



## Duck virus enteritis (duck plague)

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**Abstract:** Duck virus enteritis (DVE), also called duck plague, is one of the major contagious and fatal diseases of ducks, geese and swan. It is caused by duck enteritis virus (DEV). DVE has worldwide distribution, wherein migratory waterfowl plays a crucial role in its transmission within and between continents. Furthermore, horizontal and/ or vertical transmission plays a significant role in disease spread through oral-fecal discharges. Either of sexes from varying age groups of ducks is vulnerable to DVE. The disease is characterized by sudden death, vascular damage and subsequent internal hemorrhage, lesions in lymphoid organs, digestive mucosal eruptions, severe diarrhea and degenerative lesions in parenchymatous organs. Huge economic losses are connected with acute nature of the disease, increased morbidity and mortality (5%–100%), condemnations of carcasses, decreased egg production and hatchability. Although clinical manifestations and histopathology can provide preliminary diagnosis, the confirmatory diagnosis involves virus isolation and detection using serological and molecular tests. Most of the affected birds die without ample clinical manifestations and even sometimes the carcasses are found floating on the water surface. This review describes DEV, epidemiology, transmission, the disease (DVE), pathogenesis.

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### Introduction

#### Duck virus enteritis (DVE) is also known as duck plague (DP)

The disease is caused by Anatid herpesvirus type 1, a member of the Herpesviridae family and subfamily Alpha-herpesvirinae (Fadly et al. 2008; Li et al. 2009; King et al. 2011).

With an acute but sometimes chronic and highly contagious nature, DVE causes considerable mortality among domestic and wild ducks, swans, geese and other waterfowl of different ages. The disease is known to have global distribution, wherein migratory waterfowl plays a crucial role in disease transmission within and between continents. However, mortality and severity of the disease varies between epizootics and species involved or affected (Keymer and Gough 1986; Kaleta et al. 2007). Extensive epizootics have been reported in duck farms in the United States of America.

However, the majority of investigations have failed to isolate the virus (Brand and Docherty 1984). Besides Anseriformes, outbreaks have never been seen in other avian species, mammals & humans (Sandhu and Shawky 2003; Sandhu and Metwally 2008). Most of the affected birds die without ample clinical manifestations and even sometimes the carcasses are found floating on the water surface

(Montali et al.1976). However, when clinical symptoms are evident, high mortalities especially in older ducks, vascular damage and subsequent internal hemorrhages (Proctor 1975), lesions in lymphoid organs, digestive mucosal eruptions, severe diarrhea and degenerative lesions in parenchymatous organs (Davison et al. 1993) following fatal outcomes;

Davison et al. 1993; Shawky et al. 2000; Campagnolo et al. 2001; Sandhu and Shawky 2003) are noticed. Partially closed eyelids with photophobia, extreme thirst, loss of appetite, ataxia, nasal discharge drooping plumage, watery diarrhea, soiled vents and tremors of head, neck and body (Davison et al. 1993). Domestic and wild ducks, geese and swans of all ages are considered susceptible, wherein the infection may, at times, exhibit chronicity or latency (Richter and Horzinek 1993; Sandhu and Shawky 2003). After establishing primary infection, duck enteritis virus (DEV) exhibits latent infection in trigeminal ganglia (TG). From this site, re-activation of the virus can occur that results in disease outburst (Shawky and Schat 2002). morbidity and mortality of birds range from 5 to 100% (Jansen 1961) persists in the flock with significant drop in egg production (Goldberg et al. 1990). Mortality usually starts at 1–5

