REVIEW ARTICLE

Advances in research on genetic relationships of waterfowl parvoviruses

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Abstract

Derzsy's disease and Muscovy duck parvovirus disease have become common diseases in waterfowl culture in the world and their potential to cause harm has risen. The causative agents are goose parvovirus (GPV) and Muscovy duck parvovirus (MDPV), which can provoke similar clinical symptoms and high mortality and morbidity rates. In recent years, duck short beak and dwarfism syndrome has been prevalent in the Cherry Valley duck population in eastern China. It is characterised by the physical signs for which it is named. Although the mortality rate is low, it causes stunting and weight loss, which have caused serious economic losses to the waterfowl industry. The virus that causes this disease was named novel goose parvovirus (NGPV). This article summarises the latest research on the genetic relationships of the three parvoviruses, and reviews the aetiology, epidemiology, and necropsy characteristics in infected ducks, in order to facilitate further study.

Introduction

A parvovirus is a small, non-enveloped DNA virus that infects a variety of animals. The viruses have been named according to the isolated hosts rather than their genetic associations (51). Currently, the International Committee on Taxonomy of Viruses stipulates that the waterfowl parvovirus belongs to the *Parvoviridae* family, *Parvovirinae* subfamily and *Parvovirus* genus (11). Molecular phylogenetics have shown that waterfowl parvoviruses can be divided into two major categories according to the specific host species range, namely goose parvovirus (GPV) and Muscovy duck parvovirus (MDPV) (3, 7). In recent years, researchers have demonstrated that novel goose parvovirus (NGPV) is a derivative strain of goose parvovirus and also one of the waterfowl parvoviruses (1).

Goose parvovirus is an important virus that can infect goslings and Muscovy ducks. It can cause Derzsy's disease, which has a mortality rate exceeding 50% in epidemic years and is highly contagious (9, 14). The most typical clinical feature of the disease is the intestinal plug formed by the coagulation of fibrinous exudate and intestinal mucosal necrotic detachment. Depending on the course of the disease, it is classified into peracute, acute and subacute types (2). At present, the Derzsy's disease vaccines on the market can prevent Derzsy's disease. Researchers are developing a convenient, non-toxic and more effective goose parvovirus vaccine (28).

MDPV causes high morbidity and mortality in ducklings under 3 weeks of age (40, 58, 65). The main clinical symptoms of this disease include wheezing, motor dysfunction and diarrhoea (31). In general, the mortality rate of infected Muscovy ducks over 3 weeks of age is much lower than that of younger birds, but survivors usually show symptoms of skeletal muscle degeneration and growth retardation (55). In 2015, an unidentified infectious disease broke out for the first time in a flock of Cherry Valley ducks in eastern China. It was manifested in a short beak and growth retardation has come to be known as beak atrophy and dwarfism syndrome (BADS). This infectious disease can cause 20% to 50% morbidity. Although the mortality rate of short beak and dwarfism syndrome (SBDS), (a synonym of BADS) is very low, the severe weight loss caused by

stunting has caused serious economic losses to the Chinese waterfowl industry (21). The pathogen was found to be a variant strain of GPV–NGPV, also referred to in some research as short beak and dwarfism syndrome virus (SBDSV).

The group of waterfowl parvoviruses combines NGPV, GPV and MDPV. They share similarities in morphology, physicochemical properties, culture characteristics and genomic structure, but their pathogenicity and antigenicity are quite different. There is a genetic relationship between GPV, MDPV and NGPV, and the biological characteristics of and detection methods for the three have attracted more researchers' attention. In this review, the pathogenic characteristics, clinical symptoms, and genetic relationship of the three parvoviruses and necropsy characteristics of infected ducks are expounded, and the findings on the genetic relationship of the three viruses will provide reference material for further research.

