

Pigeon Paramyxovirus-1 Infection and the Public Health Importance: A Review Article

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Abstract. *Pigeons are highly susceptible to Newcastle disease virus (NDV) infection, which causes economic losses in terms of increased mortalities, immunosuppression, vaccination costs, and probable trade restrictions. NDV belongs to the avian paramyxovirus serotype 1 (APMV-1). The antigenic variant of APMV-1 is termed as pigeon paramyxovirus type 1 (PPMV-1), which is classified in the genus Avulavirus of the subfamily Paramyxovirinae and family Paramyxoviridae. Infections of pigeons with PPMV-1 have been detected since the 1930s, and the virus is still circulating in many countries until now. Domestic, feral, and racing pigeons, doves, and exotic birds are susceptible to PPMV-1 infection. The virus is rapidly spreading between birds through the horizontal route. Infected pigeons may show circling, ataxia, torticollis, head and neck tremors and twisting, leg and wing paralysis, greenish diarrhea, respiratory signs, and polyuria. Some pigeons could be infected with PPMV-1 without apparent signs, and they act as reservoirs for other domestic or free-living birds. The diagnosis of suspected PPMV-1 cases is based mainly on the isolation and identification of the virus, serological detection of specific antibodies, and molecular characterization of the virus. Adoption of strict biosecurity measures as well as vaccination using traditional live or inactivated NDV or even the specific PPMV-1 vaccines are the gold standard methods for preventing pigeons from such infection. Therefore, this review article was designed to focus on PPMV-1 infection regarding the virus characteristics, epidemiology, diagnosis, human infection, and control.*

Introduction

Newcastle disease (ND) is an acute, highly contagious, and endemic viral disease of poultry worldwide (Abdisa & Tagesu, 2017; Suarez et al., 2020). Respiratory, gastrointestinal, and neurological manifestations are the main clinical pictures of ND infections. The disease is associated with significant global economic losses in the poultry industry, including high morbidity and mortality rates, immunosuppression, increasing the costs of vaccinations and control, and probable trade restrictions (Alexander, 2001; Ganar et al., 2014). The World Organization for Animal Health included ND in the list (A) of notifiable diseases (OIE, 2012). It has been reported that about 236 free-living avian species could be infected either naturally or experimentally with ND (Kaleta & Baldauf, 1988).

Avian paramyxoviruses have been divided into 12 different serotypes based on hemagglutination inhibition (HI) and neuraminidase inhibition assays (Dimitrov et al., 2016). Newcastle disease virus (NDV) belongs to avian paramyxovirus serotype 1 (APMV-1) in the family *Paramyxoviridae* and the genus *Avulavirus* (Cox & Plemper, 2017). The virus is a single-stranded, negative-sense, and non-segmented RNA genome that contains 6 genes in the sequence of 3'-NP-P-M-F-HN-L-5' (Gogoi et al., 2017; Dimitrov et al., 2019). APMV-1 is circulating as an enzootic infection in many continents, such

as Europe, Africa, Asia, and America (Miller et al., 2010). Infection with APMV-1 has shown variation in pathogenicity, from asymptomatic to lethal disease, due to the significant differences in strains virulence (Heiden et al., 2014). Three major pathotypes of NDV have been recognized in poultry based on the degree of the pathogenicity indices. They include the mean death time (MDT), intracerebral pathogenicity index (ICPI), and intravenous pathogenicity index (IVPI). Apathogenic NDV strains are non-virulent and show enterotropism, while lentogenic strains are of low virulence and produce mild respiratory manifestations (Alexander, 1997). Mesogenic strains of NDV are of moderate pathogenicity and infect mainly the respiratory tract, causing death in birds under 8 weeks of age (Beard & Hanson, 1984), while highly virulent velogenic strains (viscerotropic and neurotropic) induce systemic infections and a high mortality rate (Susta et al., 2011; Miller & Koch, 2013).

