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REVIEW ARTICLE

Avipoxvirus in Egypt and African continent: A review

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Abstract

Fowl pox disease is a slow-spreading viral infection of wild and domesticated birds of both genders, all ages and breeds. The disease occurs in two distinct forms; the more common cutaneous or dry form and the less common diphtheritic form. Fowl poxvirus (FWPV) is a member of the Avipoxvirus (APV) and it is one of the greatest challenges facing the poultry industry, its incidence is higher in tropical and subtropical countries. It causes a significant level of morbidity and increased mortality, especially in the diphtheritic form which may reach to 50%. Avipoxvirus has been recorded in Egypt and Africa in the early of 1960, since then, it has been recorded in variable domesticated and wild bird species in different countries and Governorates. The free-living and wild birds represent a potential threat and source of infection for the domesticated poultry species. In the last ten years, the phylogenetic analysis of the partial genome sequences has gained insight into the evolutionary biology of APV in Africa. One of the main characters of APV is relative genetic stability, especially in *fpv167* region of the genome. This area of the genome clustered the APV of chicken and turkey origin phylogenetically into fowl poxvirus subclade A1 together with other avipoxviruses (APVs) isolated from *Galliformes* worldwide. However, the pigeon poxviruses phylogenetically belong to subclade A2 with other APVs isolated from *Columbiformes* worldwide. The analysis of the *fpv140* region provides a further comprehensive taxonomic classification based on the virus-host origin and distribution, especially in the case of the pigeon poxvirus (PGPV), which clustered separately into different subclades according to their geographical distribution. This review focus on the origin, distribution, classification and taxonomy of APVs circulating in Egypt mainly with a brief report on the situation of APVs in the other neighboring.

Keywords: Fowl pox, Avipoxvirus, Africa, *fpv140*.

Introduction

Avipox disease is a slow-spreading viral infection of higher incidence in tropical and subtropical countries [1-4]. Avian pox virus (APV) is amongst the largest and most complex viruses known to infect multiple avian species (canary (CNPV), fowl (FWPV), junco, mynah, pigeon (PGPV), psittacine, quail (QLPV), ostrich (OSPV), sparrow (SRPV), starling (SLPV), turkey (TKPV), crow, peacock, penguin (PEPV), alala, apapane and condor which are recognized within the APV genus within the *Chordopoxvirinae* subfamily of the family *Poxviridae* [5-7].

The genome of FWPV is composed of 260-365 kilo base pair (kbp), which contains a central coding region surrounded by identical 9.5kbp inverted terminal repeats and contains 260 open reading frames, of which 101 demonstrate similarity to genes of known function. Comparison of the APV genome with those of other chordopoxviruses (ChPVs) exposed 65 conserved gene homologues, encoding proteins involved in transcription and mRNA biogenesis, nucleotide metabolism, DNA replication and repair, protein processing, and virion structure. The genome of avipoxvirus includes six genes with putative

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protein modification functions. These genes involve three serine/threonine protein kinase (PK), one tyrosine PK, a metallo-protease and a tyrosine/serine protein phosphatase, these are involved in phosphorylation of virus proteins during virion assembly, viral protein processing and virion morphogenesis [8]. The APV elementary body is large, brick shaped and its genome is composed of single linear double-stranded DNA molecule measures about 260-365 kbp. Avipoxvirus consists of an electron-dense centrally located biconcave core or nucleotide and two lateral bodies in each concavity and surrounded by an envelope; the outer coat consists of random arrangement of surface tubules. APV replicates easily in the cytoplasm of infected avian epithelial cells, resulting in a characteristic cytopathic effect (CPE) and large acidophilic intracytoplasmic inclusions (Bollinger bodies) 4-6 days post infection depending on the virus isolate [9-11].

