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PARVOVIRUS INFECTION: A THREAT TO DOMESTIC WATERFOWL INDUSTRY

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ABSTRACT

Parvovirus infection affects geese and ducks, causing an acute, contagious, and fatal disease known as gosling plague or Derzsy's disease and Muscovy duck parvovirus infection. Moreover, a novel goose parvovirus was first characterized in mule ducks and Cherry Valley ducks and causes a disease termed short beak and dwarfism syndrome. Parvovirus infection in waterfowl has gained worldwide attention due to the substantial economic losses in commercial production. The virus is a member of the genus *Dependoparvovirus* of the family *Parvoviridae*. It infects mainly goslings and ducklings during the first 3 weeks of life, causing high morbidity and mortality rates, and belongs to locomotor disturbance, growth retardation, and diarrhea. Transmission of parvovirus in geese and duck flocks occurs either through the horizontal or vertical route. The most common findings of parvovirus in affected birds are myocardial degeneration, intestinal necrosis, myocarditis, perihepatitis, ascites, and sometimes nervous system affection. Diagnosis of parvovirus depends mostly on isolation and identification of the virus using conventional methods, detection of the virus using recent molecular techniques, and seroconversion. Protection of ducklings or goslings in the early life via passive maternal immunity is regarded as the most suitable method for the prevention of parvovirus infection. Vaccination of breeder flocks and their offspring is required to decrease the disease impact. Live, inactivated, and recombinant vaccines have been used against such infection. In conclusion, this article discussed parvovirus infection in waterfowl regarding the virus characteristics, susceptibility, transmission, clinical picture, laboratory diagnosis, and prevention and control of such infection.

Keywords: Goose parvovirus, Derzsy's disease, Muscovy duck parvovirus, short beak and dwarfism syndrome

INTRODUCTION

Raising domestic waterfowl requires the presence of water bodies, which may enhance the mixing with free-living waterfowl species and promote disease transmission. Therefore, they are susceptible to many serious viruses that have adverse effects on production (Abd El-Ghany, 2021).

Goose breeding is done in some countries, such as Egypt, China, Turkey, Romania, Ukraine, Poland, Hungary, and Madagascar (Tilki & Saatci, 2016). Parvovirus (PV) of waterfowl causes a dreadful disease in rearing farms of goslings (*Anser anser*

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domestica) and Muscovy ducklings (*Cairina moschata*) (Palya, 2013). Infections with parvovirus have been reported in Asia, Europe, and America, where goose and duck production is common (Jansson *et al*., 2007; Wan *et al*., 2016a; Liu *et al*., 2017; Soliman *et al*., 2020; Kardoğan *et al*., 2021). Despite vaccination against PV since the last 30 years, it still regarded as the most common infection with striking losses in goose farms (Tarasiuk *et al*., 2019). The virus caused high morbidity and mortality in goslings and Muscovy ducklings in 1960, which was reported as Derzsy's disease or gosling plague (Derzsy, 1967). This disease was termed as goose PV (GPV) in 1978. However, in China, in 1989, Muscovy ducks showed signs like those of GPV (Lin *et al*., 1991; Le Gall-Recule & Jestin, 1994) and the infection was then termed as Muscovy duck PV (MDPV) or (three-week disease) based on 85% protein sequence homology to GPV. Then, MDPV infection was distributed in different countries (Jestin *et al*., 1991; Takehara *et al*., 1994; Woolcock *et al*., 2000; Glávits *et al*., 2005; Poonia *et al*., 2006). In south-western France in the 1970s (Villatte, 1989), a novel goose parvovirus (NGPV) was firstly detected in Mulard (mule) ducks (crossbreed of Pekin duck and Muscovy duck) and Cherry Valley ducks with retarded growth, atrophied beak, protruded tongue, and 15-25% morbidity rate and the disease was termed as short beak and dwarfism syndrome (SBDS) or beak atrophy and dwarfism syndrome (BADS) (Palya *et al*., 2009; Li *et al*., 2016; Bian *et al*., 2019). The GPV isolated from SBDS shares a partial antigenic relationship with the classical GPV strain and it can be considered as a variant GPV (Li *et al*., 2016; Ning *et al*., 2017). Repeated outbreaks of SBDS were then detected in Taiwan (Lu *et al*., 1993), Poland (Samorek-Salamonowicz *et al*., 1995; Wozniakowski *et al*., 2009a), Hungary (Palya *et al*., 2009), mainland China (Chen *et al*., 2015, 2016b; Yu *et al*., 2016), and Egypt (Soliman *et al*., 2020). Cherry Valley ducks, mule ducks, Muscovy ducks, Pekin ducks, and partridge ducks are susceptible to SBDS caused by NGPV (Huang *et al*., 2015; Wan *et al*., 2019a; Saleh & Khodier, 2020; Soliman *et al*., 2020). Moreover, goslings have been found to be infected with NGPV, but appear as asymptomatic carriers of (Xiao *et al*., 2017).