Pigeons could be divided into 3 categories: meat-type pigeons, homing pigeons, and fancy pigeons. Recently, the pigeon industry has shown rapid development, but the disease situation caused by viral affections is not helping and is not hopeful. Owing to the migratory nature of pigeons, difficulties in vaccinations, and their existence in live bird markets and backyard houses, they are regarded as a major threat for NDV transmission to domestic chickens. APMV-1 infects almost all avian species, including pigeons. Therefore, pigeons are regarded as a natural host of APMV-1 and play an important role in the

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virus ecology (Pestka et al., 2014). ND in pigeons is called paramyxovirus and is caused by an antigenic "pigeon variant" of the virus (pigeon paramyxovirus type 1, PPMV-1). PPMV-1 is an antigenic variant of APMV-1, which is mainly associated with infection of pigeons (Pestka et al., 2014). Since the genotype VI of NDV is frequently detected in pigeons, this strain is commonly named PPMV-1 (Alexander et al., 1985b; Tian et al., 2020; Xie et al., 2020). Genotypes VI, XX, and XXI of NDV are sometimes assigned as PPMV-1, which is regarded as the most genetically diverse group among the virus strains (Dimitrov et al., 2016). Despite the fact that PPMV-1 is responsible for severe disease conditions in domestic pigeons (Alexander, 2009), it occasionally spreads to a wide variety of other species, including feral pigeons, doves, and exotic birds (Alexander, 2000). Nowadays, PPMV-1 has been reported as an enzootic infection of feral, racing, and fancy pigeons (Alexander, 2011). Young pigeons are more susceptible to PPMV-1 and show neurological disorders with high morbidity and mortality rates (Chang et al., 2021; Badr et al., 2022). The central nervous system, respiratory system, alimentary tract, and kidney are usually affected by PPMV-1. Routine diagnosis of PPMV-1 is based on primary isolation in specific pathogen-free (SPF) embryonated chicken or pigeon eggs or on tissue culture (Gough et al., 1988), followed by a hemagglutination (HA) assay. Confirmation of infection should be carried out using conventional serological tests such as HI test or molecular-based techniques (OIE, 2021).

Infection of humans with APMV-1 is usually rare and may cause an asymptomatic condition or a mild disease, especially in immune-competent people (Capua & Alexander, 2004). The direct contact of humans with diseased pigeons may lead to conjunctivitis or, rarely, to flu-like symptoms (Zehetbauer et al., 1971; Schemera et al., 1987). In severe cases, infected people may show long-term vision damage (Beard & Hanson, 1984; Lamb & Parks, 2007). The disease usually shows self-limiting symptoms, which develop within 24 hours of exposure to PPMV-1 and resolve within a week (Swayne & King, 2003). Though the zoonotic potential of PPMV-1 is low, veterinary authorities urge people not to touch diseased or dead pigeons.

Despite vaccination, PPMV-1 is still enzootic in pigeons in some countries (Alexander, 2001). The gold standards for the prevention of PPMV-1 infection in pigeons are the administration of vaccines (Vindevoel & Duchatel, 1985; Zhao et al., 2010; Soliman et al., 2019), along with the adoption of effective biosecurity measures.

From the abovementioned, this review article was designed to focus on PPMV-1 infection in regards to the virus characteristics, epidemiology, diagnosis, human infection, and control.

The virus

According to the International Committee on Taxonomy of Viruses, *Orthoavulavirus 1* (NDV) belongs to the order *Mononegavirales*, family *Paramyxoviridae*, subfamily *Avulavirinae*, and genus *Orthoavulaviruses* (ICTV, 2020). It is an enveloped virus with single-stranded RNA that is linear, pleomorphic (mostly spherical), non-segmented, and has a negative polarity (Amarasinghe et al., 2019). The genome of NDV is around 15–19 kilobases in length and encodes 6 structural proteins: nucleocapsid protein (NP), phosphoprotein (P), matrix (M) protein, fusion (F) protein, hemagglutinin-neuraminidase (HN), and RNA large polymerase (L) protein in the following order of 30-NP-P-M-F-HN-L-5 (Czegledi et al., 2006). Besides, the gene P encodes 2 additional non-structural (V and W) proteins by RNA editing (Steward et al., 1993; Karsunke et al., 2019). Both F and HN glycoprotein spike projections on the virus envelope are important for infection, fusion, and propagation of most paramyxoviruses in the host cell (Lamb & Parks, 2007). Moreover, the F protein is regarded as a key factor in NDV pathogenicity (Panda et al., 2004). Other internal proteins are involved in some functions, including transcription and replication of the virus (Dimitrov et al., 2016; Gogoi et al., 2017).