Clinically, fowl pox disease is a slow-spreading viral infection occurs mainly in two distinct forms; the most common cutaneous or dry form is characterized by the expansion of nodular proliferative skin lesion on multiple unfeathered parts of the skin (comb, wattle, eyelid, feet, cloaca aperture and under the wings). In young chicks, corner of mouth, nostril and eyelidsare mostly affected. Removal of the pox scale (local epithelial hyperplasia) resulted in bleeding. Firstly, the nodules seem as small, whitish foci which rapidly increase in size and become yellowish in color as they develop. In some cases, closely adjoining lesions may coalesce and the large developing lesions are rough and gray or dark brown in color. After about 2 weeks of development, the lesions may exhibit area of inflammation at their base and become hemorrhagic. The lesion then subjects a process of desiccation and scar formation which may last for another week or possibly two weeks in uncomplicated cases. The process ends with desquamation of the degenerated parts of the epithelial layer [1], and the less common wet form with diphtheritic and fibrinonecrotic lesions on mucous membranes of the mouth and oropharyngeal tract. The lesions on the mucous membranes are white, opaque with slightly elevated nodules. These lesions rapidly increase in size, often coalescing to become a yellowish, cheesy, necrotic material with the appearance of a diphtheritic membrane. Where these diphtheritic membranes are removed, they leave bleeding abrasions. The inflammation may extend from the mouth to the sinuses, especially the infra orbital sinuses, resulting in a tumor like swelling and may extend into the pharynx resulting in respiratory disturbance. Lesions in the mouth, tongue and esophagus intervene with the feeding and lesions of the trachea often result in the formation of tracheal plugs. In such cases, serious difficulty in respiration and suffocation may result. The mortality rate is higher in the wet form than in the cutaneous or dry form, sometimes reach 50% especially in young birds [12]. Mechanical transmission of the APV infection to the injured lacerated skin is considered one of the transmission routes. Insects act as mechanical vectors of the APV, resulting in ocular infection, and the virus may reach to the laryngeal region via the lacrimal duct to cause infection of the upper respiratory tract [13]. Mosquitoes are capable of infecting a number of distinct birds after a single feeding on a bird infected with APV. Eleven species of Diptera have been reported as vectors of APV as well as the mite "*Dermanyss gallinae*" has been implicated in the spread of fowl poxvirus [14]. Additionally, mechanical transmission of APV from infected toms to turkey hens via artificial insemination has been reported [15].

In this review, we emphasize our current knowledge and understanding regarding APV biology, evolution and distribution among different domesticated and wild bird species in Egypt, in particular, and other African countries.

Causative agent identification

Virology

Several trials were conducted in Egypt and African countries for isolation and propagation of APVs. For example, up to our knowledge, the first isolation trial of fowl poxvirus in Egypt was conducted in 1962 by El-Sabbagh. The isolation and propagation trial was applied through the inoculation into chorioallantoic

membrane (CAM) of 10-13 days-old embryonated chicken egg (ECE) producing typical pocks lesions [16]. Later on, in turkey species, the first isolation trial of poxvirus infection from a natural outbreak in turkey flocks was recorded during summer of 1988 in Egypt [17]. All the samples were isolated and propagated onto CAM of 11 days old ECE. The harvested infected CAMs showed typical pock lesions. In ostrich, the poxvirus was isolated from young domestic ostrich (*Struthiocamelus australis*) in South Africa from nodular cutaneous and diphtheritic oral lesions. Examination of the histological sections from the nodular cutaneous and diphtheritic oral lesions showed typical eosinophilic intracytoplasmic inclusion bodies [18].Due to the spreading and intensive breeding system of ostrich in the Egyptian field and appearance of several clinical cases suffered from skin lesions, the first isolation trial of ostrich poxvirus from an outbreak in Egyptian ostrich farms was reported in young birds (2-6 weeks old). The suspected samples were isolated on CAM of 10 days old ECE. Typical pock lesions were recorded after 6 days post the inoculation [19].