Characteristics of the viral genome

Parvoviruses are non-enveloped single-stranded DNA viruses, which include 23 genera and 107 species, usually characterised by a single-stranded DNA genome, and can be further classified into the *Parvovirinae* and *Densovirinae*. Parvoviruses are widely transmissible and can spread from invertebrates to mammals, and also have an evolutionary ability distinct from those of other DNA viruses (3, 29). NGPV, GPV and MDPV are members of the *Dependoparvovirus* genus, and are pantropic single-stranded DNA viruses (36). Waterfowl parvoviruses have a linear genome that is approximately 5.1 kb in length and a small icosahedral capsid assembled from 32 capsids with a diameter of 20–22 nm (7, 8, 41). Parvoviruses utilise the self-priming system at the end of the genome to interfere with the hairpin transfer mechanism during the replication of autonomous viral DNA. The full-length genome of these viruses contains two open reading frames: the left encodes the non-structural (NS) proteins NS1 and NS2, and the right encodes the structural proteins VP1, VP2 and VP3 (62). The length of the NS

nucleoside of NGPV, GPV and MDPV was 1881nt by sequence analysis, and they all encoded 626 amino acids. Comparison of NS genes showed that the similarity between NGPV and GPV was greater than that of MDPV, and that there are 12 synchronous specific mutation sites (1).

In the structural proteins VP1, VP2 and VP3, the peptide chain of VP1 is longer than that of VP2, and in turn its chain is longer than that of VP3 (34). The entire amino acid sequence of VP2 and VP3 is contained in the carboxyl terminal portion of VP1 (43). The VP1, VP2 and VP3 genes are located in the "basket structure", which means that they have the same gene sequence and the same termination codon in the 3′ end region, but their initiator codons are located at different positions (38, 42, 44). The 58-kDa molecular weight VP3 protein is the main capsid protein of the virus, main protective antigen and contains the main epitope of the virus. This protein can induce the body to produce neutralising antibodies and provide protective cross-immunity to other waterfowl parvoviruses; results of investigations showed that the VP3 protein is the main candidate antigen for vaccine development and serodiagnostic tests (22, 56, 63). Of these three capsid proteins, accounting for about 80% of the total capsid protein amount, VP3 is the largest element and can serve as a scaffold protein (40). The VP2 protein is highly immunogenic, easily inducing antibodies in ducks and geese, and is abundant during viral infection (59, 20). This protein has different functions throughout the life cycle of the virus and can also take part in shell assembly and DNA packaging (18) (Fig. 1).

Pathological changes and clinical symptoms

The main characteristics of Derzsy's disease caused by GPV is high mortality, and the main clinical symptoms include somnolence, ataxia, dysphagia, weight loss, bilateral periorbital, eyelid swelling and frequent yellowish-white diarrhoea. Goslings infected with GPV convulse before death, and mortality rates in the first four weeks of life reach 70% to 100% (15, 25, 32, 57).
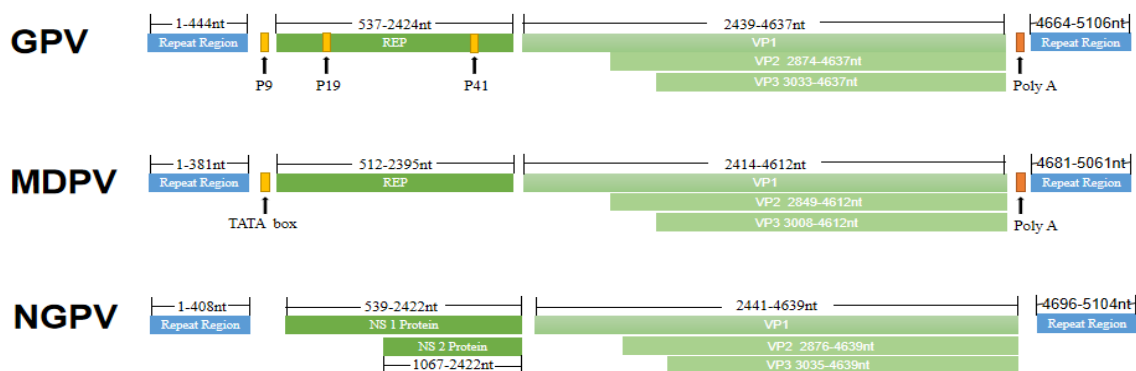

