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Research Article

MOLECULAR AND PATHOLOGICAL EVIDENCE OF DUCK VIRAL ENTERITIS: FIRST CONFIRMED REPORT IN MANILA DUCK (*CAIRINA MOSCHATA*) IN SOUTHERN INDIA

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ABSTRACT: This study reports the foremost incidence of duck viral enteritis (DVE) outbreak in a free-range flock of 250 Manila ducks from Tamil Nadu, Southern India. A sudden 20% (n=50) mortality was observed in the duck flock, presenting clinical signs such as greenish or blood-stained diarrhea, anorexia, ocular discharge, incoordination, and death. Postmortem examination of randomly selected 10 carcasses revealed blood in the body cavities, hemorrhages in the heart, air sacs, proventriculus, liver, and spleen, and enlargement of kidney, spleen, and liver with necrotic foci. Histopathological examination evidenced congestion and hemorrhages in the proventriculus, heart, and air sacs. The liver exhibited diffused congestion and hepatic steatosis. Spleen evinced congestion, hemorrhages, and multifocal necrotic areas. The small intestine showed blunting and fusion of villi with mononuclear cell infiltration. Screening of pooled viscera demonstrated no specific bacterial etiology and was negative for NDV and DHAV genomes. PCR screening for the DVEV genome targeting partial UL-31 showed amplification of 446bp amplicon in all the 10 carcasses examined. Sequencing and alignment of PCR amplicon from one sample yielded 373 nucleotides encoding 94 amino acids and gene sequence submitted to the GenBank with accession number of OR725707. BLAST homology search showed 100% sequence identity with published *Anatid alphaherpesvirus 1* genomes available in GenBank. Phylogenetic analysis revealed specific grouping along with published DVEV genomes of India, Bangladesh, China, USA, and Germany. Further, deduced amino acid comparison with established DVEV amino acid sequences also showed a 100% match to the published DVEV sequences.

Keywords: Duck viral enteritis, Manila Duck, Gross pathology, Histopathology, Molecular diagnosis.

INTRODUCTION

Duck rearing is a significant traditional animal husbandry activity in rural India. In recent years non-vegetarian people's preference has shifted from chicken to duck meat mostly in Asian countries [1]. As per the 20th (2019) Livestock census of India, the total poultry population of India is 851.81 million of which duck constitutes 4% (33.51 million). The growth percentages of both duck and fowl increased by 42.36% and 16.64% respectively in comparison to the 19th (2012) livestock census of India signifies the emergence of duck farming in India [2]. There is a variety of duck breeds like Khaki Campbell, Indian Runner, White Pekin, Rouen, Muscovy, and Aylesbury maintained as a livelihood to earn substantial income. Hence, understanding duck farming, disease dynamics, and its control strategies is essential for improving alternate poultry species, especially duck which is 2nd line of source for both egg and meat production and contributes to the nutritional food security of the country.

Manila (Muscovy) duck, *Cairina moschata* domesticated from South America is spread throughout the world [3]. It is a meat-type breed and produces good quality lean meat rich in polyunsaturated fatty

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acid than the other species of duck which makes it healthier to consume [4, 5]. Though, Muscovy duck is highly resistant to infectious diseases in comparison to meat-type chickens [6] and they are susceptible to diseases such as avian influenza, duck viral enteritis/ duck plague, Newcastle Disease, duck viral hepatitis, Pasteurellosis, Salmonellosis, Aflotoxixosis, etc., [7]. *Anatid alphaherpesvirus 1* (AnAHV-1) causes most of the duck viral enteritis outbreaks in ducks or Duck viral enteritis virus (DVEV) belongs to the genus *Mardivirus* and family *Herpesviridae*, affects both wild and domestic ducks and water birds [8, 9, 10]. The first report of DVE was documented in the Netherlands in 1923 followed by the USA, Canada, UK, Hungary, Denmark, Austria, the Netherlands, France, Belgium, China, Vietnam, India, Bangladesh, and Thailand with huge mortality in domestic and wild duck, geese, swan and other waterfowl [11, 12, 13]. In India, the outbreak of DVE was recorded from West Bengal in the year 1965 as first and foremost [14, 15] with varying mortality from 50-100 percent in different flocks.