Variable studies were focused on the isolation and propagation of recent APVs specific for extraordinary bird species plentiful in the African wild nature. One example, APV isolated from African penguins was reported in 2009, which produced typical pock lesions as those induced by TKPV, OSPV and PGPV after ECE inoculation. On the other hand PEPV cannot be propagated successfully in chick embryo fibroblasts (CEFs) [20].The first Egyptian trial for description of the gross morphological characters and phenotypic characterization of the infected CAM was conducted with PGPV isolates by Metwally in 1994 [21]. Hence, isolation of PGPV from eighty clinically affected pigeons, were positive in 47 field samples from cutaneous lesions (n=24) feathers (n=8) *pseudolynchia canariensis* (n=6) mosquitoes (n= 5) and red mites (n=4) onto CAM of ECE resulted in large grayish white localized and or diffuse pock lesion, suggesting different phenotypic characters virus isolates. In 2011, Abdallah and Hassanin [22] studied the CAM morphology of six APVs isolates from Sharkia Governerate. The CAM morphology of the FWPV and TKPV isolates showed grayish-white discoloration, compact shape with marked thickening. While, the PGPV isolate showed yellowish discoloration, nodular shape, and moderate thickening of the CAM [22]. In another detailed growth characteristics comparison of elven APVs propagated into CAM of 10-11 days-old ECE from different bird species in South Africa was studied by Offerman and colleagues [3].They classified the APV isolates into six groups, based on pock and CAM morphology. Interestingly, there was no correlation between virus phenotype (pock morphology) or genotype and geographical distribution. Recently, a wide study on the isolation and morphological characterization of CAM was conducted on 136 bird flocks of different species, ages and breeding systems in Egypt. The successful propagation on CAM of ECE resulted in 130 positive samples with induction of pocks on the CAM with various morphological characteristics and variable degrees of membrane thickening. The isolated viruses revealed two distinct CAM phenotypes, the first one, is the classical pock shape, small to large in size, white to yellow in color and the second phenotype, is abundant white to yellow thickening of different sizes of CAMs [23].

Histopathology

Histopathological examination is a valuable diagnostic tool for diagnosis and detection of APV infection. Several studies based mainly on histopathology for the diagnosis of APV infection in different bird species. Those studies reported that the most characteristic features of infection, either the lesion is cutaneous, diphtheritic or from infected CAM, are hyperplasia of the epithelium and enlargement of cells, with associated inflammatory changes [3,22,23]. Abdallah and Hassanin [22] studied the histopathological changes of the cutaneous lesions of 6 APV clinical cases which revealed ballooning degeneration of the keratinocytes having large eosinophilic intracytoplasmic inclusions with central pale zone (Bollinger bodies). The six APVs replication were observed in the CAM comprehend of hyperplasia of the epithelium with cellular edema (hydropic degeneration) and Bollinger bodies [22]. Later on, Offerman

et al. [3] studied the histopathology of different APV isolates in CAM. The histopathological examination of these infected CAMs showed extensive mesodermal hyperplasia and less epidermal hyperplasia. All the infected CAMs revealed different degrees of hyperplasia and hypertrophy of both epidermal and mesodermal cells. Infected tissues showed ballooning degeneration of keratinocytes, necrosis and large eosinophilic intra-cytoplasmic inclusions (Bollinger bodies).Variable degrees of heterophil and lymphocyte infiltration were observed in the mesoderm and to a minor degree in the epidermis of the infected membranes. Some APVs isolates manifested marked immune infiltration, and angiogenesis in the mesoderm. Moreover, hyperplastic epithelial nests were noted in the mesoderm of some APV isolates. Angiogenesis and fibroplasia were noted in different degrees in most isolates [3]. Recently, the histopathology was utilized for the detection of APV infection in Egyptian aquatic geese, firstly observed in Egypt. Microscopically, the cutaneous lesions revealed hyperplasia of the epithelial cells of the stratum spinosum, with appearance of eosinophilic intracytoplasmic inclusions. Interestingly, the presence of avian poxvirus in the cytoplasm of hyperplasic infected epithelial cells was confirmed immunohistochemically using immunolflouroscent and immunoperoxidase techniques [24]. In a recent study in 2019, the histopathology of cutaneous lesions from different clinical cases and infected CAMs revealed that the epidermal hyperplasia may be covered with crust formation of superficial layers (necrotic and calcified) and the remaining layers (i.e. stratum spinosum) suffered ballooning degeneration and necrosis with eosinophilic intracytoplasmic inclusion bodies. Furthermore, the hyperplastic epidermal layer form downward epithelium growths in the dermis and subcutis resemble rete-ridges. The dermal area contains inflammatory cell aggregates, mainly consisting of lymphocytes and proliferation of the blood vessels within the skin eruption. Moreover, the histopathological examination of the CAM sections showed a hyperplastic ectodermal layer which suffered from focal destruction with high infiltration of inflammatory cells with the presence of elementary or eosinophilic intracytoplasmic inclusion bodies. Frequently, mesodermal reaction was seen with intense clusters of inflammatory cells around infected mesoderm epithelial nests [23].

