OPEN ACCESS
DOI: 10.5455/javar.2015.b90

J. Adv. Vet. Anim. Res., 2(3): 296-303.
Available at- *http://bdvets.org/JAVAR*

JAVAR Journal of Advanced Veterinary and Animal Research

ORIGIAL ATICLE

Volume 2 Issue 3 (September 2015)

Molecular characterization of Duck Plague virus isolated from Bangladesh

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ABSTRACT

Duck plague (DP) is the most feared duck disease in the world. For isolation, identification, molecular detection and characterization of DP virus (DPV), a total of 94 samples were collected from commercial farms (n=6) and households (n=13) from Rajshahi (n=37), Netrokona (n=35) and Mymensingh (n=22) districts of Bangladesh. The samples were processed and inoculated into 11-13 days old embryonated duck eggs for virus propagation. Virus was identified using agar gel immunodiffusion test (AGIT) and passive hemagglutination (PHA) test, and was confirmed by polymerase chain reaction (PCR) targeting *DNA polymerase* and *gC* genes, followed by sequencing. Pathogenicity tests were performed using duck embryos, ducklings and ducks. Among the 94 samples, 17 isolates were confirmed as DPV by PCR amplification of partial *DNA polymerase* (446-bp) and *gC* genes (78-bp), respectively. One of the isolates (Anatid herpes 1 BAU DMH) was sequenced and found to be closely related with a Chinese variant of DPV (GenBank: JQ647509.1). Thus, we assume that both Bangladeshi and Chinese isolates of DPV may have a common ancestor.

Keywords

*g*C gene, Isolation, Identification, PCR, PHA test, *DNA polymerase* gene

ARTICLE HISTORY

Received : 10 May 2015, **Revised**: 3 June 2015,
Accepted : 6 June 2015, **Published online**: 6 June 2015.

INTRODUCTION

Duck comprises about 16% (42.68 million) of the total poultry population (270.71 million), occupying the second position next to chicken for production of table eggs in Bangladesh (Bangladesh Economic Review, 2010). Considering the infectious diseases, ducks are relatively resistant as compared to chickens; thus, popularity of duck production is increasing in Bangladesh. However, duck diseases devastate almost every year in Bangladesh (Sarker et al., 1980; Hoque et al., 2011). Among the duck diseases, duck plague (DP) is the most feared disease (Hanan et al. 2014), which is caused by Duck Viral Enteritis Virus (DVEV) belonging to *Herpesviridae* family, *Alphaherpesvirinae* subfamily, *Mardivirus* genus as Anatid Herpesvirus 1 denoted after the host family *Anatidae* (ICTV, 2014).

DP is an acute, infectious and usually fatal disease of domestic ducks and wild waterfowl (Kaleta et al., 2007). The disease was first reported from Netherlands in 1923 and later from other countries (Wang et al., 2013). Morbidity and mortality due to DP varies from 5-100% (Calnek et al., 1997). Susceptible birds are usually infected through close contact to diseased birds under natural conditions (Kaleta et al., 2007). Indirect contact through environment can also result in infection. Migratory waterfowl and domestic waterfowl may spread the infection from one another (Kathryn et al., 2001; Wang et al., 2013). During outbreaks, infected birds act as a source of infection for uninfected birds, which will act as the new source of virus transmission contributing to faster and wider distribution of the disease (Burgess et al., 1999; Converse and Kidd, 2001; Campagnolo et al., 2001; Wang et al., 2013).

In Bangladesh, DP virus (DPV) was first reported and confirmed by Sarker et al. (1980, 1982). Then, Islam et al. (1989), Khan et al. (1990) and Akter et al. (2004) studied on different aspects of DPV from Bangladesh. Later, Hossain et al. (2005) and Islam et al. (2005) evaluated immunogenicity of DPV vaccine from local isolates.

DPV can be identified by virus neutralization (VN) test (Wu et al., 2011; OIE, 2012), passive hemagglutination (PHA) test (Hossain et al., 2005; Islam et al., 2005; Das et al., 2009), by inoculation into 11-13 days old duck embryo through chorioallantoic membrane (CAM) route (Akter et al., 2004; OIE, 2012; Hanaa et al., 2013), by propagation in duck embryo fibroblast cell culture (Gao et al., 2014), by inoculation into day-old ducklings or into ducks, and finally by molecular detection using polymerase chain reaction (PCR) (Li et al., 2009). Accurate detection and isolation of the virus are crucial for successful controlling the disease. However, there is paucity of literature describing PCR-based identification of DPV in Bangladesh. To the best of our knowledge, this is the first report in Bangladesh describing molecular detection and characterization of DPV by PCR targeting *DNA polymerase* (Hansen et al., 1999; OIE, 2012), and *gC* genes (Lian et al., 2010). The present study was conducted for isolation, identification, and molecular characterization of DPV from suspected ducks of different districts in Bangladesh.