DVE outbreaks result in huge mortality, reduction in egg production, and hatchability leading to abrupt economic loss to duck farmers [16]. Mortality pattern in infected birds varies from 5 percent to 100 percent depending on the pathogenicity of the infecting virus, immune status, species of bird, and breed of the duck [17, 18, 19]. The main symptom in infected ducks includes massive bleeding in the body cavity, intestine, spleen, thoracic cavity, and lungs. DVE is diagnosed and confirmed based on symptoms, history of the diseases that occurred, postmortem and histopathological findings, virus isolation, and molecular confirmation [19, 20]. This study documents, clinical manifestations, gross and histopathology, molecular detection, and phylogenetic study of DVE in Manila duck (*Cairina moschata*) from Tamil Nadu, Southern India.

MATERIALS AND METHODS

Case description, sample collections

A duck farm with a capacity of 250 Manila ducks from Cheyyur village in Chengalpattu District, Tamil Nadu, India, had a sudden death of 50 birds within two days of clinical signs in 26 weeks age group birds during June 2022 with no previous vaccination history in the flock. Necropsy examinations were performed on 10 randomly selected carcasses. Representative samples were collected in ice and 10% neutral buffered formalin and sterile container for bacteriology/ molecular studies and histopathological studies respectively following standard protocol. The collected samples were transported to the laboratory as earliest under refrigerated conditions and processed. The remaining samples were stored at - 40°C till analysis.

Histopathological study and screening for bacterial etiology

Cryostate sectioned, hematoxylin and eosin-stained tissues were examined for histopathological changes in light microscopy as per the methods described by Akter *et al*. [21]. Sterile pooled tissue samples from each bird were subjected to isolation of various bacterial organisms as per methods described by Quinn *et al*. [22]. For isolation of bacteria Nutrient agar (NA), Blood agar (BA), MacConkey agar (MCA), Brain heart infusion (BHI) agar, and Clostridial agar (CA) were used. The bacterial growth was confirmed on the basis of microbiological and biochemical characterization [23].

Molecular screening for viral etiologies - DVEV, NDV, DHAV genomes

Pooled tissue samples were stored at - 40ºC from each bird and subjected to nucleic acid extraction using commercial DNeasy Blood and Tissue Kit (Qiagen, Germany) as per the manufacturer's protocol. DVEV genome screened by PCR assay using primer sets of 5'AAGGCGGGTATGTAATGTA3' and 5'CAAGGCTCTATTCGGTAATG3' respectively that bind to the 57857 - 57876 and 58283- 58302 positions targeting partial UL-31 and polymerase gene in DVEV genome was used to amplify a particular product of 446 bp [24]. PCR mixture contained 12.5 µl of 2x ampliqon red dye master mix (Ampliqon, Denmark), 10 picomoles of each forward and reverse primer (Eurofins Genomics of India Pvt. Ltd, Bengaluru), 1 µl of template DNA, and nuclease-free water up to 25 µl were used with the published cycling conditions.

Further, total RNA was extracted from all the ten pooled carcasses using TRIZOL® LS reagent (Invitrogen, USA) and cDNA was synthesized using Prime Script™ RT reagent kit, (Takara, Japan) following manufacturer instructions and screened for Ranikhet disease virus by amplification of 254 bp product specific to the F gene of NDV [25, 26, 27] and duck hepatitis A virus (DHAV) by amplification of 467 bp specific to 3D gene of DHAV primer [28].

Sequencing, BLAST homology search, and Phylogenetic analysis

The specific PCR amplicon was purified by a PCR purification kit (Qiagen, Germany) and sequenced at

an outsourcing facility (Eurofins Genomics of India Pvt. Ltd, Bengaluru). Contig sequence aligned in BioEdit version 7.2 with published DVEV sequences from NCBI-GenBank. The specificity of the assembled contig sequence obtained in this study was analyzed in NCBI-BLAST search. For Comparative phylogenetic analysis, DVEV partial UL-31 and DNA polymerase gene sequence seen in this study was compared with DVEV sequences from GenBank and subjected to the Molecular Evolutionary Genetics Analysis version 11 (MEGA version 11) tool for Phylogenetic and evolutionary studies [29, 30].