Both GPV and MDPV are members of the species *Anseriform dependoparvovirus* 1 in the genus *Dependoparvovirus* of the subfamily *Parvovirinae* and family *Parvoviridae* (Wan *et al*., 2018; Cotmore *et al*., 2019). PV is a single-strand DNA genome of about 5 kb in length. Despite there is approximately 85% identity in protein sequences of GPV and MDPV, limited cross-protection between both viruses has been observed. Transmission of PV is relied either on the direct or indirect contact with infected waterfowl or on the vertical transmission (Gough, 2008; Palya, 2013). Infection with PV is chiefly age dependent and poses a constant threat to waterfowl production as represented by decreasing performance, retardation of growth, and increasing mortality (Chen *et al*., 2015; Soliman *et al*., 2020). Muscovy ducklings infected with MDPV between 2 and 4 weeks old usually show locomotor dysfunction, ataxia, wheezing, watery diarrhea, growth retardation, and stunting, along with a morbidity rate of 27-62% and a mortality rate of 20-55% (Lin *et al*., 1991; Chen *et al*., 1993; Glávits *et al*., 2005). Skeletal muscle myopathy, myocarditis, hepatitis, neuritis, polioencephalomyelitis, and atrophy of lymphoid organs are the most characteristic lesions in geese and ducks (Glávits *et al*., 2005). Diagnosis of PV is based on isolation in goose or ducks eggs and propagation on embryonic fibroblast cell cultures. Rapid detection of the virus using molecular techniques has been effectively applied as sensitive and specific tests (Bulut *et al*., 2021; Isidan *et al*., 2021). Protection against infections caused by PV is important to prevent serious economic losses in geese and duck flocks. Therefore, many types of living

attenuated vaccines have been used to protect goslings from GPV infection (Fang *et al*., 1987; Wang *et al*., 2014a).

In the present review article, PV infection in waterfowl has been discussed regarding the virus characteristics, susceptibility, transmission, clinical picture, laboratory diagnosis, and prevention and control of such infection.

The virus

According to the virus neutralization tests and genetic analysis, waterfowl PV can be divided into GPV and the MDPV (Poonia *et al*., 2006). They belong to *Dependo-parvovirus* genus of the *Parvoviridae* family, and all of them are members of the same *Anseriform dependoparvovirus* 1 species (Walker *et al*., 2019). Within the GPV-related group, 4 subgroups are investigated: the Hungarian virulent strains, the vaccine and low pathogenic strains, the West-European strains, and the Asian strains (Tatár-Kis *et al*., 2004; Chen *et al*., 2015; Yu *et al*., 2016; Ning *et al*., 2017). PV is a single-stranded and non-enveloped DNA virus of approximately 5-5.1 k nucleotides in length and 20-22 nm diameter (Wang *et al*., 2016). The inverted terminal repeats (ITRs) at each end of GPV and MDPV genomes have lengths of approximately 442 and 456 nucleotides, respectively (Wang *et al*., 2016). The ITRs act as start sites for genome replication.

Complete genomic sequence of GPV strains isolated from different parts of the world revealed high identities with each other (93% or more) (Shien *et al*., 2008; Wang *et al*., 2015). However, the complete genomic analysis of MDPV strains is still very limited. Genetic analysis of new strains of GPV could be gained via sequencing of viral protein (VP) genes, which are essential for pathogenicity, antigenicity, attachment, viral infectivity, and production of neutralizing antibodies (Tullis *et al*., 1993; Zadori *et al*., 1995; Chen *et al*., 2016b; Yu *et al*., 2016; Fan *et al*., 2017). For instance, the sequences of VP1 and VP3 showed that GPV strains that isolated from Pekin and mule ducks with SBDS belonged to the West-European lineage of GPV (Palya *et al*., 2009; Ning *et al*., 2017). Besides, the NS protein plays a role in viral DNA replication, expression, and regulation (Naeger *et al*., 1990; Christensen & Tattersall, 2002). Both right and left open reading frames (ORFs) are separated by 18 nt (Poonia *et al*., 2006). The receptor binding sites of structural protein play a key role in host range and pathogenicity of the virus (Shien *et al*., 2008; Kailasan *et al*., 2015; Ning *et al*., 2017). The genome of GPV and MDPV possess ORFs; the right ORF encodes for structural viral capsid (Cap) proteins (VP1, VP2, and VP3), while the left one encodes for non-structural gene 1 (NS1 and NS2) (Rep) proteins (Zadori *et al*., 1995). VP2 and VP3 contain similar C-terminal to VP1 (Zadori *et al*., 1995). It has been found that VP3 is the most variable protein and is important to produce neutralizing protective antibodies (Ju *et al*., 2011; Shao *et al*., 2015; Chen *et al*., 2021). Though GPV and MDPV have different host and antigenic features (Brown *et al*., 1995; Zhang *et al*., 2008), the comparison of both using southern hybridization revealed similar genomic nucleotide and amino acid sequences (Le Gall-Recule & Jestin, 1994; Zadori *et al*., 1994). This identity is due to the presence of at least one NS1 epitope and three VP1 epitopes that induced cross-reactivity immunization between both viruses (Li & Yu, 2013).