Although APMV-1 has a single serotype, the phylogenetic characterization of F gene sequences reveals the presence of 2 distinct classes (class I and class II) (Diel et al., 2012). Both classes are consequently subdivided into many different genotypes and clades. Class I of the virus consists of a single genotype, and it is mostly isolated from natural reservoirs of free-living wild and domestic waterfowl without pathogenicity to chickens (Kim et al., 2007; Chen et al., 2021; Lu et al., 2021). Class I virus strains are characterized by a 15,198-nt genomic RNA. On the other hand, class II can be separated into 21 genotypes (I to XXI, no XV) and represents the major cause of the disease outbreaks all over the world (Diel et al., 2012; Courtney et al., 2013; Dimitrov et al., 2019). Class II viruses show variable pathogenicity (both avirulent and virulent strains), as genotypes V, VI, VII, VIII, and XI–XVIII are the most pathogenic ones (Snoeck et al., 2013). Two new genotypes of XX and XXI and some strains of genotype VI have been designated as variant NDV strains, particularly in Columbiformes (Dimitrov et al., 2019). The strains of PPMV-1 are clustered into genotypes VI and XXI (Aldous et al., 2004). Genotype I–IV viruses are early lineage with a genome size of 15 186 nt, while genotype V–VIII viruses are recent lineage with a genome of 15 192 nt (Ujvari et al., 2006). Moreover, class II genotype VI is further divided into VIa–VIg. Moreover, Aldous et al. (2003) defined 6 lineages (class I viruses were lineage 6) with many sub-lineages. PPMV-1 viruses are placed in class II and lineage 4b or VIb, depending

on the used nomenclature. NDV has a specific amino acid sequence at the fusion protein gene cleavage site [“multiple basic amino acids (positions 113–116) at the C-terminus of the F2 protein and phenylalanine at position 117 at the end of the F1 protein”] (OIE, 2021). At the cleavage location of the F0 precursor, the most virulent strains of NDV has a sequence of “112R/K-R-Q-R/K-R*F117”, in comparison with avirulent strains that have “112G/E-K/R-Q-G/E-R*L117”, which is considered as a major determinant of the virus virulence (Collins et al., 1993). Therefore, the virulence of NDV strains could be determined through the F protein cleavage site amino acid sequence analysis (Le et al., 1988), as well as through the ability of separation of specific cellular proteases to cleave the F protein pathotypes (Ogasawara et al., 1992). The World Organization for Animal Health has defined virulent NDV as APMV-1 with at least 3 multiple basic amino acid residues (arginine or lysine) at the C-terminus of the F2 protein (between residues 113 and 116) and phenylalanine at residue 117, which is the N-terminus of the F1 protein (Alexander, 2008, 2011). It has been documented that lentogenic or avirulent NDV strains have fewer basic amino acids in the F protein cleavage site than either mesogenic or velogenic isolates, which have similar cleavage site sequences (Glickman et al., 1988).

Epidemiology

Distribution

Despite the fact that infections of pigeons with PPMV-1 have been detected since the 1930s (Doyle, 1933), the 3rd NDV panzootic started during the late 1970s (Yang et al., 1999) and is present in many countries until now. Around 1979, PPMV-1 was also reported in the Middle East (Kaleta et al., 1985a), and then the virus strains were detected in some European countries (Alexander et al., 1985a, b). In 1984, feedstuff was contaminated by feral pigeons in the docks in Liverpool, United Kingdom, resulting in 19 NDV outbreaks due to PPMV-1 in chickens (Alexander et al., 1985a). Moreover, outbreaks of PPMV-1 in commercial and backyard poultry have been reported in Brazil (Zanetti et al., 2001; Souza et al., 2018; Thomazelli et al., 2021; Pereira et al., 2022), Poland (Smietanka & Minta, 2011), Ireland (O'Reilly et al., 1994), Germany (Werner et al., 1999), Switzerland (Alexander et al., 1985b; Annaheim et al., 2022), Slovenia (Krapez et al., 2010), Macedonia (Dodovski et al., 2013, 2017), Japan (Mase & Kanehira, 2015), China (Liu et al., 2006; Wang et al., 2015; Qiu et al., 2017; Wei et al., 2018; He et al., 2020; Tian et al., 2020; Xie et al., 2020; Zhan et al., 2022), South Africa (Pienaar & Cilliers, 1987; Abolnik et al., 2004, 2008), Nigeria (Snoeck et al., 2013), Iran (Mayahi et al., 2017; Rezaei Far et al., 2017), and Egypt (Mohamed et al., 1980; Ahmed & Sabri, 1989; Shakal, 1989; Abou Hashem, 1993;

Ibrahim et al., 2005; Soliman et al., 2016; Mansour et al., 2017).