Fig. 1. Genetic map of three waterfowl parvoviruses

MDPV only affects ducklings (2–4 weeks old) and the mortality from its associated disease is up to 80% depending on the age of the infected Muscovy duck. The characteristic symptoms of MDPV infection are watery diarrhoea, wheezing and locomotor dysfunction (13, 16, 19, 49).

The SBDS caused by NGPV occurs primarily in ducks which are 10 to 30 days old. Some ducklings were unable to walk at 5 to 6 days of age and some were found to be growing slowly at 9 to 10 days of age. The shortness and poor growth of the ducks were more evident after 3 weeks of age. Ducks infected with NGPV also showed weakness in the feet, suffered fractures of the tibia and exhibited truncation of the beak. The disease does not cause high mortality but in its clinical incidence of up to 60% (12) it is grave in its impact. The final beak size of a duckling suffering from SBDS is about 10–30% of the normal beak size of healthy ducklings. Beak dysplasia causes the infected ducks' tongues to protrude, and this significantly affects their eating ability. The body weight of infected ducks when brought to market is 20–30% lower than that of healthy ducks, and the most serious infected ducks are 50% lighter. Furthermore, some of the ducks suffer from walking difficulty with a unilateral dysfunction, paralysis and other symptoms (35, 39, 57).

Anatomopathology

Most of the corpses of geese infected with GPV are visibly thin, the eye sockets are depressed, the mouth has a lot of secretions, the oral mucosa is brown, and the whole body has subcutaneous haemorrhages, there is pleural effusion, fatty degeneration in the liver and gallbladder, kidney swelling, and congestion in the pancreas and spleen. The mucosa in the middle part of the small intestine of a typical goose is necrotic and shows shedding, while the lower part of the small intestine forms a sausage-like embolus, and the intestinal lumen is blocked. Haematoxylin and eosin staining of paraffin-fixed tissue sections showed hepatic congestion and inflammatory cell infiltration, villous necrosis, shedding, duodenal, ileal and jejunal mucosa with the presence of inflammatory cells and cellulose-like exudate, dilation of capillaries in the brain and turbidity of renal tubular epithelial cells (52). Liu *et al.* (26) found that in healthy Cherry Valley ducks infected with GPV, at the time of autopsy most of the ducklings in the infected group had swollen spleens, livers and intestines, blood spots in the thymus and spleen, and moderate to severe bleeding in the thymus and spleen visible under a microscope. Congestion was also observed in the Harderian gland. The results indicate that GPV is pathogenic to Cherry Valley ducks, but less virulent in them than in goslings.

It can be observed in MDPV duck necropsies that most of the cloaca is surrounded by thin faeces, the heart has assumed a round shape and its wall is slack, the liver is swollen and brittle, the spleen is swollen. There are white necrotic spots in the pancreas, intestinal catarrhal inflammation, haemorrhaging, congestion and exfoliated intestinal mucosa in the intestinal contents. Decreased the body skeletal muscle mass and pallor of the leg skeletal muscles were observed in some MDPV duck necropsies (13, 16, 37).

In ducks infected with novel GPV, the lesions are quite different from those in ducks infected with the other two viruses. In addition to the short beak and long tongue, some ducks infected with NGPV have scattered bleeding spots or diffuse bleeding on the surface of the pancreas, as well as swelling in the liver, spleen, kidney, lung, small intestine, and pancreas. Thymic medullary lymphocytes and reticular cells show scattered necrosis, inflammatory cell infiltration, and tissue interstitial bleeding, the renal tubules also have interstitial haemorrhaging, and heavy inflammatory cell infiltration, and tubular epithelial cells present with disintegration and apoptosis. Hepatocytes diffusely infiltrate macrophages in the hepatic lobules and show other histopathological changes, such as hepatocyte swelling and steatosis, as well as various sizes of fat droplets in the cell mass. Moderate tongue lesions are characterised by interstitial inflammation, connective tissue interstitial loosening and oedema (5).