Some antigenic epitopes are shared between GPV and MDPV (Ju *et al*., 2011; Li and Yu, 2013). There is immunogenic cross-reactivity between GPV and MDPV as they share 76% to 80% nucleotide similarity at the VP1 level and 88% similarity at the amino acid level (Chu *et al*., 2001; Yu *et al*., 2012; Li & Yu, 2013). It has been reported that 7 antigenic regions of VP1 could react with serum from PV infected geese (Yu *et al*., 2012). There are epitopes of the non-structure protein and 3 epitopes on the structure protein between GPV and MDPV that may react to each other. Moreover, the

four epitopes might react with GPV and MDPV antisera after expression in *Escherichia coli* (Li & Yu, 2013).

The sequence analysis of GPV, MDPV, and SBDS virus genes showed that the VP1 of GPV and MDPV shares a nucleotide identity of 79.6-85.5% (Chu *et al*., 2001), but SBDS virus had 95.1-98.2% similarity with GPV and 88-92.6% similarity with MDPV, which may indicate an immunogenic cross-reactivity of SBDS virus with GPV than with MDPV (Bian *et al*., 2019; Chen *et al*., 2021). However, Palya *et al*. (2022) proved that the antigenic relationship between GPV and MDPV is inadequate to stimulate the cross-immunity between them.

In China, the fragment of DNA of the classical MDPV and is replaced by a homologous region from GPV, resulting in a recombination in the VP3 gene and formation of a new recombinant MDPV strain (Wang *et al*., 2017). These new strains caused outbreaks with high mortality rates among 19-days-old Muscovy duck flocks (Zhu *et al*., 2014). Other recombination processes in MDPV strains have also been detected in China (Wang *et al*., 2013; Wan *et al*., 2016b). Moreover, the sequence alignments of ITRs showed that the new MDPV strains shared higher similarities (96.0% to 97.2%) with the classical MDPV strains than with GPV (Wang *et al*., 2019). Emergence of new MDPV strains results in vaccination failure after using some living attenuated vaccines from the classical MDPV strains (Chen *et al*., 2002; Zhu *et al*., 2014).

Susceptibility

Host

The host range of PV is very limited. Its natural hosts are geese and Muscovy ducks. The Classical GPV principally infects geese and Muscovy ducks, while MDPV only infects Muscovy ducks and some crossbreed duck species. Geese are completely resistant to MDPV infection, while Muscovy ducks are susceptible to GPV and MDPV (Glávits *et al*., 2005). Mule ducks and Cherry Valley ducks are resistant to classical GPV infection. Besides, SBDS/BADS causes significant growth retardation in mule ducks and Cherry Valley ducks (Yu *et al*., 2016). All breeds of ducks including Muscovy ducks, Pekin ducks, and mule ducks, could be affected by PV. For the first time, the nucleotide sequence of GPV revealed a close similarity ranging from 95.6%-97.9% with European and Asian pathogenic GPV in 2–8-week-old ornamental ducks (mandarin, wood, falcated, and silver teal ducks) (Shehata *et al*., 2016). Muscovy ducks have played a critical role in the evolution of GPV to NGPV (Zhu *et al*., 2022).

The susceptibility of chickens and turkeys to PV infection has been documented. The virus was isolated from turkeys with enteritis, stunting, and high mortality (Trampel *et al*., 1983). Moreover, PV particles were detected in the droppings of chickens using electron microscopy and genome sequence (Kisary *et al*., 1985). Later, PV was isolated from chickens and turkeys with enteric infections in many countries of the world (Zsak *et al*., 2009; Bidin *et al*., 2011; Palade *et al*., 2011; Domanska-Blicharz *et al*., 2012; Murgia *et al*., 2012; Sharafeldin *et al*., 2017).

Age

Susceptibility of waterfowl to PV is highly age dependent. The virus affects goslings and ducklings less than 1-week-old with up to a 100% mortality rate, but the losses decrease to 10-60% in birds 1-3 weeks of age (Fang, 1962; Derzsy, 1967; Schettler, 1971; Hoekstra *et al*., 1973; Fang *et al*., 1981; Jestin *et al*., 1991; Glávits *et al*., 2005). Infection with MDPV “three-week disease”, mainly affects 3-week-old Muscovy ducklings. Derzsy’s disease outbreaks have been reported in 1-month-old goslings, with the mortality rate up to 90% (Fang *et al*., 1987).

PV infects rapidly the dividing cells, and this explains why clinical forms of infection occur approximately in birds up to 6 weeks of age. After this age, PV usually induces a late form of infection with rare signs. Nevertheless, the synergistic effect of PV with other immunosuppressive viral or bacterial infections tends to aggravate the clinical infection and prolongs the sensitivity period to the clinical signs up to 9 weeks of age (Palya, 2013; Ting *et al*., 2021). It is important to note that, following the susceptible age, PV infected birds show serological response without clinical manifestations.