Susceptibility

All ages of pigeons are susceptible to PPMV-1 infection with high morbidity and mortality rates (Kim et al., 2008; Qiu et al., 2017; Tian et al., 2020). Not only domestic pigeons are susceptible to PPMV-1, but also feral pigeons may carry the virus as asymptomatic carriers (Teske et al., 2013; Mase & Kanehira, 2015). Feral or wild pigeons have shed PPMV-1 during foraging in rural areas and posed a potential threat to the spread of the disease to free-range chickens (He et al., 2018). The PPMV-1 isolate that belongs to sub-genotype XXI.2 has been reported in collared doves in Italy (Bonfante et al., 2012) and in Eurasian collared doves in Iran (Esmaeelzadeh-Dizaji et al., 2022). PPMV-1 has also been isolated from wild North American doves (Kim et al., 2008). The study of Smietanka et al. (2014) has shown that PPMV-1 strain was highly virulent to pigeons, followed by chickens and turkeys, but quails and geese showed the highest level of innate resistance to the used strain. Nevertheless, the passing of PPMV-1 in chickens has resulted in increased ICPI. There is no evidence of PPMV-1 infections in wild non-Columbiformes. However, the virus is occasionally isolated from migratory birds (Alexander et al., 2012).

Transmission

PPMV-1 could be excreted in the nasal, buccal, and ocular secretions and droppings of the infected pigeons. The virus is transmitted horizontally among birds either by inhalation or ingestion (Kaleta et al., 1992). Direct contact between healthy and diseased birds helps in the rapid spread of infection. The rapid transmission of PPMV-1 among Columbiforms may also be possible during competition flights (racing pigeons), exhibitions (show pigeons), live bird markets (meet pigeons), or intensive trade (Aldous et al., 2014; Sabra et al., 2017).

Clinical picture

The incubation period of PPMV-1 infection is 7–14 days. The clinical picture of infection varies according to the virulence of the infective strain, the immune status of the host, and the presence of other infections (Alexander & Senne, 2008). Pigeons show clinical signs similar to neurotropic NDV-infected chickens. Affected pigeons may display moderate to severe depression, circling, ataxia, torticollis, head and neck tremors and twisting, leg and wing paralysis, greenish diarrhea, and respiratory manifestations (Vindevogel & Marlier, 2006; Wang et al., 2015; Badr et al., 2022). Viscerotropic strains of PPMV-1 exhibit specific affinity for the kidneys; thus, the first observed sign is polyuria, and neural symptoms appear only in individual birds (Pestka et al., 2014). However, an asymptomatic course of infection may occur (Alexander et al., 1984a). Infection with PPMV-1 may cause a mortality rate that ranges from 10% to

70%, as does the morbidity, which ranges from 30% to 80% in the infected pigeons (Hutchison, 1984; Mansour et al., 2017; Qiu et al., 2017). Concurrent infections with other bacterial or parasitic infections increase the mortality rate. Survived pigeons may shed the virus and, therefore, represent a source of infection (Alexander et al., 1984b). The post-mortem lesions of dead pigeons with PPMV-1 have revealed soft or friable tissues of the brain and/or hemorrhages, severe hemorrhagic enteritis, petechial hemorrhage in the gizzard, and congested liver (Dodovski et al., 2017; Badr et al., 2022). It has been documented that PPMV-1 in chickens is of intermediate virulence (a mesogenic virus) (Collins et al., 1994; Hüppi et al., 2020), and the infection may vary from subclinical to a marked drop in egg production (Guo et al., 2014). Though PPMV-1 strains sometimes cause mild disease, multiple passages in chickens may result in increasing virus virulence in the form of signs, mortality, and neuro-invasiveness (Kommers et al., 2001, 2003; Dortmans et al., 2011). Moreover, many NDV outbreaks in chickens have been attributed to PPMV-1 infection (Alexander et al., 1984b; Werner et al., 1999). Several point mutations are sufficient to increase the pathogenicity of PPMV-1 in chickens (Meulemans et al., 2002; Dortmans et al., 2009). The histopathological examination of the examined brains of PPMV-1-infected pigeons has revealed the presence of non-suppurative encephalitis, necrosis, and microgliosis (Wakamatsu et al., 2006; Pereira et al., 2022). Diffuse inflammation of the respiratory and intestinal tract tissues has also been detected (Aldous & Alexander, 2001).