days after the onset of clinical signs and is more evident in adult breeder ducks. However, death rarely occurs in chronically infected flocks. Recovered birds usually become carriers and excrete the virus in the feces over a period of several months (**Horzinek 1993; Shawky and Schat 2002**). Though carrier birds are resistant or immune to the disease, virus shedding results in disease spread to susceptible waterfowls (**Sandhu & Shawky 2003**). The main causes, that can perpetuate an outbreak, are bird-to-bird contact and/or contact of susceptible birds-to-contaminated environment (**Burgess and Yuill 1983; Richter and Horzinek 1993; Shawky and Schat 2002**). Scavenging and decomposition of infected carcasses may also contaminate and spread the virus in environment. Several reports indicate virus transmission through eggs of infected birds. However, its significance in the disease cycle remains unclear (**Burgess and Yuill 1983; Sandhu and Shawky 2003**). Due to high mortality, condemnations, decreased egg production & hatchability, significant economic losses are associated with DVE across the globe. An excess of \$1 million losses were reported in 1967 during the first outbreak in the United States of America' duck industry of long island, New York (**Walker et al. 1969**). The virulent strain of DEV can be adapted by several passages in duck embryo and chicken embryo fibroblast (CEF) cell culture (**Johnson et al. 1990; Mondal et al. 2010; Doley et al. 2013**)

#### **Etiology**

**The etiological agent of DVE is DEV or Anatid herpesvirus-**

As per the recent taxonomic classification by the International Committee on Taxonomy of Viruses (ICTV), DEV has been classified into the genus *Mardivirus*, subfamily *Alpha-herpesvirinae* of the family *Herpesviridae*.

(**Fadly et al. 2008; Li et al. 2009; King et al. 2011**). The virus has non-hemagglutinating and non-hemadsorbing properties (**Jansen 1961; Hess and Dardiri 1968**). Having a diameter of about 120-130 nm, with globular shape, the enveloped Herpesvirus has four structural components that includes a bilayer-lipid envelope, an amorphous tegument, an icosahedral capsid and a linear double-stranded DNA with G+C content of 64.3% or G+C content of 44.9% (**Gardner et al. 1993; Yuan et al. 2005**). The genomes may vary in base composition, sequence arrangements and size. A significant difference may also be seen in arrangement of inverted and directly repeated sequences (**Hayward et al. 1975; Wadsworth et al. 1976**). Partial or complete genomic sequences of DEVs are rapidly accumulating and its analyses demonstrated that, although similar to other herpes viruses, the DEV genome also varies (**Li et al.**

**2009; Wang et al. 2011; Liu, Han, et al. 2011; Liu, Chen, et al. 2011; Wu et al. 2012a; Wu et al. 2012b; Yang et al. 2013**). the virus is sensitive to ether and chloroform. While using heat during inactivation experiments, 10 minutes at 56 °C or 90–120 minutes at 50 °C, the infectivity has been nullified (**Sandhu & Shawky 2003**). At room temperature (22 °C), infectivity lasts up to 30 days. Drying over calcium chloride at 22 °C resulted in inactivation of the virus after 9 days. The DEV rapidly gets inactivated beyond pH 3–11. The infectivity has been found to be destroyed by treating the virus for 18 hours at 37 °C to trypsin, chymotrypsin and pancreatic lipase (**Hess & Dardiri 1968**). Recently, the bioinformatics data based codon usage bias analysis between the newly identified DEV gD gene (GenBank accession no. KC915041) and the gD like gene of 23 other reference herpesviruses revealed that codon of gD gene of DEV had strong bias towards the synonymous codons with A and T at the third position; existence of a high level of diversity in codon usage; and the G + C content constrained the genetic heterogeneity in gD gene. The study pointed out yeast expression system to be more appropriate for the expression of DEV genes. These findings may help towards understanding the evolution and pathogenesis of this virus in a better way, offer a basis for knowing the associated mechanism for biased usage of synonymous codons, identifying suitable heterologous expression system for improving target gene expression, and pave way for development of newer vaccines and diagnostics (**Aravind et al. 2014**).