Transmission

PV can be shed through the droppings of the infected waterfowl. Accordingly, the virus can rapidly be transmitted both directly and indirectly within the flock causing outbreaks (Yu *et al*., 2016). Both GPV and MDPV showed possibility of vertical transmission and eggshell contamination resulting in hatchery infection in disease-free birds (Irvine & Holmes, 2010; Palya, 2013; Fan *et al*., 2017). Besides, NGPV-infected breeder Pekin ducks and mule ducks showed vertical infection to their offspring (Chen *et al*., 2016a; Ning *et al*., 2017; Wan *et al*., 2019b).

Signs and Lesions

The severity of clinical signs of PV infection depends on the age and the immune status of infected goslings and ducklings (Palya *et al*., 2009). Susceptible age waterfowl between 1-3 weeks show an acute, subacute, or chronic form of the disease, with morbidity and mortality rates reaching 100% and 10-60%, respectively. An acute PV infection may develop in ages less than one week and the affected birds show off-food, polydipsia, prostration, and weakness, leading to up to 100% deaths within a few days. However, 4-week-old birds rarely show clinical manifestations, though a later form of infection may occur in older waterfowl (Gough, 2008). Waterfowl usually displays anorexia, growth retardation, stunting, loss of feathers on the back and neck, ocular and nasal discharge, palpebral swelling, watery diarrhea, drowsiness, ataxia, locomotor dysfunction, polydipsia, and drastic mortalities (Gough, 2008; Wozniakowski *et al*., 2012; Palya, 2013). It has been reported that NGPV may cause up to 60% dwarfism of ducks in China (Cao, 2019). The clinical signs of SBDS in mule and Tsaiya ducks were growth retardation, short, atrophied beak or bills, protruded swollen tongue, and short tibia bone with a low morbidity rate (Palya, 2013).

Experimental infections of susceptible 6 to 8-week-old Muscovy ducks with PV resulted in a chronic disease with significant growth retardation (Fournier & Gaudry, 1992; Yu *et al*., 2016). In China, Cherry Valley ducks showed feather shedding following a single NGPV infection or mixed infection with duck circovirus hepatitis (Yang *et al*., 2020).

The recorded mortality of GP is usually related to peak atrophy and the inability of birds to eat or drink (Yu *et al*., 2016). Death usually occurs within 2-5 days after the onset of the signs. The mortality rate differs according to the age and immune status of the bird (Palya *et al*., 2009). For instance, 3-4-week-old PV vertically transmitted birds exhibited a 70-100% mortality rate (Glávits *et al*., 2005; Jansson *et al*., 2007). During 1989 and 1990, SBDS was reported in ducklings with an 86-100% mortality rate (Lu *et al*., 1993). It has been shown that NGPV infection demonstrated an overall morbidity rate of 10-30% to classical PV infections (Chen *et al*., 2016b; Kapgate *et al*., 2018).

The characteristic post-mortem lesions of the classical and variant PV strains in waterfowl are usually thin wall sloughed intestinal mucosa with fibrinous exudates, fibrinous hepatitis, congested liver, degenerative skeletal muscle, myopathy, myocarditis, dilatation of the heart, hydropericardium, ascites, neuritis, polioencephalomyelitis, and atrophy of

lymphoid organs (thymus glands, bursa of Fabricius, and spleen) (Gough, 1991; Glávits *et al*., 2005; Palya, 2013; Ning *et al*., 2018).

Microscopically, experimental infection of a day-old gosling and 3-week-old Muscovy ducklings revealed severe enteritis with necrosis of enterocytes in the mucous membrane and the crypts of Lieberkühn, as well as formation of intranuclear inclusion bodies (Glávits *et al*., 2005). Other lesions, including hepatitis, myocarditis, degeneration of muscle fibers, lymphoid depletion of bursa of Fabricius, thymus, and spleen, mild sciatic neuritis, and polioencephalomyelitis were also reported (Glávits *et al*., 2005).

Laboratory diagnosis

Samples including liver, spleen, kidney, thymus glands, bursa of Fabricius, and brain can be selected for isolation of PV (Limn *et al*., 1996; Yang *et al*., 2009). The conventional laboratory diagnosis of PV is based on isolation in embryonated goose or duck eggs and requires 3- or 4-blind serial passages to cause death of embryos with hemorrhages (Saleh & Khodier, 2020). Moreover, 4 blind adaptation passages of PV cause a cytopathic effect on duck embryo fibroblasts which is characterized by rounding and shrinking of cells (Palya *et al*., 2009; Chen *et al*., 2015; Saleh & Khodier, 2020). Some strains of PV, such as H-1 replicate showed apoptosis in the infected cells (Poonia *et al*., 2006). The NGPV is distinguished from other types of PV by its ability to replicate autonomously *in vitro,* causing death of cells without helper-viruses (Brown *et al*., 1995; Zhang *et al*., 2019).