RESULTS AND DISCUSSION

Clinical signs, gross pathology, and histopathological findings

Clinical examination revealed greenish or blood-stained diarrhea, anorexia, ocular discharge, and incoordination, followed by death. Overall, carcasses are highly emaciated and dehydrated, gross findings include blood in the body cavities, hemorrhages in the heart, air sacs, proventriculus, liver, and spleen, enlargement of kidney, spleen, and liver with necrotic foci (Fig. 1). Histopathological examination revealed congestion and hemorrhages in the proventriculus, heart, and air sacs. The liver exhibited diffused congestion and hepatic steatosis. Spleen evidenced the presence of congestion hemorrhages and multifocal necrotic areas. Small intestines were revealed with blunting and fusion of villi and mononuclear cell infiltration (Fig. 2).

Screening for bacterial and viral etiologies

Bacterial isolation studies of all ten pooled viscera (liver, lungs, spleen, and heart) from each carcass individually in NA, MCA, BA, BHI agar, and CA evidenced no specific bacterial etiology. NDV and DHAV viral genome screening by targeting its specific FPCS gene and 3D gene respectively evidenced no specific amplification confirming its absence in the sample.

Table 1. List of published DVEV sequences retrieved from GenBank and used in phylogenetic analysis.

S. No.	Accession No.	Virus and strain	Country	Year of submission
1.	OP502744	Aalpha HV1DPV	Bangladesh	2020
2.	MN937272	Anatidalphaherpesvirus-MHBAU-DPV-BR1-UL31	Bangladesh	2020
3.	EU121283	DEV strain CHv DNA polymerasegene	China	2006
4.	EF643559	DEVUL31	China	2010
5.	MZ824102	Anati dalpha herpesvirus-1DEV India IVRI	India	2016
6.	MZ911871	Anatid alpha herpesvirus-1-DPvac IVRI	India	2021
7.	KJ451479	Anatid herpesvirus1-India 2014	India	2014
8.	KM012009	Anatid herpesvirus1CDIO-duck01-UL31Kerala	India	2014
9.	KJ958921	Anatid herpesvirus1CDIOdpv-01 Kerala	India	2015
10.	JQ655152	Anatid herpesvirus-1isolate DEV-Bud Bud-11West Bengal	India	2012
11.	MZ574076	Anatid alpha herpesvirus-Assam	India	2021
12.	MH384834	Anatid alphaherpesvirus-1	Egypt	2016
13.	AF064639	Anatid herpesvirus-USDA-Vaccine strain	USA	1999
14.	NC075687	Anatid herpesvirus-1	Germany	2005

Fig. 1. Carcass condition and gross lesions of ducks infected with DPV at autopsy. [A: Emaciated carcass with soiled vent and beak; B: Heamorrhages in intestine, spleen and liver; enlarged of spleen, liver with few necrotic area].

Molecular detection, sequencing, and phylogenetic analysis of DVEV

PCR-based screening for the DVEV genome targeting partial UL-31 showed 446 bp amplification in pooled viscera of all the 10 carcasses examined (Fig. 3). Randomly specific amplicon from one sample was sequenced, and annotated with a contig sequence length of 373 nucleotides encoding 94 amino acids. The sequence was submitted to the Gene Bank database, with an acceptance number of OR725707. BLAST homology search showed 100% sequence homology with published *Anatid alphaherpesvirus 1* genome of India, Bangladesh, China, USA, and Germany available in GenBank. Phylogenetic analysis of the contig

