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Review Article

A COMPREHENSIVE REVIEW ON THE COMMON EMERGING VIRAL DISEASES AFFECTING DUCKS WITH SPECIAL EMPHASIS ON EGYPTIAN SITUATION

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Abstract: Poultry production is considered as a major source of livelihood for millions of people all over the world. In the last decades, domestic ducks have become increasingly important for meat and egg production. Ducks are susceptible to some specific important viral diseases. Duck viral hepatitis, and duck viral enteritis are diseases of significant importance with severe drastic losses for the world as well as the Egyptian duck industry. This review article aims to discuss these emerging duck viral diseases regarding epidemiology, diagnosis, prevention, and control with a special reference to the Egyptian situation.

Key words: control; duck viral hepatitis; duck viral enteritis; Egypt; incidence

Introduction

Water fowl including ducks are aquatic birds which are belonging to the order Anseriformes, family Anatinae, and species Anserine (1). Ducks such as Mallard (*Anas platyrhynchos*) and Muscovy (*Cairina moschata*) breeds are kept mainly for meat and egg production and in some instances for feathers and show. There are more than 35 breeds of wild non-domestic duck species that may be hunted. In the worldwide poultry industry, ducks are considered as the second most important avian species after chickens. Duck production has gained great attention due to its higher profitability compared with other species (2). Comparing ducks with other domestic birds as chickens, they are more adapted to different environmental and rearing conditions, require less management, show higher feed conversion ratio, rapidly growing and relatively resistant to the most common avian diseases (3). All of these advantages give this species of birds the priority in commercial poultry production.

In Egypt, the most common breeds of ducks are Muscovy, Pekin, Mallard (a hybrid of Muscovy and Pekin) (4), as well as other local breeds that adapted to Egyptian environmental conditions. Ducks are susceptible to some viral, bacterial, parasitic and mycotic diseases as well as nutritional disorders and myctoxicosis. However, the number of diseases that affecting ducks is much lower than those in chickens due to natural species genetic resistance. Duck viral hepatitis and duck viral enteritis are emerging viral diseases affecting ducks. These diseases induce severe economic losses like mortality, low growth rate, and increasing cost of prevention and control which badly reflect on Egyptian duck industry.

Therefore, the objective of this review article is to spotlight on some emerging viral diseases affecting ducks all over the world regarding their epidemiology, diagnosis, prevention, and control with a special reference to the Egyptian situation.

Duck viral hepatitis

Duck viral hepatitis (DVH) is an acute, highly contagious, rapidly spreading viral disease affecting ducklings with great loss (5). The disease is fetal and characterized by high morbidity and mortality rates as well as liver lesions (6). The World Organization for Animal Health (OIE) considered DVH as a notifiable disease (7).

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The virus

Duck hepatitis A virus (DHAV) is belonging to the Family *Picornaviridae* and the Genus *Avihepatovirus*es (8). The virus could be classified into 3 subtypes. The first isolated subtype of DHAV is type I (DHAV-I), which is original (classical), and the nucleotide sequences have been reported worldwide (9). Type II of DHAV (DHAV-II) has been isolated in Taiwan and its genomic sequence was first recorded in UK (10), while type III of DHAV (DHAV-III) has been isolated in South Korea and China (11, 12) with full genomic sequences. Types I and III of DHAV are caused by *Picornaviruses*, while type II is caused by an *Astrovirus*. Type I of DHAV is considered as the most widespread and virulent type (13). There is no antigenic relationship or cross-immunity among the different types (9). The virus is non-enveloped single-stranded RNA genome with approximately 7800 nucleotides in length (14). It remains in the environment for long periods due to its resistance to the most common disinfectants, acids, ether, chloroform, and trypsin (5).

Transmission and host range susceptibility of DHAV

Infection and transmission of DHAV occur through both the digestive and respiratory routes, but not through the vertical mean. Mechanical transmission especially via wild ducks was reported (6). Ducks are more susceptible to infection under 4 weeks of age but later on become resistant or chronic carriers. Mallard and Peking ducklings are more susceptible breeds to DVH infection, but now, the disease is identified in Muscovy ducks (15). Recent study of Ahmed *et al.* (16) revealed that goslings are susceptible to DHAV-I and may play a role in transmission of the virus to ducklings.