#### **Transmission**

The susceptible population may get exposed both by direct contact to infected bird as well as indirectly from virus contaminated environment (**Sandhu & Shawky 2003**). As the waterfowl is dependent on an aquatic environment, transmission through water seems to be a prime source. Most of the outbreaks in domestic ducks have occurred in the proximity of open water bodies which are often shared by free-flying waterfowl (**Richter & Horzinek 1993; Sandhu & Shawky 2003**). Oral, intranasal and/or parenteral administration of virus infected tissues can establish experimental infection in susceptible ducks. The convalescent birds may get immune or resistant to reinfection. However, they may become carriers and shed DEV into the environment for a prolonged period (**Shawky & Schat 2002**). The role of migratory waterfowls as carriers of DEV has been reported during many outbreaks (**Woźniakowski and Samorek-Salamonowicz 2014**) Horizontal spread is the principal mode of transmission (**Sandhu & Shawky 2003**). Attempts to isolate DEV from eggs laid during a natural outbreak have not been successful (**Burgess & Yuill 1981a**). Vertical transmission has

been reported in persistently infected waterfowl (**Burgess & Yuill 1981b; Burgess & Yuill 1983; Gough 2008**). The course and direction of DVE infection are dependent on population density as well as rate of transmission between infected and susceptible birds (**Sandhu & Shawky 2003**). The DVE outbreaks, except in August and September, have been regularly reported throughout the year.

Approximately 86% of these outbreaks have been reported from March to June. Hitherto reports have revealed spontaneous shedding of the virus from convalescent birds during the spring season. This pattern of disease outbreak might be due to the stresses resulting from the physiological changes in the duration of daylight and onset of breeding that trigger virus release during spring season (**Pearson & Cassidy 1997; Converse & Kidd 2001; National Wildlife Health Center 2011**).

#### Pathogenesis

**Xuefeng et al. (2008)** studied the pathogenesis of DVE in experimentally infected ducks by oral route. The results indicated a close relationship between the amount of DEV in internal organs and disease progression. In duck plague virus (DPV) infected ducklings, antiviral immunity comprised of noticeable presence of pattern recognition receptors (PRRs) significantly appears along with observable typical pathological lesions and symptoms. Study based on real-time quantitative PCR and TaqMan<sup>TM</sup> fluorescent quantitative real-time PCR with specific primers and probes also confirmed hike in the protective innate immune response and is helpful in finding the break points during pathogenesis to lower the establishment of infection of DPV/DEV in ducks (**Zou et al. 2010; Li, et al. 2016**). Even though differences in virulence have been observed among DEV strains, antigenic nature remains identical for most of the isolates (**Kisary and Zsak 1983; Akter et al. 2004**). Upon its entry into a susceptible host, the virus multiplies in the mucosal epithelial cells of the gastrointestinal tract, the esophagus, and proceeds towards thymus, bursa of Fabricius, spleen and liver (**Islam & Khan 1995; Shawky and Schat 2002; Sandhu and Shawky 2003**). Protein kinase C inhibitor has been suggested as the receptor for nucleoprotein of DEV (**Hang et al. 2012**). The epithelial cells/ macrophages in these organs are the major predilection sites of virus multiplication (**Shawky 2000; Yuan et al. 2005**). The virus induces apoptosis as well as necrosis in lymphoid tissues such as epithelial cells between the cortex and medulla of the follicles in the bursa of Fabricius, Hassall's corpuscle of the thymus, germinal centers in B lymphocytes, periarteriolar lymphoid sheath in T lymphocytes, sinusoidal lining cells in the spleen resulting in the depletion of lymphocytes and

subsequent immunosuppression. Before necrosis occurred, all these organs contained nucleocapsids of virus in the nuclei and virions in the cytoplasm of the host cells (**Guiping et al. 2007**). The DEV also has strong predilection for vascular endothelial cells. Virus replication in vascular endothelial cells of small blood vessels, venules and capillaries results in their destruction leading to severe hemorrhages, eruptions and progressive degenerative changes of parenchymatous organs (**Richter and Horzinek 1993**). In young birds, the virus primarily targets the lymphoid organs rather than other systems. While in adult birds, the pathologic effects are more pronounced in digestive tract and other internal organs. To know more regarding pathogenesis of DEV after infecting the duck embryonic fibroblast cells under in vitro conditions either after antiviral therapy, immunization or through biomarker response, a cDNA library has been framed with the use of switching mechanism at 5' end of the RNA transcript technique for proteomic analysis of DEV infected duck embryonic fibroblast cells (**Gao et al. 2014**).