Molecular techniques such as polymerase chain reaction (PCR) (Sirivan *et al*., 1998; Wozniakowski *et al*., 2009b; Bulut *et al*., 2021; Isidan *et al*., 2021) and a loop-mediated isothermal amplification assay (LAMP) (Ji *et al*., 2010; Yang *et al*., 2010), as well as electron microscopy (Ning *et al*., 2018), immunofluorescent test (Cheng *et al*., 1997; Takehara *et al*., 1999; Zhu *et al*., 2012), and hemagglutination test (Chen *et al*., 2016b) have been used for detection of PV. Recent PCR techniques show many advantages over serological tests in the diagnosis of GPV (Yu *et al*., 2016; Ning *et al*., 2018). It is a highly sensitive and specific method for quantitatively detecting GPV DNA. The VP3 is a highly protected protein of the GPV gene and it is very appropriate for PCR (Yang *et al*., 2009). Real-time PCR is faster, less laborious, and more reliable than the classical PCR and LAMP technique. Moreover, it provides quantitative data for the pathogenesis and the epidemiological studies than other tests (Bi *et al*., 2008; Yang *et al*., 2009; Wan *et al*., 2011; Wozniakowski *et al*., 2012).

Chicken can be used as a model to produce humoral antibodies against PV (Ramadan *et al*., 2019). Serological tests, including neutralization test, western blot assay (Wang *et al*., 2005; Tarasiuk *et al*., 2019), agar gel precipitation test (Gough, 1984), indirect immunofluorescent assay (Chen *et al*., 2016b), indirect latex agglutination assay (Chen *et al*., 2016b), and enzyme-linked immunosorbent assay (ELISA) (Kardi & Szegletes, 1996) have been used to detect antibodies to PV infection in waterfowl. Indirect ELISA based on VP3 protein has been used for the detection of GPV and MDPV antibodies, as well as analysis of the immune status of vaccinated flocks (Zhang *et a*l., 2010). A competitive ELISA using a monoclonal antibody has been used to detect GPV-like particles and vaccine titers in goose sera (Wang *et al*., 2014b). A high sero-conversion to PV infection in 5- and 9-week-old Muscovy ducks has been detected when using MDPV antigen in ELISA. Despite the neutralization test is considered a sensitive assay for the detection of antibodies against GPV or MDPV in ducks or geese, it is very laborious, requires at least a week to complete, and the technique requires experienced technicians for the detection of the cytopathic effect. It is important to note that GPV can agglutinate cattle sperm cells,

and this feature can be used for virus identification (Brown *et al*., 1995; Saleh & Khodier, 2020).

Prevention and control

Biosecurity measures are a must due to the severe resistance of PV to physical and chemical inactivation, which creates difficulties in the virus elimination (Palya, 2013).

Vaccination of breeder flocks and their progenies against PV is required to reduce the impact of infection. Naturally infected or vaccinated breeder geese and Muscovy ducks can transfer maternal antibodies to progeny through the egg yolk. The acquired passive antibodies can persist in the offspring until 2 to 6 weeks of age, depending on the level of antibodies at day-old (Palya, 2013). Birds can acquire antibodies through receiving passive convalescent or hyper-immune serum or active immunization with vaccines (Kisary *et al*., 1978; Takehara *et al*., 1995, 1998; Maurin-Bernaud *et al*., 2013).

The levels of maternal-derived antibodies play a minor role in the induction of a primary immune response to PV, particularly when the vaccine is given very early at day-old. Goslings and ducklings with no or very low titers of maternal antibodies showed a fast and strong protection following administration of living attenuated PV vaccines (Kisary, 1977; Kisary *et al*., 1978). Hatchery vaccination may be beneficial for ducklings with a low-level passive immunity, as the birds remain protected even after the decline of the maternal-derived antibodies (Maurin-Bernaud *et al*., 2014). Maurin-Bernaud *et al*. (2014) compared the efficacy of ducklings' vaccination with a new live attenuated MDPV vaccine as a single shot at 17 days of age or double shots at 1 and 17 days of age. The results indicated that the protection rate against the viral challenge, the body weight, and the immune response were significantly higher in the groups vaccinated once and twice than control non-vaccinated ones. Besides, the highest weight gain was detected in birds vaccinated twice, which indicated that an early vaccination at hatchery, followed by a second vaccination at 17 days of age, is a practical protocol for the prevention of PV infection.

An active immune response against PV infection should be induced by early immunization using inactivated vaccines. Inactivated PV vaccines are available against MDPV (Kisary *et al*., 1978; Fournier & Gaudry, 1992; Takehara *et al*., 1995). Though these vaccines are less sensitive to maternal antibodies than living vaccines, they induce a slow immune response in vaccinated birds (Le Gall-Reule *et al*., 1999). Moreover, these types of vaccines may be either monovalent (GPV) or bivalent (GPV and MDPV) (Palya, 2011). A monovalent inactivated GPV vaccine has been used to control GPV infection in Muscovy ducks (Takehara *et al*., 1995). Recently, vaccination with a bivalent vaccine containing both GPV and MDPV antigens provided an adequate clinical protection against both viruses and a reasonable, rapid immune response even in the presence of maternal antibodies (Palya *et al*., 2022). It has been found that vaccination of day-old birds with an inactivated PV vaccine followed by a booster dose induced an immune response that lasted until the end of the age susceptibility (Palya *et al*., 2022).