Incidence and distribution of DHAV

The first record of DVH disease was in Long Island, USA, in 1949, where DHAV was isolated from white Pekin ducklings with high mortality (17). At present, the disease becomes wide spread in many countries all over the world. For instance, in Poland, DVH was first described in the 1960s and 1970s, but the disease was not reported for many years (18). However, in 2014, DHAV-I was molecularly identified again from two flocks of 12-dys-old Pekin ducklings (19). Gough et al. (20) succeeded in isolation and identification of DHAV-II from ducklings in England. In China, DVH disease is prevalent (21, 22). No outbreaks of DVH has been recorded in Japan since 1963 (23), but DHAV was genetically identified as a causative agent of an acute disease in ducklings in 2015 (24). The epidemiological studies about the incidence and distribution of DHAV in Egypt are illustrated in Table (1) (25-37). For many years ago, strains of DHAV-1 were the predominant among duck flocks in Egypt. However, the recent genetic characterization and phylogenetic analysis of the circulating viruses revealed that strains of DHAV-3 are present in Egyptian duck flocks causing great losses and they are genetically distant from the used vaccine strain (36, 37).

Clinical Signs, necropsy and field diagnosis of DHAV

Infected ducklings with DHAV-I suffered from weakness, dullness, ataxia, laying on one side with spasmodic movement and kicking of legs, loss of balance and finally died in the opisthotonus position (5). The morbidity and mortality rates vary according to the age and breed of ducks as well as the subtype of DHAV. The mortality rate may reach 95% in ducklings less than 1-week-old, 50% or less in 1 to 3-weeks-old duckling, and low or unremarkable in 4 to 5-week-old ducklings (5). Dead ducklings with DVH had an enlarged liver with petechial and ecchymotic haemorrhages and enlarged spleen and kidneys (32). Pancreatitis and encephalitis have been also recorded in Muscovy ducks (15).

Laboratory diagnosis of DHAV

Laboratory diagnosis of DVH is based mainly on the isolation and identification of DHAV (38). Processed samples should be inoculated in the allantoic sac of 8-10-day-old embryos with monitoring for 5 days for stunting, subcutaneous hemorrhages, and greenish necrotic liver (7). Serological tests such as Agar gel diffusion assays (39), Enzyme Linked Immunosorbant Assay (ELISA) (40), Serum Neutralization (SN) test (41), and Indirect Hemagglutination (IHA) test (42) are used also for antibody detection against DHAV. Detection of DHAV using Polymerase Chain Reaction (PCR) and reverse transcriptase assay were reported (28-32, 35-37).

Table 1: The incidence and distribution of duck viral hepatitis, and duck plague in Egypt