Progress in research on the genetic relationship between waterfowl parvoviruses

Genetic relationship between goose parvovirus and Muscovy duck parvovirus. In recent years, with the development of enabling technology, scientists have given more attention to GPV and MDPV, and presently they continue to conduct in-depth research on the relationship between the two. In previous studies, VP gene sequencing of three GPV strains (DY, PT and D) and two MDPV strains (GX5 and SAAH-SHNH) was performed by phylogenetic and recombinant analysis, and recombination events in the GPV strains and the MDPV strains were found, which led to the recombinant GPV strains and the two MDPV strains had different breakpoints and when the recombination event was confirmed using the SimPlot program, it was found to show the same recombination position in VP1 (40). A major breakthrough in the identification and detection of GPV and MDPV viruses came in the comparison of their genomes, which indicated that the two share a nucleotide similarity of 79.6–85.0% at the genome level (60, 61). Researchers have found that due to the immune cross-reaction of GPV and MDPV in Muscovy ducks, it is difficult to simultaneously detect infection with either virus and differentiate between them. However, a one-tube double PCR assay method which incorporates three pairs of highly specific primers designed for this purpose can be used to simultaneously detect and differentiate GPV and MDPV viruses, which suggests that waterfowl can be vaccinated with GPV and MDPV vaccines (47).

Genetic relationship between goose parvovirus and novel goose parvovirus. The effect of NGPV on Cherry Valley ducklings was studied from the perspective of gut microbiota, and it was found that it caused an ecological imbalance of intestinal microflora and was accompanied by short-chain fatty acid content in the cecum. The result of the research provided a new direction for further study of NGPV (11). A qPCR based on TaqMan probe was developed and validated for applicability to NGPV quantification. Compared with a conventional PCR, a qPCR is more sensitive and the reaction under the relevant protocol can quantitatively detect NGPV DNA in different duck species in different geographical regions and enables the epidemiological investigation of NGPV (33).

According to the sequence analysis of the waterfowl parvovirus genes, there are two important amino acid changes (Asn-489 and Asn-650) in the VP protein of NGPV compared with GPV, which may be the only reason why the host of GPV changed from the goose to the duck. By immunological epitope prediction, the aa515–aa528 region in NGPV is considered to be the binding site of the VP3 domain, which will trigger the production of neutralising antibodies. The discovery of this immunological epitope makes an important contribution to the development of a vaccine against SBDS (30). After continuous passages in duck embryos, SBDSV M15 was isolated from the allantoic fluid of dead embryos, and its nucleotide and amino acid homology with Hungarian GPV strains and Chinese GPV strains was 96.0–97.1% and 97.4–98.3%, respectively. Seven SBDS duck samples were found to be positive for GPV antigen by a latex agglutination assay, while being negative for MDPV. As a consequence, it was found that SBDSV M15 is a strain of a novel parvovirus associated with GPV that causes SBDS (6). In order to confirm the pathogenicity of SBDSV M15 in other domestic waterfowl species, three day old Muscovy ducks, sheldrake ducklings and goslings without GPV antibodies were infected with a high dose of SBDSV M15. It is seen in Table 1 that Muscovy ducks infected with SBDSV M15 lost 41%, 43%, and 57.5% of body weight at 14, 21, and 28 days post infection (dpi), respectively compared with the control group. The weight of the sheldrake ducklings infected with SBDSV M15 lost 5.9%, 23.6%, 27.9% at these three intervals. The rates of weight loss in gosling infection group were 19.0%, 32.0%, 36% at 14, 21, and 28 dpi, respectively (Table 1). In summary, it was found that SBDSV M15 is pathogenic to Muscovy ducks, sheldrake ducklings and goslings. The infected birds were found to exhibit significant growth retardation, anorexia and diarrhoea; however, no atrophied tendons or protruding tongues were observed in any inoculated birds. These results indicated that the emerging duck-derived goose parvovirus in China has extensive pathogenicity to major domestic waterfowl, and its symptoms are diverse. With deleterious effects such as