#### Clinical signs

The incubation period of disease ranges from 3 to 7 days (Fenner et al. 1993). Clinical signs vary according to species, age, sex, immune status of the affected bird and the strain of DEV involved (**Sandhu & Metwally 2008; OIE 2012**). Severity in clinical symptoms is observed with the progression of infection in the flock. Beside sudden death, the common clinical signs include depression, loss of appetite, increased thirst, dehydration, weakness, ruffled feather, nasal discharge, ataxia, photophobia, tremor of head and neck, greenish and watery diarrhea, and soiled vent (**Campagnolo et al. 2001; Sandhu & Shawky 2003; Gough 2008; Sandhu & Metwally 2008**). Haematochezia is a common feature. In some birds, ophthalmic signs such as lacrimation, watery ocular discharge, photophobia and diptheroid plaques around the eyelids are observed. Due to ocular signs, some birds often refuse to drink which further exacerbate the dehydration and its sequel. Respiratory signs are often manifested as a hoarse chirp. However, it is non-specific followed by a drop in egg production and a ruffled, unkempt appearance. Death usually occurs within 5 days of onset of clinical symptoms with high mortality (60%–90%) and about 25%–40% drop in egg production (**Sandhu & Shawky 2003; Carter et al. 2006**). In case of ducklings of 2–7 weeks of age, symptoms like dehydration, loss of weight, conjunctivitis, lacrimation, nasal exudate, bluish discoloration of beak and blood-stained vent are noticed (**Gough 2008; Sandhu & Metwally 2008**).

#### Gross lesions

The gross lesions inflicted by DEV depend upon the species infected, age of the bird, and stage of infection in host, strain and inoculum of virus (Sandhu & Shawky 2003; Sandhu & Metwally 2008; OIE 2012). Commonly observed lesions are vascular damage, disseminated intravascular coagulopathy and necrotic changes, eruptions on the mucosal surface of the digestive tract and degenerative lesions in parenchymatous and lymphoid organs. Severe enteritis, hemorrhage in intestine, body cavities, heart, pericardium, liver and spleen, plaques in esophagus and intestine, lesions in thymus and bursa are highly suggestive of infection (Jansen 1961; Wobeser 1987; Davison et al. 1993; Richter & Horzinek 1993; Shawky 2000; Campagnolo et al. 2001; Sandhu & Shawky 2003; Konch et al. 2009). Patechial or larger extravasations of blood could be seen on myocardium and epicardium giving a red 'paintbrush' appearance (Weingarten 1989; Richter & Horzinek 1993; Konch et al. 2009). The lesions in the digestive tract are commonly seen in oral cavity, esophagus, ceca, rectum and cloacae. The oral lesions comprises of erosions and presence of diphtheritic sub-lingual membranes. Chronically infected waterfowl have oral erosions at the openings of sub-lingual salivary gland ducts (Konch et al. 2009). In esophagus, the lumen gets lined with yellowish-white membrane or, in some cases, there may be sloughing of the entire mucosa. The esophago-proventricular sphincter maybe seen as a hemorrhagic ring. The lumen of intestine may be filled with blood and the mucosal surfaces may have erosions and hemorrhages, which later become elevated, yellowish-white crusty plaques (Weingarten 1989; Shawky et al. 2000; Campagnolo et al. 2001; Sandhu & Shawky 2003; Konch et al. 2009). The lymphoid organs including spleen may look dark and mottled. The thymus becomes atrophied with multiple patechial and necrotic focal areas surrounded by clear yellow fluid that infiltrates and discolors sub-cutaneous tissues of the adjacent cervical region from the thoracic inlet to the upper-third of the neck. This lesion is important in meat inspection that can be detected easily when the opened neck of the carcass is observed on the processing line. During early infection, bursa of Fabricius is intensely reddened surrounded by clear yellow fluid that discolors adjacent tissue of the pelvic cavity. When the lumen of the bursa is opened, pin-point yellow areas and hemorrhagic surfaces are noticed. Later, walls of the bursa become thin as well as dark and get filled with white coagulated exudates. Even though these lesions are consistent with DEV infection, each age group responds characteristically. Lymphoid lesions are more prominent in ducklings than tissue hemorrhages. Outbreak with low virulent strain of DEV in white Peking ducklings (2 to 6 weeks