MATERIALS AND METHODS

Study area and period: The study was conducted with the samples collected from three districts of Bangladesh namely Netrokona, Rajshahi and Mymensingh duging the period of July 2013 through September 2014. Out of these three districts, Netrokona and Mymensingh districts have vast areas of low lands and water bodies, where duck population is significantly high.

Samples and methods of sampling: A total of 94 samples comprising of 77 cloacal swabs from suspected ducks, and 17 visceral organs (e.g., esophagus, liver, intestine, and proventriculs) from dead ducks were collected (**Table 1**) from commercial farms (n=6) and households (n=13) located in the three districts mentioned above, as per the procedure described by Hanaa et al. (2013). The samples were collected aseptically and were placed separately in sterile falcon tubes with proper labeling, and then transported to the Virology Laboratory at the Department of Microbiology and Hygiene, Faculty of Veterinary Science, Bangladesh Agricultural University (BAU), Mymensingh, Bangladesh by maintaining proper cool chain. The field samples were either processed immediately or stored at -20°C until used. The sample collection from the suspected live birds or experimentation with laboratory animals were performed as per the ethical guidelines set by the Laboratory Animal Unit of the Department of Microbiology and Hygiene, BAU. The samples were collected from the commercial farms and households after taking necessary permission from the owners.

Preparation of inocula: The samples were grinded, and 10% suspension was prepared using phosphate buffer solution (PBS). The suspension was centrifuged at 4500 rpm for 10 min (OIE, 2012). Inocula were prepared from each sample according to the method mentioned in OIE (2012). Then, the supernatant was collected in a sterile falcon tube and treated with Gentamycin dosed at 100 µg/mL.

Sterility test and propagation of virus: Antibiotic treated inocula were tested for sterility in fresh blood agar media at 37°C for 24 h (Rana et al., 2010). Sterile inocula were then injected through CAM route in 11-13 days old embryonated duck eggs (EDE) (Akter et al., 2004; OIE, 2012). After 6-8 days of post-infection (PI), all live EDEs were chilled overnight and allantoic fluid (AF) and CAM were collected. the EDE died earlier was also chilled, and in similar way, the AF and CAMs were collected.

Preparation of hyperimmune sera against DPV: Attenuated Duck Plague Vaccine (FnF®) was inoculated in rabbit with increasing doses for 7 consecutive days (0.1-1 mL, intra-peritoneally). After 14 days of last injection, blood was collected aseptically, and serum was separated, and heat inactivated at 56°C for 30 min. Finally, the serum was stored at -20°C for agar gel immunodiffusion test (AGIT) and passive hemagglutination test (PHA) test, as described by Morrissy et al. (2008).

Agar gel immunodiffusion test (AGIT): The DPV isolates were confirmed through AGIT (Wu et al., 2011; Shen et al., 2011). In brief, 100 mL Agar Noble (1.25%) was prepared with agar (1.25 gm), NaCl (8 gm) and phenol (0.5 gm); the ingredients were dissolved completely by heating (OIE, 2008). The solution was poured in petri dish with a thickness of 3-5 mm and allowed to set. On solidification, circular wells were cut 6 mm in diameter and 3 mm apart using a template and tubular cutter. The bottom of each well was sealed with melted noble agar. The central well was loaded with hyperimmune serum and peripheral wells were loaded with suspected AFs containing DPV along with known positive and negative controls. The plates were incubated at 37°C in humidified chamber for 48 h for the appearance of bands.

Detection of duck plague virus by PHA test: The suspension prepared from the CAMs of positive samples were used as the source of virus, which was used for PHA test. All the positive CAM suspensions

Table 1: Oligonucleotide sequences used for polymerase chain reaction.