It has been reported that recombinant PV antigens could be expressed and induce immunity in Muscovy ducks (Le Gall-Reule *et al*., 1999; Takehara *et al*., 1999). Immunization of ducks with VP2 and VP3 recombinant proteins induced neutralizing antibody titers which were consistent with the titers observed in ducks immunized with inactivated commercial vaccine (Le Gall-Reule *et al*., 1999). The immune response against the recombinant VP2 (rVP2) vaccine containing aluminum adjuvant and CpG oligodeoxynucleotides with three copies of the GACGTT motif was evaluated

in ducks (Lee *et al*., 2010). A significant increase in the rVP2-specific antibodies, expression of cytokines in peripheral blood mononuclear cells, and the percentage of CD4 +/ CD8 + cells were observed in vaccinated ducks when compared with the non-vaccinated control group (Lee *et al*., 2010). Moreover, Lee *et al*. (2016) observed similar results, along with an increase in the egg yolk maternal antibodies for the protection of the ducklings from PV infection. Wu *et al*. (2019) evaluated the effect of immunization of Pekin ducks with DNA PV vaccine on day 14 of age, followed by a booster dose with either the same DNA vaccine or rVP2 vaccine. The results declared that both immunization protocols increased the titers of antibodies, lymphocyte proliferation index, percentage of CD4+ and CD8+ cells in peripheral blood mononuclear cells, and mRNA expression of interferon -a and c, and interleukin-6 and 12. Moreover, using of DNA and rVP2 vaccine protocol induced a greater increase in lymphocyte proliferation, percentage of CD4+ and CD8+ cells, and upregulation of mRNA expression of cytokines when compared with using of primary and booster DNA vaccine.

Table 1: Parvovirus infection in different countries worldwide (1984-2024).

Country	Findings	Methods of detection	Reference
China	The new strain of SBDS (M15) was a GPV-related parvovirus causing mule duck and Cherry Valley duck SBDS disease.	SPF embryonated duck eggs, DEF, electron microscopy, latex agglutination assay, indirect immunofluorescence assay, ducklings, and PCR genomic sequencing	Chen *et al*. (2016b)
	A recombinant Lactobacillus plantarum strain was used as a mucosal vaccine vector to express GPV viral protein 2 (VP2) against infection in goslings.	PCR genomic sequencing, BALB/c mice, Western blotting analysis, and ELISA	Liu *et al*. (2017)
	The variant GPV from SBDS of Pekin ducks caused high mortalities and severe lesions similar to the classical GPV in SPF goose embryos. It induced Derzsy's disease in 2-day-old goslings with 80% mortality.	SPF embryonating goose eggs, goslings, agar gel precipitin test, PCR genomic sequencing, and histopathology	Ning *et al*. (2018)
	3 GPV strains were detected in 11 molt cases of 40-day-old Pekin ducks. They have 52 nucleotide variance relative to GPV associated SBDS.	PCR genomic sequencing	Jin *et al*. (2019)
	The 6 rMDPV strains shared 96.0%-97.2% identities with classical MDPV strains than with GPV and contained typical one-nucleotide-pair deletions in the palindromic stems of their ITRs.	PCR genomic sequencing and Muscovy ducklings	Wang *et al*. (2019)

Country	Findings	Methods of detection	Reference
China	CV21 strain displayed stronger cytopathic effect on cell culture and higher lethality to ducks embryos than MD17 strain. The mortality rates of CVD21 and MD17 infected Cherry Valley ducklings were 100% and 80%, respectively. MD17 showed higher homology to GPV, while CVD21 showed stronger similarity with NGPV. Both strains shared 95.8% homology. CVD21 severely affected duckling's growth.	DEF, embryonated Muscovy and Cherry Valley duck eggs, ducklings, electron microscopy, indirect immunofluorescence assay, and PCR genomic sequencing	Zhu *et al*. (2022)
Taiwan	15 parvovirus isolates in more than 500 ducklings had inappetence, ataxia, watery diarrhea, lameness, opisthotonos, and high mortality. Tolerant maturing ducklings displayed atrophic upper bills with a protruding tongue and stunting before maturity. Bill atrophy syndrome were reproduced in ducklings by inoculating the parvovirus isolates.	Embryonated duck eggs and histopathology	Lu *et al*. (1993)
Japan	GPV in Muscovy ducks had abnormal feathering, weakness of legs, and high mortality.	Embryonating Muscovy duck eggs, electron microscopy, agar gel precipitation test, and DEF	Takehara *et al*. (1994)
Vietnam	Out of 38 duck farms, 14 (36.84%) were positive. The Vietnamese novel GPV group was closely related to the Chinese group.	PCR genomic sequencing	Dong *et al*. (2022)
	94.35% mortalities in duck embryos. Specific signs (80% and 66.67%) following intramuscular and neck intravenous administration of ducks, respectively. The virus belonged to NGPV and was closely related to previous Vietnamese and Chinese strains.	Embryonated ducks eggs, inoculation in ducklings, and PCR genomic sequencing	Huong *et al*. (2024)
Poland	5% genetic variance (VP2 and VP3) of the Polish GPV strains with other strains. Besides, differences in amino acid sequence in Polish GPV and MDPV FM strains.	Goose embryo fibroblast cells and PCR genomic sequencing	Wozniakowski *et al*. (2009a)
	A significant correlation between age of the infected birds, signs, and DNA copy number of GPV and MDPV in 13 geese and 3 Muscovy ducks flocks.	Real-time PCR	Wozniakowski *et al*. (2012)