Human infection

Infection of humans with APMV-1 is rarely detected, and it is not considered a life-threatening condition so far. However, sporadic infections have been reported in patients with occupational exposure to commercially infected poultry flocks (Capua & Alexander, 2004). Most of the APMV-1 human cases have occurred through direct contact with infected birds; mostly, workers in poultry farms, laboratories, and processing plants are at a high risk of getting infections. The clinical signs are usually transitory mild conjunctivitis, and most of the patients resolve without medical or clinical interference (Steele & Beran, 1981). Frequent symptoms of eye disorders (Lippmann, 1952; Capua & Alexander, 2004), unilateral or bilateral eye redness, eyelid edema, conjunctivitis, sub-conjunctival hemorrhage, and acute keratoconjunctivitis have been reported in a case with concurrent NDV and human adenovirus infection (Prajna et al., 2021). Nevertheless, lethal pneumonic studies of APMV-1 have been found in immune-compromised persons following blood stem cell/allogeneic bone marrow transplantations (Goebel et al., 2007; Kuiken et al., 2018). Another child case caused by APMV-1 has shown fatal encephalitis after

hematopoietic stem-cell transplantation (Winter et al., 2021). The strain of APMV-1 isolated from a pneumonic patient was also of pigeon origin (Goebel et al., 2007). Kuiken et al. (2017) have found that PPMV-1 from a fatal human case induced pneumonia in experimentally infected *Cynomolgus* macaques. Moreover, APMV-1-associated pneumonia and death have been detected in a 64-year-old person following contact with a live pigeon (Abbo et al., 2007). Following extensive laboratory investigation of the previous case, both APMV-1 and *Acinetobacter baumannii* (ABA) have been detected. Recently, a similar case was reported in 2020 in China, where a 64-year-old man presented with severe acute respiratory distress syndrome and sepsis, followed by death within a few days (Zou et al., 2022). The patient had close contact with pigeons before illness, and PPMV antibodies were detected in his blood within 20 days of illness. Besides, the metagenomic sequencing revealed the presence of ABA and APMV-1 genotype VI.2.1.1.2.2 in the broncho-alveolar lavage fluid, and the virus nucleic acid was found in the pigeon feathers. This study emphasizes the cross-species transmission of PPMV-1 between infected pigeons and humans.

Diagnosis and differential diagnosis

The primary isolation of PPMV-1 could be achieved via inoculation of SPF embryonated chicken or pigeons eggs. The embryos die between the 2nd and 8th day of inoculation and show curling and sometimes dwarfing with head and body hemorrhages as well as presence of urates even mixed with allantoic fluids. The HA of allantoic fluid may confirm the presence of PPMV-1. Tissue culture, including chicken embryo fibroblasts, chicken primary neuronal cells, pigeon embryo fibroblasts, and pigeon primary neuronal cells have been used for primary isolation of PPMV-1 (Guo et al., 2018; Zhan et al., 2020). Serological tests, particularly the HI assay, have been used for detection of antibodies in the serum of PPMV-1 infected pigeons (Ibrahim et al., 2005).

The pathogenicity of the virus could be determined by assessing the MDT in 10-day-old SPF embryonated chicken or pigeon eggs and the ICPI in 1-day-old SPF chicks based on standard procedures (OIE, 2012). Virulent NDV strains are those with MDT less than 60 hours and ICPI values of 0.7 or more (defined as the mean observed score per bird as 0 if normal, 1 if diseased, or 2 if dead over the 8-day period) (Dortmans et al., 2009, 2010). The IVPI of PPMV-1 could be determined after inoculation of freshly infective allantoic fluids into a 6-week-old pigeon, as each bird should be examined daily for a period of 10 days and scored for assessment of the virus virulence (mesogenic, velogenic, etc.).

The molecular identification of PPMV-1 has been comprehensively carried out using polymerase chain

reaction (Aldous et al., 2004, 2014; Wei et al., 2018; Esmaelzadeh-Dizaji et al., 2022). Virulent PPMV-1 strains have a specific amino acid sequence at the F protein gene cleavage site (amino acids positions 113–116 at the C-terminus of the F2 protein and phenylalanine at position 117 at the end of the F1 protein) (Dimitrov et al., 2017; OIE, 2021).

Signs of PPMV-1 in pigeons are similar to pigeon herpes virus infection, sodium chloride poisoning, and overdose of ronidazole or vitamin B1 deficiency. Therefore, laboratory tests are essential for an accurate diagnosis (Hamouda et al., 2017).