Name of gene	Primers	5′-Sequence-3′	Amplicon size	Reference
DNA polymerase	F	5´-GAAGGCGGGTATGTAATGTA-3´	446-bp	Wu et al. (2011)
	R	5´-CAAGGCTCTATTCGGTAATG-3´		
gC	F (P3)	5´-GAAGGACGGAATGGTGGAAG-3´	78-bp	Zou et al. (2010)
	R (P4)	5´-AGCGGGTAACGAGATCTAATATTGA-3´		

were found to hemagglutinate the tanned sheep RBC (sRBC) (Akter et al., 2004; OIE, 2012) indicating the samples as positive for DPV. The PHA test was done according to the method described by Das et al. (2009).

Pathogenicity tests of DPV: Pathogenicity test was done according to the method described by Hanaa et al. (2013). At first, the $dELD_{50}$ was determined and then ducklings were inoculated with 0.5 mL ($10^{5.6}$ $dELD_{50}$/mL), and adult ducks were inoculated with 1 mL ($10^{5.6}$ $dELD_{50}$/mL) of the infectious AF.

$dELD_{50}$: $dELD_{50}$ of isolated DPV was determined following the method of Tripathy et al. (1970). A 10-fold serial dilution of 0.5 mL CAM suspension was made (10^{-1} to 10^{-12}) and 5 EDE were inoculated with 200 µL of the diluted virus; one group of 5 EDE were inoculated with each virus dilution. The EDE were incubated at 37°C and observed twice daily for 8 days. Death patterns were recorded (**Table 2**), and the 50% duck embryo lethal dose ($dELD_{50}$) was calculated by following the method of Reed and Munch (1938).

Pathogenicity test in day old duckling: An amount of 0.5 mL ($10^{5.6}$ $dELD_{50}$/mL) DPV suspension was inoculated in ducklings, and the ducklings were observed for 8 days.

Pathogenicity test in adult ducks: A small group of adult ducks were intramuscularly inoculated with 1 mL ($10^{5.6}$ $dELD_{50}$/mL) of DPV, and were observed for 12 days. Few adult ducks were kept separately as control.

DNA extraction and PCR: DNA was extracted using DNA extraction kit (Promega®, USA), following the instructions of the kit manufacturer. The primers described by Wu et al. (2011) and Zou et al. (2010) (Table 1) were used for amplification of the targeted DNA segments of DPV. A 50 µL reaction mixture was prepared by mixing nuclease free water (16 µL), PCR master mixture (25 µL) (Promega-Madison, WI, USA), forward primer (2 µL), reverse primer (2 µL), and DNA template (5 µL). Thermal condition used for the amplification of *DNA polymerase* gene was: initial denaturation at 94°C for 2 min; followed by 35 cycles of reaction comprising with 94°C for 1 min, 56°C for 1 min, 72°C for 2 min, with a final extension at 72°C for 7 min. On the other hand, thermal profile for PCR targeting *gC* gene was initial denaturation at 95°C for 5 min; followed by 45 cycles of reaction comprising with 94°C for 1 min, 58°C for 1 min, 70°C for 1 min, and a final extension at 70°C for 10 min.

Electrophoresis of the PCR products: An amount of 5 µL PCR products was mixed with 1 µL 6X loading dye (Promega, USA), and the mixture was loaded to the appropriate well of the 2% agar gel. After electrophoresis, the DNA was stained with ethidium bromide, and was visualized using UV trans-illuminator (Biometra, Germany).

Sequencing and phylogenetic analysis: PCR product of partial *DNA polymerase* gene was sequenced from International Center for Diarrheal Disease Research' Bangladesh (ICDDR'B). The partially amplified *DNA polymerase* gene product (446-bp) was sequenced. The nucleic acid sequence obtained from the PCR products was aligned with the known sequence of DPV available in the GenBank, and phylogenetic analysis was done.

RESULTS AND DISCUSSION

Isolation of duck plague virus: The prevalence of DPV in the Rajshahi, Netrokona and Mymensingh districts were 6.62% (n=6/37), 28.57% (n=10/35), and 4.55% (n=1/22), respectively. The overall prevalence rate of DP virus was 18.1% (n=17/94). Among the samples, 6.49% (n=5/77) cloacal swabs and 70.58% (n=12/17) visceral organ samples were positive for DPV; the results were confirmed by PCR (**Table 3**); these findings were almost similar with the reports of Hansen et al. (2000), Wallace et al. (2000) and Campagnolo et al. (2001). Several researchers could isolate DPV from cloacal swabs, liver, esophagus, intestine and proventriculus (Shawky et al., 2002; Akter et al., 2004; Wang et al., 2013), as we found in this study.