Molecular biology (genes, clades and subclades)

Polymerase chain reaction (PCR) is one of the most recent, useful and delicate diagnostic techniques used for detection and characterization of APVs. Avipoxvirus genomic DNA sequences of different sizes can be amplified by PCR using specific primers designated previously [3,4,22,23]. This technique is helpful when a very small amount of virus is present in the sample and in case of mixed infections as fragments of different sizes could be amplified in single PCR reaction using pathogen-specific primers. Also, it can differentiate between the natural infection and the vaccinal strains. Few records of molecular characterization of APVs were recorded previously. The first characterization of PGPV was recorded in 2009 by Fahmy and his colleagues via using PCR and real-time PCR techniques from 8 clinical cases of suspected diseased pigeons. The study showed that there were five out of the eight suspected field samples positive for the presence of PGPV with expected size band of 578 bp from the *P4b* gene and the results were confirmed by real time PCR [25].In 2009, another gene locus (*fpv140*) was studied phylogenetically with the conserved (*fpv167*) gene in African penguins in south Africa. The PEPV was a unique species specific virus but it belongs to APV genus, especially, clade A, subclade A2 and mostly related closely to TKPV, OSPV and PGPV [20]. Later on, amplification of different APV specific genes and sequencing methods were conducted in Egypt and African countries especially in the recent years giving us more characteristic information about the molecular map concerning APVs characterization and classification (clades and subclades). For example, in South Africa, the *P4b* gene (*fpv167*) of the FGPVKD09 shown to be grouped phylogenetically in clade A, subclade A3 with high percentage similar to fowl pox-like viruses (clade A) rather than canarypox-like viruses (clade B) or psittacine-

like viruses (clade C). Interestingly, it was noticed to be 100% identical to falcon isolates FLPV1381 and black-browed Albatross (*Thalassarche melanophris*) ABPV [26]. In 2011, the first molecular record for APVs circulating in the Egyptian birds was recorded by Abdallah and Hassanin [22]. Six clinical cases from backyard reared birds were phylogenetically and biologically studied and recorded in the Genbank (Table 1).

Table 1: Orders, families and representative species of birds throughout Africa recorded with avian poxvirus infections.