Findings	References
Duck viral hepatitis (DVH)	
The virus causing DHA was identified for the first time in Pekin, Balady, and Rowan diseased ducklings in 1969.	Refaie (25)
In 1970s, DHAV was characterized.	Shalaby (26, 27)
From 2012-2014, DHAV-I was identified in 46 Egyptian commercial Pekin, Muscovy, Mallard, and Green Winged duck farms showing high mortality in 3-15-days-old ducklings.	Erfan *et al.* (28)
Type-I DHAV was detected in Blencher duckling flocks with 70% mortality in each batch.	Bayoumie and Abd El-Samie (29)
The molecular characterization and epidemiology of DHAV from different duck breeds have been studied.	El-Samadony et al. (30)
In 2017, DHAV-I was noticed in 10 out of 20 commercial duck flocks with different breeds and the sequence and phylogenetic analyses revealed that the Egyptian viral strains are distinguishable from the vaccine and similar to the contemporary Asian viruses.	Zanaty et al. (31)
During 2014-2016 in Sharkia governorate, DHAV-I was isolated and identified using conventional and molecular methods from 2-11-days-old duckling with nervous manifestations and high mortalities along with liver, spleen and kidney lesions.	Hassaan et al. (32)
From Sharkia governorate, concomitant infections with both highly pathogenic avian influenza a (H5N1) virus and DHAV-I in ducklings with mortalities and nervous signs.	Mansour et al. (33)
The molecular characterization of DHAV confirmed the circulation of DHAV-I (subclade B2) in Lower Egypt and elucidated the phylogenetic characters of the viral protein 1 gene.	Mansour et al. (34)
The pathogenicity of DHAV-1 isolates has been compared in Pekin and Muscovy duckling breeds in Egypt and the Middle East region for the first time. Samples have been collected from 3-21-day-old suspected commercial Pekin, Mulard, and Muscovy duck farms as well as backyards in Beheira, Alexandria, Gharbia, Kafr El-Sheikh, and Giza governorates, Egypt in a period from 2017-2019. They were subjected for conventional viral isolation and screening by PCR. Experimental infection of Pekin and Muscovy ducklings revealed similar typical specific clinic-pathological picture of DHAV-I and the histopathological changes supported the gross pathology in both breeds. The authors concluded that the genetic divergence between recent Egyptian strains and the used commercial vaccines urges evaluation of the vaccine efficacy and/or developing a new vaccine candidates.	Hisham et al. (35)
In Sharkia province of Egypt, isolation of DHAV-3 from 54 duckling flocks has been attempted. Positive DHAV was detected in 27.8% (15/54) and the genetic characterization of the strains revealed presence of 33.3% (5/15) contained DHAV-3 RNA. Moreover, those strains were displayed as a separate cluster and they were unrelated to the Egyptian DHAV-1-based vaccine.	Hassan et al. (36)
The genetic characterization of VP1 gene of 20 DHAV-positive farms representing 7- to 28-day-old commercial Pekin ducks in 6 Egyptian governorates during 2019 has been carried out. The results revealed detection of DHAV-3 in 18 samples as well as the classic DHAV-1 in 2 samples and the newly introduced strains were genetically distant from the used vaccine strain.	Yehia et al. (37)
Duck plague (DP)	
The first Egyptian outbreak of DP was in 1986 in Bahtem province where white Pekin duck flocks showed high morbidity and mortality rates.	Sabry et al. (94)
In 1990s, DP was recorded in breeder and broiler duck flocks with a mortality rates ranging 1-16% and 1-40%, respectively, as well as a drop in egg production 0.5%-99.5 %.	Sultan (95) Kheir El-Dine et al. (96)
The sequence analysis of glycoprotein enveloped genes of DP was analyzed and the results showed great similarity between the UL35 genes amplified from either local or imported vaccinal strains.	El-Mahdy et al. (97)
From 2012-2013, DEV was identified using conventional and molecular methods from various ages of Peking, Mallard, and Muscovy ducks in different Egyptian governorates.	El-Samadony et al. (98)
The Egyptian DEV that circulated in Pekin and Muscovy duck flocks in Dakahlia and Gharbia governorates were genetically characterized, and the results revealed that Egyptian strains have a close relationship with Indian and Chinese viruses.	El-Tholoth et al. (99)

Control and prophylaxis against DHAV

There is no effective commercial treatment or drug against DVH, but prevention of the disease is mainly based on vaccination (43). Ducks can survive after infection with solid long life immunity. However, it is very crucial to protect flocks from such devastating disease using effective specific vaccine. Immunization against DHAV infection is serotype specific without heterologous protection (11). Attenuated live DHAV vaccines are used for the prevention of DHAV-I infection in ducks (44). Vaccination of white Peking ducks with live DVH vaccine at 2-3-days-old followed by inactivated vaccine at 22-weeks-old induced significant higher antibody levels than that induced from one dose of live vaccine or 3 doses of inactivated one and ducklings from vaccinated breeders were resistant to challenge for up to 3 weeks old (45). Interestingly, Zou et al. (46) investigated that ducks received a single dose of duck enteritis virus recombinant (RC-KCE-2VP1) containing both viral protein 1 from DHAV-I and DHAV-III acquired efficient humoral and cellular immunity and were completely protected from both pathogenic DHAV-I and DHAV-III strains challenges.

In Egypt, live attenuated vaccine containing DHAVI is given intramuscularly to adult breeder duck flocks at 2-3 weeks before egg laying (47, 48). This type of vaccine provides offspring ducklings with maternal or passive immunity that protects them for up to 2 weeks post-hatching. Moreover, 2-days-old ducklings could receive live attenuated DHAVI vaccine and then boostered with another dose of the same vaccine at 2-3 weeks later. Although duck flocks are routinely vaccinated against DVH infection, but sometimes the vaccine couldn't produce effective protection and the infection may occur. It has been found that the vaccine DHAV strain was maintained and induced mortality in both vaccinated and non-vaccinated ducks (28, 29). This may be related to the antigenic differences between the vaccine virus strains and the field strains or due to the low titers of maternal-derived antibodies that cannot protect the offspring. It is important to note that DVH vaccination stoppage induced recurrence of severe aggressive disease outbreaks (49).