Control

There is no specific treatment for PPMV-1 infection because infected birds usually die within 72 hours; however, some recovered birds may survive with supportive treatment. Control of PMV-1 infection can be achieved via the adoption of strict biosecurity measures and a proper vaccination regimen (Miller & Koch, 2013). Vaccination of pigeons could reduce the losses and the hazards of the spread of infection to other pigeons and/or other bird species. Pigeons could be protected against infection using chicken NDV-live or -inactivated oil-emulsion vaccines (Viaene et al., 1984). Ibrahim et al. (2005) have found a close immunogenic relationship among PPMV-1 and NDV vaccine strains, which may answer the question of why we use NDV vaccines for controlling PPMV-1 infection in pigeons. Immunization of commercial or racing pigeons with inactivated APMV-1 vaccines is also important (Alexander et al., 1985b). Lentogenic strains of NDV showed some virulence for pigeons and might be excreted 3–7 days post inoculation; thus, inactivated vaccines are preferred for racing pigeons (Vindevogel et al., 1982). Viaene et al. (1983, 1984) reported that LaSota and oil-inactivated NDV vaccines provided protection for pigeons against PMV-1 infection for up to 6 months. However, it has been reported that the Hitchner B1 live NDV vaccine could not protect pigeons against the virus as it could not propagate in pigeon tissues and thus could not induce suitable immunity (Kaleta et al., 1985b). Alexander et al. (1986) demonstrated that vaccination of pigeons against PPMV-1 was not analogous to vaccination against NDV in chickens due to the differences in the hosts or the antigenic variations between pigeons and classical PPMV-1 strains, including the vaccinal viruses.

Homologous vaccine is necessary for a complete protection of pigeons against the disease (Fritzsch et al., 1984; Eskelund, 1986; Stone, 1989; Amer, 2008). Vaccinated pigeons with a homologous oil emulsion vaccine against PPMV-1 infection were more highly protected than pigeons vaccinated with commercial live NDV vaccines for a year (Alexander and Parsons, 1984; Amer et al., 2013). Inactivated NDV vaccines provoked a relatively high antibody titer, which was sufficient to protect pigeons against PPMV-1,

while the Hitchner B1 live vaccine did not (Polten et al., 1985; Kusters et al., 1986). Duchatel and Vindevogel (1986) found that inoculation of 0.2 mL of inactivated aqueous-suspension vaccines prepared from the LaSota strain of NDV gave a high resistance to a severe challenge with PPMV-1. Box et al. (1985) suggested using double doses of an inactivated oil-emulsion vaccine 4 weeks apart to provide a good protection for pigeons. Inactivated vaccines from the local PPMV-1 strain were developed, and the results revealed a protection rate of 80% with a high level of immune response that lasted for 5 months (El-Zanaty et al., 1992; Hassan, 1997). Wawizkiewicz et al. (1991) demonstrated that the oil emulsion PPMV-1 vaccine gave a higher HI antibody response on the 3rd week after the 2nd vaccination dose. Besides, Amer et al. (2013) have reported that the PPMV-1 vaccine gave 100% protection for vaccinated pigeons against the homologous virus compared with only 10% protection in non-vaccinated birds. A combined inactivated vaccine containing oil adjuvanted local strains of PPMV-1 and *Salmonella typhimurium* is safe, potent, and provides a full protection in terms of increasing the HI antibody titers and reducing mortality post-challenge (Khedr et al., 2016). These findings suggest that sub-strains may exist in the PPMV-1, which necessitates the continuous update of the vaccine master seeds with new field isolates to maintain high protection level (Soliman et al., 2019). Subcutaneous inoculation of pigeons with an aluminum hydroxyl-based formula of the PPMV-1 YA/14 vaccine provides a higher humoral immune response with a lower shedding level than the oil-based vaccine (Soliman et al., 2019).

Conclusion

PPMV-1 infection is widespread; therefore, prevention of the pigeon's infection looks significant and needs thorough investigation. Besides, the mechanisms by which APMV-1 causes severe human infection should be more thoroughly explored. Pigeons may not show clear signs of PPMV-1 infection or deaths, but they can catch the virus and induce specific antibodies. Despite vaccination, PPMV-1 is still enzootic in pigeons in certain countries. Vaccination of pigeons with the NDV vaccine or a specific PPMV-1 vaccine gains attention to avoid the virus shedding to other birds. The use of a homologous vaccine is the most suitable solution for the control of such infection. Personnel should wear protective clothes and equipment when handling and processing potentially infected poultry. The surveillance studies of PPMV-1 are necessary to prepare health authorities better.

Conflict of Interests

The author declare that there is no conflict of interests.

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