No.	Order	Family	Species	Country	References	Accession number *P4b (FPV167)*
1	*Columbiformes* *Passeriformes*	*Columbidae* *Turdidae*	Cape turtle doves and Cape Thrush	South Africa	[41]	-
2	*Columbiformes*	*Columbidae*	Pigeon	Egypt	[36]	-
3	*Columbiformes*	*Columbidae*	Pigeon	Egypt	[37]	-
4	*Galliformes*	*Phasianidae*	Japanese quail	Egypt	[42]	-
5	*Galliformes*	*Phasianidae*	Turkey	Egypt	[21]	-
6	*Struthioniformes*	*Struthionidae*	Ostrich	South Africa	[22]	-
7	*Columbiformes*	*Columbidae*	Pigeon	Egypt	[25]	-
8	*Columbiformes* *Passeriformes*	*Columbidae* *Passeridae*	Pigeon, doves and house sparrows	Egypt	[43]	-
9	*Columbiformes* *Galliformes*	*Columbidae* *Phasianidae*	Pigeon, chicken and turkey	Egypt	[44]	-
10	*Galliformes*	*Phasianidae*	Turkey poults	Egypt	[45]	-
11	*Struthioniformes*	*Struthionidae*	Ostrich	Egypt	[23]	-
12	*Galliformes*	*Phasianidae*	Broiler chickens	Egypt	[1]	-
13	*Galliformes*	*Phasianidae*	Turkey	Egypt	[2]	-
14	*Struthioniformes*	*Struthionidae*	Ostrich chicks	Egypt	[46]	-
15	*Galliformes*	*Phasianidae*	Free range chickens	Nigeria	[3]	-
16	-	-	13 different species	South African	[5]	FJ948105 KC821556 GU204249 KC821559 KC821554 KC821552 KC821551 KC821553 KC821550 KC821558 KC821555 KC821557 KC821560
17	*Columbiformes* *Galliformes*	*Columbidae* *Phasianidae*	Pigeon, chicken and turkey	Egypt	[4]	JQ665838 JX464819 JX464820 JX464821 JQ665839 JQ665840
18	*Galliformes*	*Phasianidae*	laying chickens	Egypt	[32]	KF314718 (*TK* gene)
19	*Galliformes*	*Phasianidae*	Broilers and cockerels	Nigeria	[6]	-

Table 1: (Continued):

No.	Order	Family	Species	Country	References	Accession number *P4b (FPV167)*
20	*Sphenisciformes* *Columbiformes*	*Spheniscidae* *Columbidae*	Penguin and pigeon	South African	[31]	-
21	*Columbiformes*	*Columbidae*	Squabs	Egypt	[40]	-
22	*Anseriformes*	*Anatidae*	Egyptian goose	Egypt	[27]	-
23	-	-	Different species	Nigeria	[7]	-
24	*Galliformes*	*Phasianidae*	Chickens	Egypt	[33]	-
25	*Struthioniformes*	*Struthionidae*	Ostrich	Egypt	[8]	-
26	*Galliformes*	*Phasianidae*	Indigenous and commercial backyard chicken breads	Nigeria	[48]	-
27	*Galliformes*	*Phasianidae*	Chickens	Nigeria	[35]	KP987207 KP987214
28	*Galliformes*	*Phasianidae*	Chickens and turkeys	Mozambique	[34]	KX988302 KY312501 KY312503
29	*Galliformes*	*Phasianidae*	Chickens, turkeys, peacock and quail	Mozambique	[9]	MG787350 MG787405 MH061350 MH061352
30	*Sphenisciformes*	*Spheniscidae*	Penguin	South Africa	[24]	FJ948104
31	*Phoenicopteriformes*	*Phoenicopteridae*	Lesser Flamingos	South Africa	[29]	GU204249
32	*Columbiformes*	*Columbidae*	Juvenile rock pigeon	South Africa	[47]	-
33	*Galliformes*	*Phasianidae*	Chickens	Egypt	[20]	-
34	*Galliformes*	*Phasianidae*	Turkey poults	Nigeria	[38]	-
35	*Columbiformes*	*Columbidae*	Pigeon	Egypt	[28]	-
36	*Passeriformes*	*Fringillidae*	Canary	Egypt	[39]	1584198
37	*Galliformes* *Columbiformes*	*Phasianidae* *Columbidae*	Chicken, turkey and pigeon	Egypt	[26]	MH720302-MH720305 MH720299-MH720301 MH720306-MH720309
38	*Galliformes*	*Phasianidae*	Chicken	Tanzania	[30]	KF722858-KF722863