Propagation in embryonated duck eggs: The embryo mortality started from 5 days of PI. Subcutaneous hemorrhages were observed on the dead embryos. The CAMs were also hemorrhagic and thickened. The AFs of positive cases were found negative to slide HA. CAM was found highly suitable route for DPV

Table 2: Determination of duck embryo lethal dose ($dELD_{50}$).

Dilution of Inoculum	No. of dead embryo	No. of live embryo	Accumulated numbers			% of dead
			Dead embryo (A)	Live embryo (B)	Total (A+B)	
10^{-03}	5	0	13	0	13	13/13=100%
10^{-04}	4	1	8	1	9	8/9=88.8%
10^{-05}	2	3	4	4	8	4/8=50%
10^{-06}	2	3	2	7	9	2/9=22.2%
10^{-07}	0	5	0	12	12	0/12=0%

$$\textbf{50\% end point} = \frac{\text{\% of dead immediately above 50\%} - 50\%}{\text{\% of dead immediately above 50\%} - \text{\% of dead immediately below 50\%}}$$

$$= (88.8 - 50) \,/\, (88.8\text{-}22.2) = 38.2 \,/ 66.6 = 0.57 \approx 0.6$$

Table 3: Confirmation of virus by PCR using duck plague virus using specific primer.

Location of farm	Sample from dead duck	No. of sample	Confirmation of DPV by PCR		Total		
			Positive sample	% of positive sample	Sample	Positive	% of detection
Rajshahi	Cloacal swab	31	2	6.45	37	6	6.62
	Visceral organs	6	4	66.66			
Netrokona	Cloacal swab	26	3	11.54	35	10	28.57
	Visceral organs	9	7	77.77			
Mymensingh	Cloacal swab	20	0	00.00	22	1	4.55
	Visceral organs	2	1	50.00			
Total	Cloacal swab	77	5	6.49	94	17	18.10
	Visceral organs	17	12	70.58			

propagation because of obtaining highest virus titer. Similar findings were also reported by Marius-Jestin et al. (1987), Akter et al. (2004) and Hanaa et al. (2013).

Agar Gel Immunodiffusion Test (AGIT): All the suspected positive CAMs were processed and part of the processed CAM was used for AGIT. Diffusion bands appeared for all the positive samples (**Figure 1**), as described by Wu et al. (2011) and Shen et al. (2011).

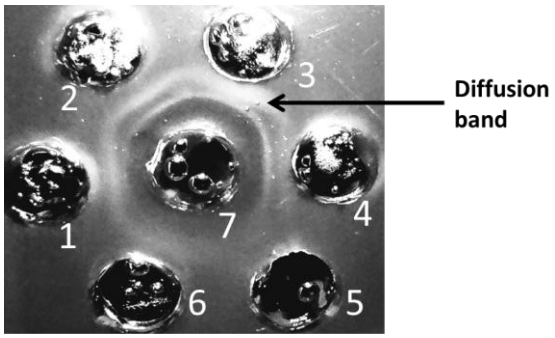

Figure 1. AGIT was carried out as per the procedures described in materials and methods. Well 1, 2, 3, 4 represents isolate 1, 2, 3, 4 of DPV respectively; well 5: Negative control; well 6: positive control (FnF® vaccine virus); and well 7: hyperimmune serum against DPV.

Passive haemagglutination test (PHA): The suspension prepared from the CAMs of positive samples were used as DPV source for performing PHA test. All the samples were found to hemagglutinate the tanned sRBC (**Figure 2**). This indicated that the samples used for PHA test were positive for DPV. Similar results were also described by Akter et al. (2004), Hossain et al. (2005), Islam et al. (2005), Rayhan (2008) and Das et al. (2009).

