations in some cases, more research work is required to underline the pathogenesis of infection and detect the causative agent. Pathogenic strains of ARV are associated with important disease conditions such as VA, RSS, and others that adversely affect the poultry production system. Therefore, development of new vaccines to cope with the new emerging mutant field strains of ARV is the must. Finally, more surveillance programs using recent molecular techniques of diagnosis should regularly adopted to understand the disease situation.

Conflict of interest

The author declares that there is no conflict of interest.

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