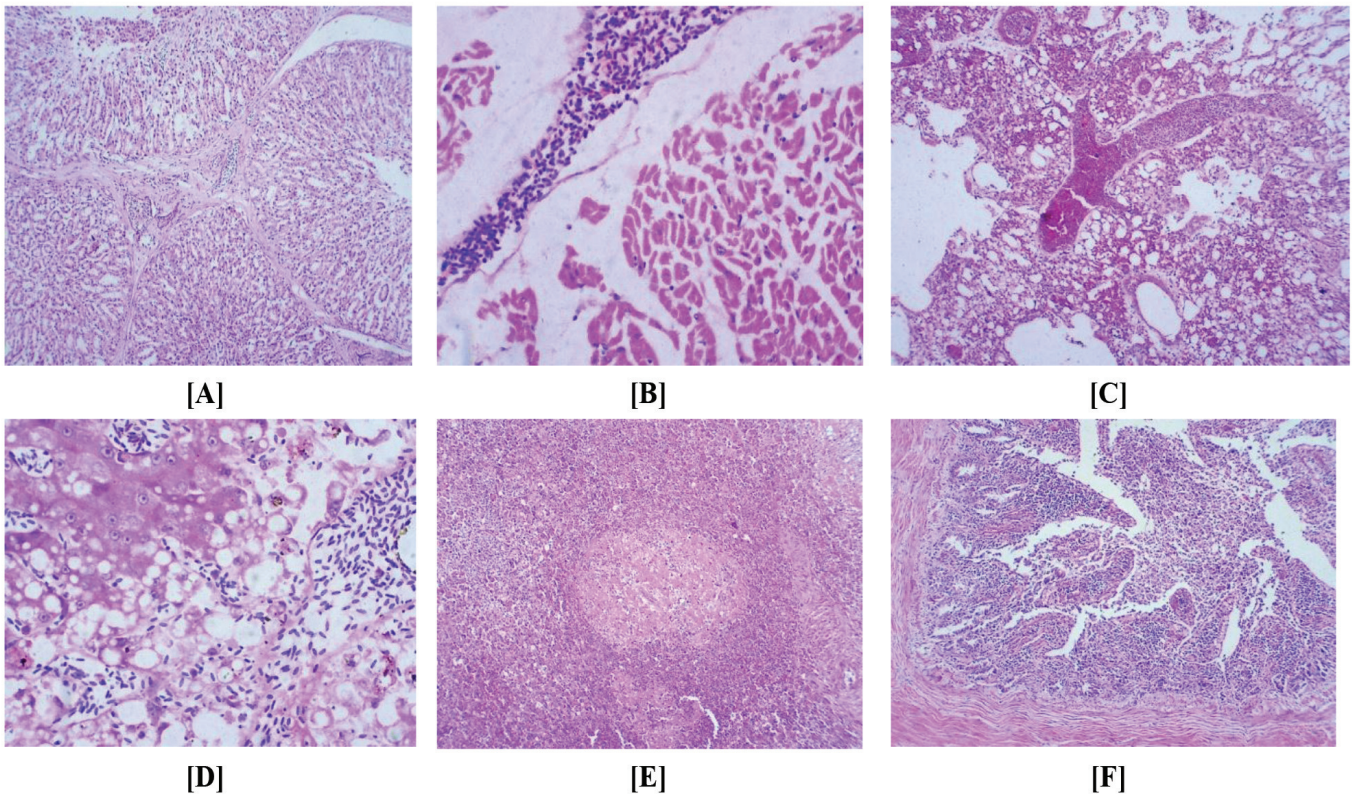
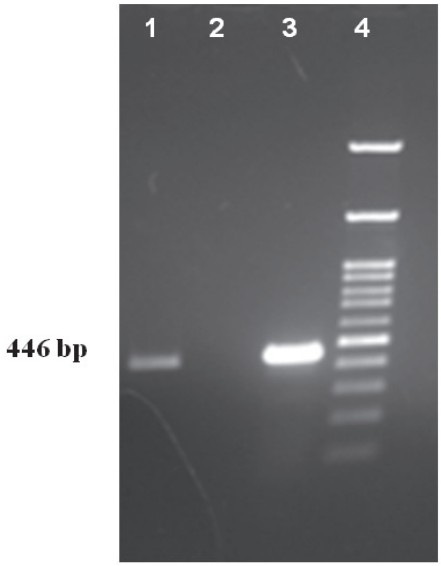


Fig. 2. Histopathological findings in the viscera. [H&E staining 40X]. [A: Proventriculus exhibited with mild congestion; B: Heart showing presence of few congestion and haemorrhages; C: Air sacs revealed with multifocal congestion and haemorrhages; D: Liver exhibited with diffused congestion and hepatic steatosis; E: Spleen showing presence of congestion and haemorrhages and multifocal necrotic areas; F: Small intestines revealed with blunting and fusion of villi and mononuclear cells infiltration].



Lane 1- DVEV-known positive DNA
Lane 2- Non template control
Lane 3- Field sample
Lane 4- 100 bp ladder

Fig. 3. Molecular screening of DVEV by specific PCR assay.