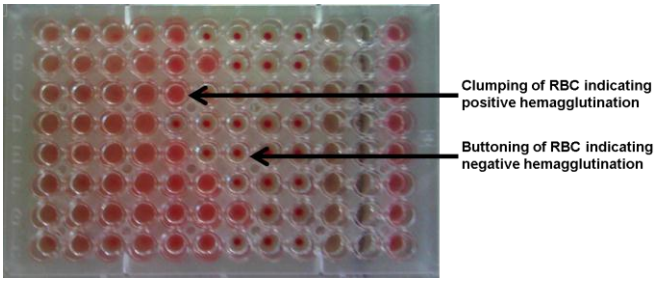

Figure 2. Passive hemagglutination test was conducted as described in materials and methods section; column 1-9; serial 2 fold dilution of known positive serum against DPV; column 10 contain negative control (negative serum + Virus + RBC); column 11 contain negative control (Virus + PBS + RBC) and column 12 contain positive control (Known serum + Virus + RBC). Raw 1 to 8 contain, virus isolate 1 to 8.

Detection of DPV by PCR: The expected PCR amplicon was appeared at 446-bp (**Figure 3**) for *DNA polymerases* and 78-bp (**Figure 4**) for *gC* gene. Our targeted *DNA polymerase* gene usually encodes UL31 protein according to the reports of Pritchard et al. (1999), Wallace et al. (2000), Zou et al. (2010), and Wu et al. (2011, 2012).

Results of pathogenicity tests:

$dELD_{50}$: The 50% end point was calculated using the formula (mentioned above) to the dilution that killed the embryo at rate immediately above 50%=$10^{-4.6}$. This dilution of the virus suspension contained one $dELD_{50}$

unit of virus in 0.1 mL. So, 1 mL of the virus suspension will contain ten times the reciprocal of the calculated dilution. Therefore, infectivity titer ($dELD_{50}$) of virus suspension/mL = 10 x$10^{4.6}$=$10^{5.6}$.

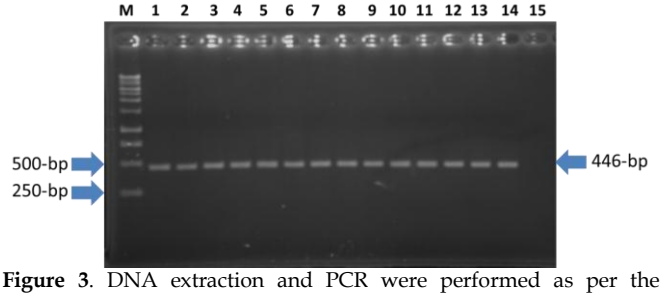

Figure 3. DNA extraction and PCR were performed as per the procedures described in materials and methods. Image showing the PCR products of duck plague virus with *DNA polymerase* gene specific primer. Lane M: 1-kb ladder; Lane 1-13: 1 to 13 isolates of duck plague virus; Lane14: positive control (FnF vaccine strain) and Lane15: negative control.

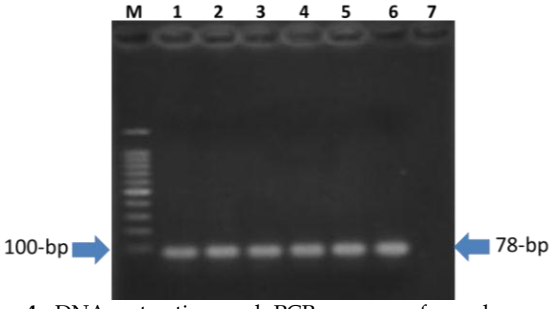

Figure 4. DNA extraction and PCR were performed as per the procedures described in materials and methods. Image showing the PCR products of DPV with *g*C gene specific primer. Lane M**:** 100-bp ladder; Lane 1-5: isolates of DPV (5 isolates of this study); Lane 6: positive control (FnF vaccine strain) and Lane 7: negative control.

Pathogenicity test in day old duckling: Our observation revealed that nervous signs began to appear as tremors of head, neck and body. Ducklings were unable to stand and they maintain a posture with drooping outstretched wings and head down showing weakness, depression, off feed, ataxia, diarrhea and death. On postmortem examination, pinpoint hemorrhages in liver were observed. These findings were similar as described by Hanaa et al. (2013). Virus was re-isolated from visceral organs and reconfirmed with PCR.

Pathogenicity test in adult ducks: All inoculated ducks showed clinical signs including paralysis of legs, back arched position, diarrhea, soiled vent and pasty eyes whereas the control ducks remained healthy without any clinical sign (Richter and Horzinek, 1993; King et al., 2012). Among these infected ducks, majority were around 6-9 days of PI. On postmortem examination, hemorrhage was observed on esophagus and liver with annular band on intestine. Body cavities were found filled with blood. These findings were inclined with the reports described by Barr et al. (1992), Davison et al. (1993), Calnek and Barnes (1997), Sandhu and Metwally (2008), and Hoque et al. (2011).