Country	Findings	Methods of detection	Reference
Poland	The incidence of TuPV and ChPV were 29.4% and 22.2%, respectively from 10 faecal swabs/flock (197, 1-19-week-old turkey and 45, 3-17-week-old chicken flocks). A clear division into ChPV-like, TuPV-like and unrecognized TuPV-LUB strains. The novel group showed 50.6-64.5% identity to the prototype chicken and turkey parvovirus.	PCR genomic sequencing	Domanska-Blicharz *et al*. (2012)
United States of America	Parvovirus was morphologically identified in the intestine of young turkeys with stunting, diarrhea, and increased mortality.	Histopathology and electron microscopy	Trampel *et al*. (1983)
	MDPV and its specific antibodies were detected in 1-4-week-old Muscovy ducks had inability to walk, high mortality rate, pale muscles and myocardium, fibrinous perihepatitis, and ascites. The virus showed 82.3% homologous with Derzsy's disease virus.	Embryonated Muscovy duck eggs, electron microscopy, PCR, immuno-electron microscopy, ELISA, immunofluorescence, and PCR genomic sequencing	Woolcock *et al*. (2000)
	Parvo virus was isolated from the spleen, liver, heart, skeletal muscle, and intestine of ducks showing locomotor dysfunction, 40-60% morbidity, and 10-40% mortality. The fragment of MDPV showed 84.5% sequence identity with similar and 84.6% identity with the GPV isolates.	Embryonated Muscovy duck eggs, MDEF, electron microscopy, and PCR genomic sequencing	Poonia *et al*. (2006)
	Out of 80 fecal samples, 41 were positive for parvovirus and 20 of 35 from non-light turkey syndrome (LTS) flocks were also positive. 98-100% identity between LTS and non-LTS samples except for one divergent TuPV strain.	PCR genomic sequencing	Sharafeldin *et al*. (2017)
Hungary	The amino acid sequence of fragment A (GPV-VP3 gene) was identical and represented Hungarian, West-European, and Asian strains. However, fragment B (upstream of the VP3 gene) showed a different clustering pattern among the Hungarian strains.	PCR genomic sequencing	Tatár-Kis *et al*. (2004)
	Parvovirus from SBDS of mule duck belonged to GPV-related group of waterfowl. Infections of 1-day-old mule ducks with a strain D176/02 from SBDS of mule ducks was reproduced.	Embryonated Muscovy duck eggs, conventional and PCR genomic sequencing, and ELISA	Palya *et al*. (2009)

Country	Findings	Methods of detection	Reference
Hungary	ChPV and TuPV in intestines of 15 and 2 chicken and 2 turkey flocks, respectively showed enteritis, lymphoid organs atrophy, and virus replication in the intestine, bursa of Fabricius, liver, and pancreas.	Histopathology, indirect immunohistochemistry, and PCR genomic sequencing	Palade *et al*. (2011)
France	A single-stranded DNA of about 5-300 bases with terminal palindromic hairpins (91, 78, and 58 kDa). The complementary DNA strands were encapsidated.	SDS-PAGE and partial analysis of DNA	Le Gall-Recule & Jestin (1994)
Sweden	GPV in a goose flock had increased mortality (more than 90%). Isolated virus was closely related to strains of Poland and the United Kingdom, but less homology with those of Hungary and Asia.	Embryonated eggs, serology, and PCR	Jansson *et al*. (2007)
Turkey	GPV in the liver, spleen, and intestine of 9 goslings showed dysphagia, bilateral ocular swelling and discharge, and diarrhea.	Conventional and real-time PCR	Bulut *et al*. (2021)
	GPV in geese flocks with decreased feed intake, increased water consumption, whitish diarrhea, feather disorder, retardation of growth, nasal-ocular discharge, death up to 50%, ascites, and swelling of the liver and spleen. The VP3 showed similarity (98.90%) with Polish and Taiwan strains.	Real-time PCR	Kardoğan *et al*. (2021)
Egypt	Increasing the antibody titers 4 weeks post vaccination with GPV/MDPV in Muscovy ducks and SPF chicks.	Muscovy ducklings, SPF chicks, SPF ECEs, and ELISA	Ramadan *et al*. (2019)
	GPV in mule ducks with retardation of growth, atrophy of the beak, diarrhea, and paralysis with mortality and morbidity rates of 2-8% and 20-50%, respectively.	Embryonated Muscovy duck eggs, DEF, ELISA, and bovine sperm agglutination test	Saleh & Khodier (2020)
	Mortality rate (20%), morbidity rate (70%), growth retardation, and decreasing performance in 3-4 weeks-old mule and Pekin duckling. A NGPV was identified.	Duck liver cells and PCR genomic sequencing	Soliman *et al*. (2020)