were noted in the research, SBDS may inflict huge economic losses on the Chinese waterfowl industry (53).

Detection of GPV and NGPV by recombinase polymerase amplification (RPA) combined with vertical flow visualisation bands is more efficient than the loop-mediated isothermal amplification technique and provides an important diagnostic tool for detecting GPV and NGPV infections because it has high sensitivity and specificity and the RPA reagent is provided as lyophilised granules for easier storage and transport (27). Li *et al.* (24) used two half-sleeve PCR assays which were generated in a single reaction using a pair of universal primers and two specific typing primers. These assays were for simultaneous diagnosis of classical GPV and NGPV, which can be achieved when both viruses are detected, and they have demonstrated high specificity and sensitivity. From January to July 2016, some Muscovy ducks infected with SBDS were collected from East China. Double chain PCR detected NGPV in these Muscovy ducks but could not detect GPV, which further proved that NGPV was the cause of short beak and dwarfism syndrome in Cherry Valley ducks rather than GPV.

Genetic relationship between three waterfowl parvoviruses. During their study of the genetic relationship between NGPV, GPV and MDPV, Yang *et al.* (54) found 93.4–98.9% identity between NGPV and GPV isolates estimated by phylogenetic tree analysis of the VP3 gene, but only 80.4–88.7% identity with MDPV, indicating that NGPV is a new variant of GPV. Sequence alignment of NGPV, GPV and MDPV revealed that the VP gene of the NGPV isolates had 90.9–97.5% identity with classical GPV and 80.9–91.5% identity with MDPV, and at the VP amino acid level, the NGPV isolates had 95.1–98.2% identity with classical GPV and 88.0–92.6% identity with MDPV, which provides an important reference for the development of an NGPV vaccine (1). Comparison of 52 NS gene sequences (including 15 MDPV and 37 GPV) retrieved from GenBank showed that the samples in the MDPV cluster had higher nucleotide identity than those in the GPV cluster. Compared with the NS gene of GPV cluster and the NS gene of the MDPV cluster, the homology ranged from 80.8% to 83.4%. These data suggest that if primers are designed for a specific region of the NS gene, erroneous results may be obtained (45) . However, these NS genetic comparison data can identify specific regions of MDPV-specific genes, and they have been used to establish TaqMan-based real-time PCR analysis methods to detect MDPV more accurately.

Table 1. Inhibited the body weights growth trend of three waterfowl species after infection with SBDSV M15

Infected waterfowl	Inhibited the percentage of weight gain compared with the control group		
	14 dpi	21 dpi	28 dpi
Muscovy duck	41.0%	43.0%	57.5%
Sheldrake ducklings	5.9%	23.6%	27.9%
Goslings	19.0%	32.0%	36.0%