There is no available DHAV vaccine to control both DHAV-I and DHAV-III. Nevertheless, there is evidence that vaccination would be of benefit even at the start of any outbreak. Liao et al. (50) reported that emergency vaccination control measures against DHAV can effectively stop the spread of infection. Another study of Egyptian researchers proved that live attenuated DVH vaccines could be used as a tool to face outbreaks in order to control such infection (51). Moreover, Egyptian researcher produced a trivalent oil emulsion inactivated vaccine against *Salmonella typhimurium*, DVH and duck plague and the vaccine was safe, potent, and able to protect ducklings effectively against these infections (52).

In spite of the adoption of effective management procedures as well as an extensive usage of vaccine for DVH control, the infection is yet not prevented or controlled and the virus is still circulating in duck farms. As a result, discovering new antiviral or therapeutic agents targeting DVH is of great importance. In some Egyptian studies, Soufy et al. (53) and Okda et al. (54) found that inoculation of an immune potentiating agent like glycyrrhizin either alone or in combination with DHAV vaccine induced protection or amelioration of the disease severity through enhancement of erythrogram, leukogram, liver and kidney functions, and lymphocytic proliferation. It was demonstrated that the antioxidant falvon (icariin and phosphorylated derivatives) enhanced the survival of ducks and decreased the oxidative stress as well as liver dysfunction induced by DHAV-I (55). In addition, Raw Rehmannia Radix Polysaccharide was found to be an efficient new anti-peroxidase antivirus against DVH infection in terms of reduction of mortality and liver lesion score (56). Du et al. (57) indicated that flavone-polysaccharide herbs can be used as a new candidate for the treatment of DHAV because it is significantly decreased the mortality rate, hepatic damage and lesion score, and the virus gene expression level as well as returned hepatic function indices and peroxidation to the normal. Recently, Chen et al. (58) studied the effect of baicalin, a flavonoid derived from the Chinese medicinal herb, as antioxidant and immuno-enhancer anti-DHAV-I drug. The results of this investigation revealed that baicalin treatment reduced mortality and alleviated liver injury of infected duckling as well as interfered with the virus replication *in vitro*.

Duck viral enteritis

Duck viral enteritis (DVE) or duck plague (DP) is an acute and sometimes chronic highly contagious viral disease affecting different ages of domestic and wild water fowl (ducks, geese and swans) (59). The disease is characterized by high mortality especially in older age, internal hemorrhage, lymphoid organ atrophy, digestive mucosal eruptions, and degenerative lesions of the parenchymatous organs (60). The economic significance of DVE is related to mortality, condemnations, and decrease in egg production and hatchability (61).

The virus

The disease is caused by duck enteritis virus (DEV) which is *Anatid herpesvirus* type 1 (62). According to International Committee on Taxonomy of Viruses (ICTV) (63), DEV belongs to Family *Herpesviridae*, subfamily *Alpha-herpesvirinae,* and genus *Mardivirus*. The virus is an enveloped double-stranded DNA of approximately 180 kbp, non-hemagglutinating, sensitive to ether and chloroform, and inactivated by heat treatment (55). Although strains of DEV are differ in their virulence, but show similar immunogenicity (55).

Incidence and distribution of DVE

The epidemiological studies of DP revealed that from the 1920s till the 1940s, outbreaks of DP were first seen in Netherlands by Baudet (64) and Bos (65). In 1960s, great losses from DP were recorded among duck farms in USA (66), India (67), Belgium (68) and China (69). Later on, from 1970-1990s, the disease was reported in different countries like England (70), India (71), France (72), Pennsylvania (54, 73), Canada (74), Denmark (75), USA (76-78), Vietnam (79), and Bangladesh (80-82). Subsequently, several outbreaks of DP have been widely distributed all over the world (83-93). Table (1) shows the different researches that carried out in Egypt to demonstrate the incidence of DP in different flocks (94-99).