Country	Findings	Methods of detection	Reference
Egypt	6 out of 12 duck flocks were positive GPV. 5 isolates were NGPV and clustered with Chinese and Egyptian NGPV strains and 1 strain clustered with the Australian duck adeno-associated virus DA.	DE liver cells and PCR genomic sequencing	Eid *et al*. (2023)
	GPV was 100% identical to the strains isolated in 2023 in Egypt, but 98.1% identical with the isolated strain in 2018.	Embryonated duck eggs, DEF, ELISA, and PCR genomic sequencing	Abd Elaziz, *et al*. (2024)
	Out of 42 samples, 4 were positive MDPV (9.5%).	PCR	Hussein *et al*. (2024)

GPV= Goose parvo virus NGPV= Novel goose parvo virus
ChPV= chicken parvovirus
TuPV= Turkey parvovirus MD= Muscovy duck
rMDPV= Recombinant Muscovy duck parvovirus
CV= Cherry Valley MDEs= Muscovy duck embryos
SPF= Specific pathogen free DEF= Duck embryo fibroblast
MDEFs= Muscovy duck embryo fibroblasts ECEs= Embryonated chicken eggs
PCR= Polymerase Chain Reaction ELISA= Enzyme-Linked Immunosorbent assay
ITRs= Inverted terminal repeats SBDS= Short beak and dwarfism syndrome
SDS-PAGE= Sodium dodecyl sulfate-polyacrylamide gel electrophoresis

CONCLUSIONS

Infection with PV is regarded as an important threat for goose and ducks especially young ages. The virus can cause economic losses in the form of growth retardation and mortalities. Accordingly, more attention should be gained to focus on this infection regarding the disease diagnosis and prevention strategies. Trial studies to produce hyperimmune protective serum or novel recombinant PV vaccines for prevention of waterfowl should be given in consideration.

CONFLICT OF INTEREST

The author declared that there is no conflict of interest.

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عدوى فيروس بارفو: تهديد لصناعة الطيور المائية المنزلية

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تصيب عدوى فيروس البارفو الأوز والبط، مسببة مرضا حادا ومعديا وقاتلا يعرف باسم طاعون الأوز أو داء ديرزسي، وعدوى فيروس البارفو في البط المسكوفي. علاوة على ذلك، وصف فيروس بارفو جديد يصيب الأوز لأول مرة في بط البغل وبط وادي الكرز، ويسبب مرضا يعرف باسم متلازمة قصر المنقار والقزامة. اكتسبت عدوى فيروس البارفو في الطيور المائية اهتماما عالميا نظرا للخسائر الاقتصادية الفادحة في الإنتاج التجاري. ينتمي هذا الفيروس إلى جنس ديبيندوبارفوفيروس من فصيلة بارفوفيريدي. يصيب هذا الفيروس صغار الإوز والبط بشكل رئيسي خلال الأسابيع الثلاثة الأولى من العمر، مسببا معدلات إصابة ونفوق عالية، بالإضافة إلى اضطرابات حركية، وتأخر في النمو، وإسهال. ينتقل فيروس البارفو في قطعان الأوز والبط إما عن طريق التهجين الأفقي أو الرأسي. من أكثر أعراض فيروس البارفو شيوعا لدى الطيور المصابة: تنكس عضلة القلب، ونخر الأمعاء، والتهاب عضلة القلب، والتهاب الكبد، والاستسقاء، وأحيانا إصابة الجهاز العصبي. يعتمد تشخيص فيروس البارفو بشكل أساسي على عزل الفيروس وتحديده بالطرق التقليدية، وكشفه باستخدام التقنيات الجزيئية الحديثة، والانقلاب المصلي. تعتبر حماية صغار البط أو الإوز في مراحل حياتها المبكرة عبر المناعة الأمية السلبية الطريقة الأنسب للوقاية من عدوى فيروس البارفو. ويعد تطعيم قطعان التربية وصغارها ضروريا للحد من تأثير المرض. وقد استخدمت لقاحات حية، ومعطلة، ومعاد تركيبها ضد هذه العدوى. وفي الختام، ناقشت هذه المقالة عدوى فيروس البارفو في الطيور المائية من حيث خصائص الفيروس، وقابليته للتأثر، وانتقاله، وصورته السريرية، وتشخيصه المختبري، والوقاية منه ومكافحته.