Table 2. Description of waterfowl parvovirus isolates compared in this study

Isolates	GenBank accession no.	Host	Origin	Collection Date
GPV-06-0329 (41)	EU583391.1	Goose	Taiwan, China	2006
GPV-VG32/1 (42)	EU583392.1	Goose	Germany	2004
GPV-MDE	MF438102.1	Mule duck	Fujian, China	2015
GPV-RC16	KY475562.1	Goose	Chongqing, China	2016
GPV-E (61)	KC184133.1	Goose	Anhui, China	2012
GPV-B (48)	U25749.1	*Anser anser*	Hungary	1995
GPV-G7 (47)	KR029617.1	Muscovy duck	Fujian, China	2013
GPV-HuN18 (61)	MK736656.1	Linwu sheldrake	Fujian, China	2018
GPV-GER (39)	KU684472.1	Ornamental duck	Poland	2015
GPV-GD	MH444514.1	Mule duck	China	2016
NGPV-DS15 (21)	KX384726.2	Cherry Valley duck	Anhui, China	2015
NGPV-SDLY1602 (23)	MF441222.1	Cherry Valley duck	Shangdong, China	2016
NGPV-sdlc01 (4)	KT343253.1	Cherry Valley duck	Fujian, China	2015
NGPV-SDHZ1604 (4)	MF441223.1	Cherry Valley duck	Shangdong, China	2016
NGPV-SDLY1512 (4)	MF441221.1	Cherry Valley duck	Shangdong, China	2015
NGPV- JS1603 (23)	MF441226.1	Cherry Valley duck	Jiangsu,China	2016
NGPV-SC16	KY679174.1	Cherry Valley duck	Sichuan, China	2016
NGPV-QH15	KT751090.1	Peking duck	China	2015
NGPV-AH1606 (23)	MF441225.1	Cherry Valley duck	Anhui, China	2016
NGPV-SDDY1605 (23)	MF441224.1	Cherry Valley duck	Shangdong, China	2016
MDPV-FJV1	KR029616.1	Muscovy duck	China	2011
MDPV-GX5 (56)	KM093740.1	Muscovy duck	Guangxi, China	2011
MDPV-ZW (16)	KY744743.1	Muscovy duck	China	2006
MDPV-JH06 (50)	MH807697.1	Muscovy duck	Zhejiang, China	2006
MDPV-FJM2	KR075688.1	Muscovy duck	China	2013
MDPV-SAAS-SHNH	KC171936.1	Muscovy duck	Shanghai, China	2012
MDPV-M8	KR029614.1	Muscovy duck	Fujian, China	2013
MDPV-GDNX (10)	MH204100.1	Muscovy duck	Guangdong, China	2016
MDPV-JH10 (50)	MH807698.1	Muscovy duck	Zhejiang, China	2010
MDPV-YL08	MG932366.1	Muscovy duck	China	2008
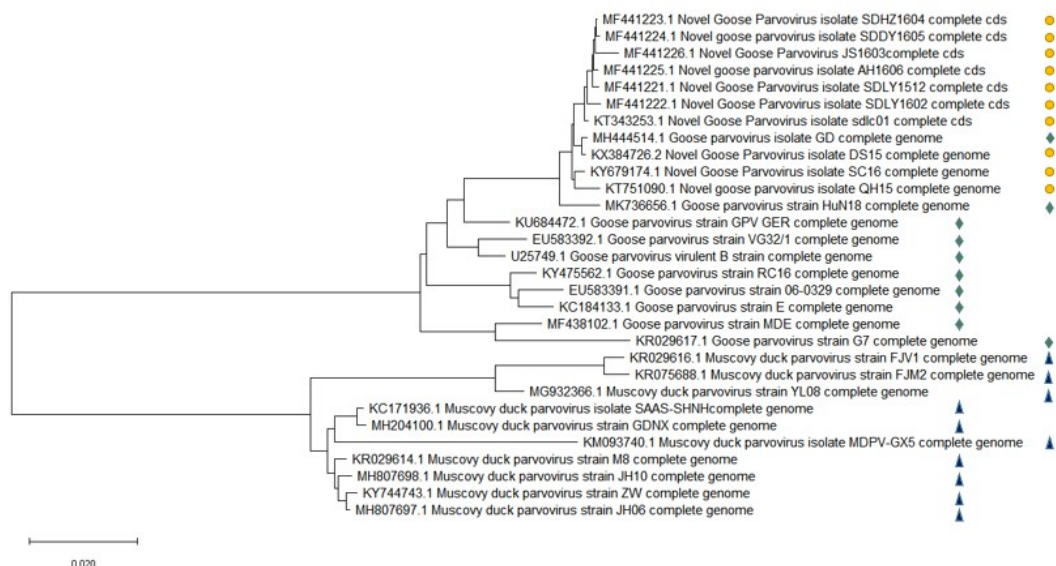
Fig. 2. Phylogenetic analysis based on the full-length genomic sequence of three waterfowl parvoviruses
🟠 novel goose parvovirus; ◆ goose parvovirus; ▲ Muscovy parvovirus