Infection and transmission of DVE

Water surfaces and free-living migratory waterfowl are considered as the primary sources for DEV infection (76). Domestic ducks usually contract the infection when they become in contact with free living water fowl (100). Recovered birds are usually chronic carriers and shed or transmit the virus to contaminate the surrounding environment (101). Horizontal transmission via ingestion or inhalation is considered the main route of DVE spread. Vertical transmission through egg shell contamination was also recorded in water fowl infected with DEV (102). Transmission can also occur through direct and indirect contact with infected birds or contaminated environments (103).

Diagnosis of DVE

Clinical signs related to DEV are mainly depending on the age, species, and immunity of birds as well as the virulence and dose of the infecting virus (104). Birds infected with DEV revealed general signs of depression and inappetence, greenish watery diarrhea, bluish discoloration of beak, and blood-stained vents (61). Ocular signs including conjunctivitis, lacrimation, diptheroid plaques around the eyelids, and photophobia were also seen. As a sequel, some birds were unable to drink and become dehydrated. Mortality rate ranges from 60%–90% has been recorded especially in adult breeders (105). Adult birds showed 25%–40% drop in egg production (61). Male birds may show prolapsed penis. Nervous manifestations appeared as drooping outstretched wings and head, ataxia, and tremors of the head and neck (60).

Macroscopic gross lesions induced by DEV are variable. Vascular damage and disseminated intravascular coagulopathy could be seen especially in older ducks as petechial or ecchymotic hemorrhages on the myocardium and epicardium giving a red 'paintbrush' appearance (100). Hemorrhages were also observed on the kidneys, lungs, intestine, and pancreas. Digestive tract lesions were usually observed in the buccal cavity, esophagus, caecum, rectum, and cloaca. Erosion with diphtheritic membrane formation was seen in the oral cavity, sublingual region and salivary glands ducts (106). Sloughing of the esophageal mucosa leaving yellowish-white diphtheritic membrane has been also observed. Hemorrhagic rings may be detected at the junction between the esophagus and the proventriculus. Hemorrhages and erosions can be seen on the intestinal mucosal surface and the lumen could be filled with bloody contents. Elevated yellow crusty plaques

with annular red bands or rings were also developed on the intestinal mucosa (61). Meckel's diverticulum may be hemorrhagic with a fibrinous core. The cloacal mucosa became red and then green plaque-like elevations. Shawky (107) showed that lymphoid and intestinal tissues are the main targets for DEV infection. The bursa of Fabricius became severely congested, hemorrhagic with white coagulated exudates, and surrounded by a yellowish fluid. The thymus glands showed atrophy, focal necrosis with a yellow fluid surrounding them (108). The spleen appeared dark and mottled. The color of the liver changed to copper like with pin-point hemorrhage and white foci giving a speckled appearance, after that the colour changed to dark bronze with large white foci. Extensive hemorrhages may be also seen on the ovarian follicles and in the abdominal cavity.

For isolation of DEV, samples should be taken from organs containing specific lesions like liver, spleen, kidneys, intestine, esophagus, bursa of Fabricius, thymus, and cloaca (104). Prepared specimens should be inoculated on the chorioallantoic membrane of 11-13-days old duck embryos (84) or propagated on cell lines as duck embryo fibroblast cells (109). For identification of DEV-specific genes, PCR, restriction fragment length polymorphism and nucleotide sequencing are used (110, 111). Seroconversions with SN, IHA, and ELISA tests have been used for detection of antibodies to DEV (112, 113, 93).

Prevention and control of DVE

Prevention and control of DVE are based mainly on the application of strict biosecurity measures as the virus can survive for several weeks under unfavorable conditions. Avoid direct contact between domestic and wild water fowl. In case of DVE outbreaks, hygienic disposal of dead birds, litter as well as any other physical instruments is a must to avoid mechanical spread of the virus (114). Proper chlorination of water and disinfestation of utensils with phenol or chlorine are important. Contact ducks with dead birds should be kept under quarantine measures for at least two weeks (61). During outbreaks of DVE, depopulation of clinically exposed ducks is necessary as the virus may become latent followed by environmental shedding. Therefore, it could be concluded that DVE outbreaks should be handled through the application of emergency measures such as vaccination along with slaughtering of infected ducks, hygienic disposal of dead carcasses, contaminated litter and equipment's as well as implementation of disinfection programs for pens, feeder and watering containers (115).