(assembled) sequence obtained in this study along with fourteen DVEV sequences (Table 1) by MLT, and NJ method in MEGA 11 revealed similar grouping along with published DVEV genomes (Fig. 4). Analysis of deduced amino acid sequences of DVEV, partial UL31gene from this study with 14 published DVEV sequences from GenBank evidenced identical AA sequences of 12 DVEV amino acid sequences confirming its genetic identity (Fig. 5). Based on gross, histopathological, and molecular findings DVE infection was diagnosed and confirmed in Manila ducks in Tamil Nadu, southern India.

DVE is a highly pathogenic and infectious disease extensively documented and focused in the past few decades in duck farming [31]. The present study documents the first incidence of DVE infection in Manila (Muscovy) ducks in Tamil Nadu, southern India with 20% mortality. Earlier, DVE in the Chara-Chemballi breed of ducks in southern India with similar clinical picture with 44.4% mortality [32]. Mortality rates in DVE vary, determined by virus genome and host immunological conditions [33, 34]. The present study documented the DVE outbreak

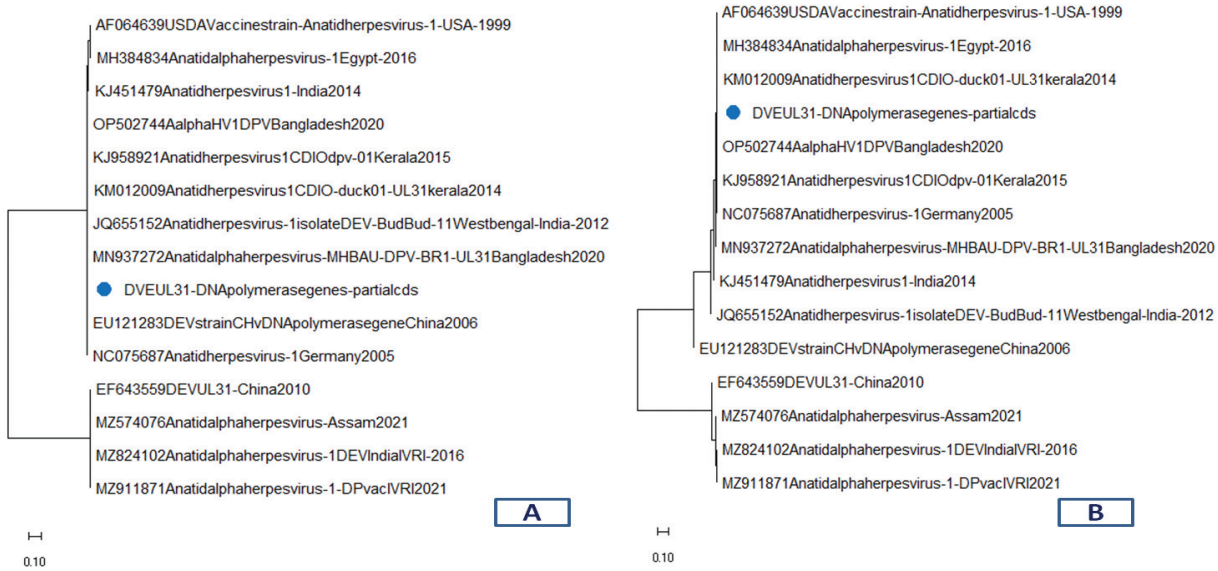


Fig. 4. Phylogenetic and Evolutionary analysis of DVEV. [A: Maximum likelihood method; B. Neighbor-joining method. The trees were drawn with obtained query sequence shown with a blue solid circle and 14 DVEV published sequences available in GenBank. The analyses were conducted in MEGA 11 with Bootstrap replicates of 1000. Bootstrap values are shown next to the branches in the phylogenetic tree].