After genetic comparison, the characteristic variable region of the GPV NS gene is obviously different from that of MDPV. The TaqMan real-time PCR method developed can effectively distinguish GPV and MDPV, and a specific detection method for GPV and MDPV is established thereby. The results of real-time PCR showed that 21 of the 37 GPV and NGPV strains share the sequence of AGAGAAGCA GGAACAATTACCAGGT and these 21 sequences belong to the GPV group. Twelve of the 37 sequences share the codon for AGAGAAGCAGGAACAATT ACCAGGT and 12 sequences belong to the NGPV group. Two probes were designed and both can be used to quantify GPV and NGPV and to distinguish between these two viruses (46).

In this review, we selected full-length sequences of 30 strains of GPV, MDPV and NGPV. The results of the phylogenetic analysis are shown in Fig. 2. It was found that NGPV has a high degree of genetic identity with GPV. The genetic identity between the 10 strains of MDPV was higher at approximately 97%, but their genetic identity with GPV and NGPV was lower. The genetic identity of GPV-VG3/1 from Germany with the GPV-B strain from Hungary reached 98%, indicating that the German strain may have been transported into the country *via* waterfowl from Hungary. The GPV-GER strain from Poland and GPV strains from China displayed 92% genetic identity at the nucleotide level, and it can be inferred that Chinese GPV may be transmitted from Europe through waterfowl transport. The GPV-HuN18 strain from Fujian in China and the new goose strain have 98% genetic similarity at the nucleotide level, indicating that the NGPV strain may derive from GPV-HuN18. It is particularly worth mentioning that the NGPV-DS15 strain has very high genetic identity with the GPV-GD strain, indicating that it may be descended from the GPV-GD strain or that

GPV-GD is an unproven NGPV strain which still requires further research (Fig. 2).

Summary and outlook

GPV, MDPV and NGPV are three waterfowl infectious diseases that cause Derzsy's disease, Muscovy duck parvovirus disease and duck short beak and dwarfism syndrome, respectively. The pathogens pose a serious threat to the development of waterfowl farming worldwide. The clinical symptoms of the infectious diseases caused by GPV and Muscovy parvovirus are similar, including loss of appetite and difficulty in breathing, and with both there are high mortality and high morbidity. In recent years, the duck short beak and dwarfism syndrome caused by a new variant of GPV (NGPV) has been incident at 60%, and although the mortality rate has not been very high, the weight gain by stunting has caused serious economic losses to the Chinese aquatic poultry industry.

The genomes and amino acids of these three viruses are highly homologous, so the genetic relationship between the three parvoviruses has attracted great interest from researchers. In recent years, many research institutes and universities have made major breakthroughs in the identification of the three parvoviruses and the differences from other parvoviruses, but the pathogenic mechanism and evolution process of GPV, MDPV, and NGPV and the full functions of structural and non-structural proteins at present are still unclear and further work should be carried out. Since the *Parvoviridae* have been mutating and evolving and the pathogenesis of the three parvoviruses has not yet been studied, there are still many challenges in studying how the pathogen mutated from GPV to NGPV. This article summarises the genetic

relationship between the three parvoviruses and lays a theoretical foundation for studying their pathogenic mechanism and variation conditions.

Conflict of Interests Statement: The authors declare that there is no conflict of interests regarding the publication of this article.

Animal Rights Statement: Not applicable.

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