In enzootic areas in Europe and USA, both living and inactivated DVE vaccines are used for broiler and breeder ducks (116). Soma et al. (117) concluded that duck embryo cell culture technique produced higher concentrations of DEV in a shorter period than conventional old methods for vaccine propagation. It was reported that the response of ducklings to live DVE vaccine has interfered with maternal immunity and subsequent susceptibility to infection (117). Subcutaneous inoculation of ducks with living attenuated DVE vaccine was efficient and provided protective systemic and mucosal immunity when maintained at optimum physical and physiological conditions (105). In a recent Egyptian study by Abdullatif et al. (119), the authors revealed that the using a commercial live attenuated DVE vaccine ameliorated the severity and frequency of the clinic-pathological picture and decreased the mortality rate to zero in DVE infected duckling when given through the subcutaneous route. In addition, live DVE vaccine enhanced intestinal mucosal immunity by increasing the production of immunoglobulin (Ig) type A to prevent viral replication as well as inducing systemic protection by increasing serum IgY (120). However, there is a problem of latency regarding the use of this type of vaccine. Revaccination of ducks with living DVE vaccines is usually practiced due to viral excretion (121).

Therefore, inactivated DVE vaccine has been developed and used for inducing a protective immune response for several weeks (122-124). This inactivated vaccine could produce significant level of antibodies in ducklings 14 days post-vaccination. It has been reported that an oil emulsion inactivated combined vaccine against salmonellosis, DVH and DP was effective and protect ducklings from such diseases (52, 125).

Both live and inactivated DEV vaccines elicited active immunity (102). It was concluded that primary vaccination of ducks with living vaccine

should be done at 35 days of age using 0.5 ml/bird followed by a booster dose after 5 months of primary vaccination using 1.0 ml/bird for better immune response against DEV (126). Polyvalent living attenuated vaccine against both highly pathogenic avian influenza virus (AIV) subtype (H5N1) and DEV was developed with long protective immune response (127, 128).

Recombinant DEV vector vaccine considered as a good choice to reduce DEV outbreaks (129). Recently, Sun et al. (130) found that vectored vaccine containing recombinant DEV inserted with a gene of AIV subtypes (H5N1 and H9N2) elicited a strong protective immune response against both viruses by reducing viral excretion or shedding. Another recombinant vaccine for infectious bronchitis and DVE was produced with induction of significant humoral and cellular immune responses, improving antibody titer, decreasing viral shedding, and reducing mortality in chickens (131). Oral immunization of ducklings with living attenuated *Salmonella typhimurium* delivered DNA vaccine encoding DEV in addition to heat labile enterotoxin subunit of *Escherichia coli* as a mucosal adjuvant induced potent mucosal and humoral immune responses after viral challenge (132). Glycoproteins B and D based DNA vaccination of Peking ducks induced effective and forceful cell-mediated and systemic immunity against DEV (133). It was concluded that DNA vaccine has a significant impact on enhancing the immune response to DEV after immunization (134).

Other treatments rather than vaccines have been developed as novel methods to counteract DEV infection. Several studies have been conducted to treat DVE infected birds with herbs. When treated with an alcohol extract of neem seed kernel DVE-infected ducks revealed improvement in viability, diminishing expression of viral proteins, and decreasing in cytopathic effects (135). Phytoalexin plant has been also found to exhibit antiviral activity against DEV (136). Moreover, sulfated polysaccharide from *Chuanminshen violaceum* plant was potent against DEV through interfering with viral adsorption on the host cell (137). Types I and II goose interferon treatment could be used as potential preventive and therapeutic anti-viral approaches against DVE (137). Outbreaks of DVE have been treated efficiently with the homeopathic drug 'Mercurus corrosives-6 /12' at a dose of 5-10 ml/1000 ducks once or twice daily for 1-3 days (138).

Conclusion

From the abovementioned it could be concluded that duck is considered as an important susceptible host for essential specific diseases such as DVH and DVE which cause severe hazard and losses. Consequently, authorities should implement preventive and control measures for eradication of such diseases.

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