old) produced atypical gross lesions like diphtheritic membranes under the tongue, nasal and infra-orbital sinuses (Shawky et al. 2000; Konch et al. 2009). In mature birds with regressed bursa and thymus, hemorrhagic lesions in internal organs and reproductive tract are prominent. Gut-associated lymphoid tissues have multifocal necrosis and ulceration covered by fibrinous pseudo-membranes; (Sandhu & Shawky 2003; Guiping et al. 2007; Konch et al. 2009). In geese, intestinal lymphoid discs (Leibovitz 1969b; Proctor 1975; Weingarten 1989; Konch et al. 2009) are analogous to annular bands in ducks. In Canada goose, lesions of the intestinal lymphoid discs resembled 'button-like ulcers' (Leibovitz 1969a; Proctor 1975; Konch et al. 2009). Diphtheritic esophagitis is a consistent lesion in swans (Keymer & Gough 1986).

#### Histopathology

Histopathology reveals that the lesion commences from the walls of blood vessels. Smaller blood vessels, venules and capillaries are more affected than larger blood vessels. The endothelial lining is disrupted and connective tissue of the wall becomes less compact with visible breaks allowing blood to pass out to the surrounding tissues (Richter & Horzinek 1993; Sandhu

& Shawky 2003; Konch et al. 2009). Hemorrhages are more pronounced in inter-lobular venules of the proventriculus, venules in the spaces between lung parabronchi, hepatic and portal venules at the margins of liver lobules and capillaries within intestinal villi. Due to vascular damage, the affected tissues undergo degenerative changes. Microscopic findings include necrosis of epithelial lining of the digestive tract together with infiltration of variable lymphocyte and macrophage numbers within mucosal and serosal connective tissues. Eosinophilic intra-nuclear and cytoplasmic inclusions have been seen in epithelial cells of the digestive, respiratory and reproductive tracts as well as in visceral organs such as liver and spleen (Tantaswasdi et al. 1988; Shawky et al. 2000; Campagnolo et al. 2001; Konch et al. 2009). The affected epithelium becomes edematous, necrotic and rose into the lumen above normal adjacent mucosal surfaces. Degeneration and necrosis of stratified squamous epithelium of the esophagus and cloacae can also be observed. Parenchymatous organs like liver, pancreas and kidneys have hemorrhages and focal necrosis surrounding blood vessels. In the liver, hepatocytes become swollen with intra-nuclear inclusion bodies (Leibovitz 1971; Konch et al. 2009). Lymphocytes undergo karyorrhexis and pyknosis. In bursa, sub-mucosal and inter-follicular hemorrhages are observed coupled with depletion of lymphocytes. The epithelial cells of bursa are often hypertrophied with a vacuolated cytoplasm.

Similarly in thymus, free blood fills the inter-follicular spaces together with depletion of cortical lymphocytes. In female breeders, there is congestion and necrosis of the oviduct and the follicles become misshapen and stained with blood (**Konch et al. 2009**).

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