Sequencing and phylogenetic analysis: The phylogenetic tree derived from the sequence data is shown in **Figure 5**. The sequence was as follows:

5'AAGGCGGGTATGTAATGTACATTCCATTTACTGGAAATGCC
GTACATCTACACTATCGTCTCATCGACTGCCTTAAATCTGCTT
GCCGGGGATACCGTCTAATGGCTCATGTTTGGCATTCTACATT
CGTACTTGTCGTGAGGCGCGACCGCGAACGGCAAACTGACGTG
GACAGCGTACCACAGATAAGTATTGAAGATATTTATTGTAAAA
TGTGCGACCTTAATTTCGATGGGGAACTTCTGCTAGAATATCG
AAAGCTCTACGCAGCTTTTGACGATTTTCCTCCTCCTCGCTGA
GTGGCATCCCTGGGTACAAGCGCACTTCTGCAAACCCGGCCGA
AGATAGCAGTGCTGCGGTTTCGTCACTCTCGCGCAT 3'

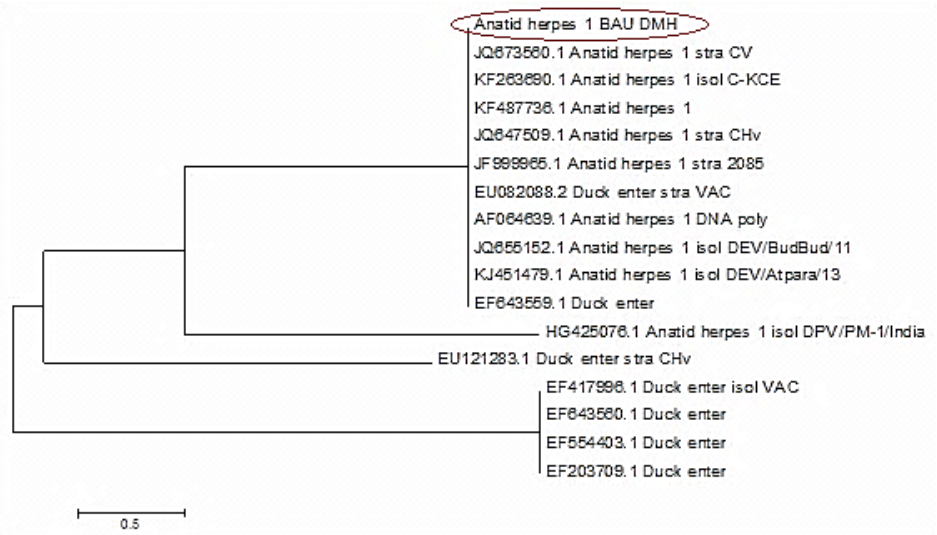

Figure 5. Phylogenetic relationship prepared from aligned sequences of the partial (378-bp) *DNA polymerase* gene of duck plague virus**.** Red circle indicates the Bangladeshi isolate.

Phylogenetic tree showed that sequenced strain of DPV (Anatid herpes 1 BAU DMH) was highly similar with the nucleotide sequence data retrieved from GenBank with JQ673560.1|:59037-59408 Anatid herpesvirus 1 strain CV and JQ647509.1|:59034-59405 Anatid herpesvirus 1 strain CHv, which were originated in China causing DP in domestic ducks and waterfowls. It also shows similarities with some other sequence data from GenBank (**Figure 5)**.

CONCLUSION

A total of 17 (18.10%) DPV isolates are obtained from 94 suspected samples. The isolates are confirmed by AGIT, PHA, PCR and sequencing. The pathogenicity tests reveal that the isolates are highly pathogenic. Sequenced data and the phylogenetic analysis indicate that our isolate (Anatid herpes 1 BAU DMH) is highly similar with Anatid herpesvirus 1 strains reported from China (JQ673560.1 and JQ647509.1).

ACKNOWLEDGEMENT

The research work has been conducted with the financial supports from the World Bank through the University Grant Commission (UGC) of Bangladesh under HEQEP project entitled- *Strengthening and expansion of post-graduate research capabilities for the development and production of inexpensive livestock and poultry vaccines* (CP007).

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