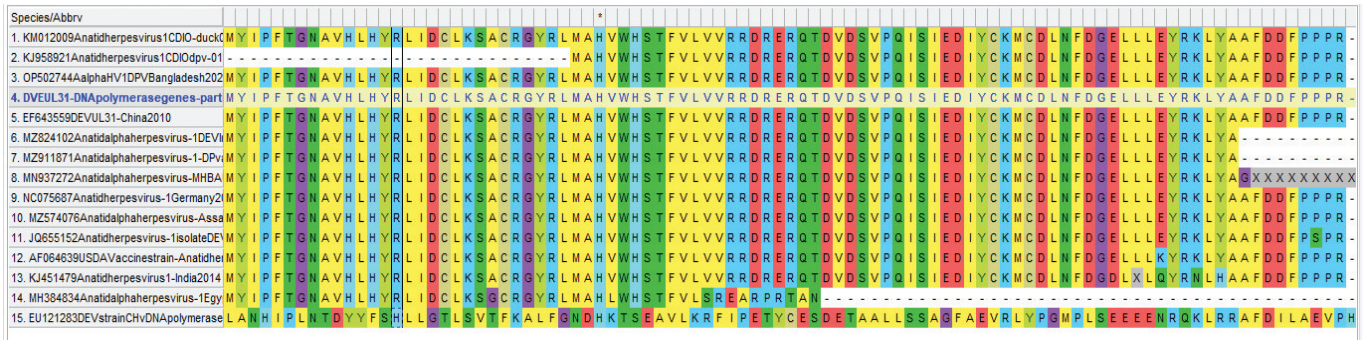


Fig. 5. Alignment of deduced amino acid sequences of DVEV partial UL31ene. [This alignment included deduced amino acid sequences of DVEV partial UL31 gene from this study (Marked as blue in the serial number 4 with 14 published DVEV sequences from GenBank. All the sequences were aligned in Clustal W].

during the hot month of June 2022. The majority of the DVE outbreaks documented in around Asia are during March-June [19]. Few studies witnessed that severe hot and humid conditions during the March - June months may induce stress which may lower immunological competence in the host that might have favored reactivation of virus from latency [35]. Clinical, gross, and histopathological findings recorded in the present DVE-infected Manila ducks are similar to earlier reports of DVE in other duck breeds [36, 37]. Hemorrhages in the viscera and the enteric tract are the predominant findings in this DVE infection, the possible reason might be increased vascular permeability associated with the multiplication of viruses leading to hemorrhages in internal organs [16].

PCR is a highly specific and sensitive molecular technique for disease detection followed all around the globe. As per OIE recommendations UL-31 and polymerase gene-based primer pair was used for molecular diagnosis in this study. There are several reports around the world on the specificity of this primer for DVEV molecular confirmation [38, 39]. The phylogenetic analysis revealed that the DVEV genome sequence in this study showed an evolutionarily close relationship with DVEV isolates of India, Bangladesh, the USA, Egypt, China, and Germany. Similarly, genetic analysis of duck plague virus from Bangladesh and evidenced similarity to Chinese and Indian isolates of DPV. Whereas, the first whole genome of the duck plague virus from India showed 99.98% and 99.8% similarity at the amino acid and nucleotide respectively to the European DVEV strain [12, 40]. The above evolutionary studies demonstrate the genetic similarity of DVE viruses identified in different water birds, ducks, and swans in different agro-climatic regions from different parts of the world. Duck viral enteritis

genomes from various countries share a high similarity [40]. DVE is categorized into pathogenic strains and nonpathogenic strains depending on the virulent-associated gene UL2, which codes for uracil-DNA glycosylase (UNG), an enzyme plays a major role in viral multiplication and pathogenesis [41, 42].

The source of the DVEV outbreak is unknown, but the ducks were purchased from a local market that trades ducks across state borders [43, 44]. DVE has been periodically reported in Tamil Nadu since 1979 [45], 2005 [46], 2017 [47] and 2019 [32]. There is the possibility of disease spread from the infected/stressed duck to healthy birds by direct contact and through fomites in the contaminated shandy where animals, agricultural goods, and other items are traded [19, 39]. The ailing and suspected ducks in the affected flocks were segregated and treated symptomatically with broad-spectrum antibiotics and vitamin supplements at the same time healthy birds were vaccinated with the Duck plaque vaccine (Holland strain live attenuated DVE vaccine) and the mortality rate was controlled within 7 days of an outbreak following strict biosecurity measures such as sanitization practices of duck pens, utensils, etc. [48]. Further study needs to be extended to other duck breeds/water fowls maintained in the near vicinity and other districts of Tamil Nadu and neighboring states to screen clinical and latent DVEV genome or specific antibodies both in vaccinated and unvaccinated flocks which through light on devising control strategies for DVE infection.

CONCLUSION

This study concludes that this is the foremost report on the molecular identification of Duck Viral Enteritis Virus from post-mortem samples of Manila ducks. Extensive studies are needed to determine the pathological potential and to select the appropriate vaccine-candidate virus for controlling field outbreaks.

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