The analysis of *fpv167* (*P4b*) gene of the 6APV isolates, gathered 4 chicken and one turkey origin strains within subclade A1. Moreover, Sharkia_PGPV strain was grouped within subclade A2. Furthermore, when the *fpv140* gene was used for the phylogenetic analysis in the same study, Sharkia PGPV was grouped within subclade A4 (PGPV) with the other *Columbiformes* [22]. In 2013, the analysis of different loci *fpv26, fpv167, fpv140* and *fpv175–176* revealed various patterns of the classification and novel APVs (n=13), were isolated from several regions of South Africa. All the APV isolates in this study were clustered in clade A, subclades A2 and A3. [3]**.** In Tanzania, fowl pox was confirmed in 12 localities along the country. The phylogenetic analysis revealed that all Tanzanian isolates belonged to clade A, subclade A1 [27]**.**

The first full sequences of APVs in the African continent for two APVs from South Africa were published. The first one was for a Feral pigeon (*Columba livia*) (FeP2) and the second one for an African penguin (*Spheniscus demersus*) (PEPV). The FeP2 (282 kbp) and PEPV (306 kbp) genomes encode 271 and 284 open reading frames independently and were

closely related to one another (94.4%) than to either fowl pox virus (FWPV) (85.3% and 84.0%, independently) or Canarypoxvirus (CNPV) (62.0% and 63.4%, independently). This study revealed that the independent expansion of the South African APVs was from a common inherited virus to FWPV and CNPV [28]. Another sequence was obtained from laying chicken flock in Egypt and named as Ch-08Tk. Sequencing and phylogenetic analysis of 305 bp fragment from the thymidine kinase gene of isolated virus revealed that this virus has 95% identity with the other vaccine strains [29]. However, the limited published sequences of the thymidine kinase gene did not allow a proper phylogenetic comparison for this virus. Another outbreak of APV infection in 35 days-old broiler chicken flock was recorded in Sharkia Governorate, Egypt. The studied isolate was characterized as a fowl pox-like virus and named as Sharkia_APV_OMI. Phylogenetically, the analysis of *fpv167* (*P4b*) gene grouped Sharkia_APV_OMI within subclade Al with 100% identity with the other APV strains from Egyptian backyard system. The previous indicates the similarity of the circulating APVs in both commercial and backyard systems proposing that the backyard birds can act as a source of infection [30].In Mozambique, from August 2015 to November 2016, amplification of another specific gene (DNA polymerase) in addition to *fpv167* was conducted. Amplification of the p4b (*fpv167*) and DNA polymerase genes resulting in clustering of APV strains isolated from 16 separate FWPV outbreaks in clade E [31]. In West and Central Africa, the first available fowl poxvirus sequences (KP987207-KP987214) were obtained by Meseko and colleagues. Nucleotide analysis of these isolates and a vaccine strain showed 100% similarity and also shared 72 – 100% homology with selected sequences from the GenBank, while clustering on the phylogenetic tree was in clade A, subclade A1 [32]. Later on, in Mozambique, from 2016-2018, the amplification of fpv167 and DNA polymerase genes resulting in identification of clade A2 with the confirmation of the circulation of clade A1. Phylogenetically, the sequencing analysis revealed that the 49 APVs strains were clustered in both clade A1 and clade A2. Furthermore, by PCR, all of the clade A1 viruses were positive for the integration of reticuloendotheliosis virus (REV), moreover, the clade A2 APV samples were negative [4]. Recently, the amplification of three gene loci p4b, *fpv140* locus and DNA polymerase gene for different APV isolates was conducted by Lebdah and his colleagues, which were characterized as fowl poxviruses. The phylogenetic analysis of twelve APV isolates from chickens, turkeys and pigeons (n= 4, each) revealed that all the sequenced strains clustered in clade A. Based on full sequence of fpv140 locus, the phylogenetic analysis revealed that the four chicken strains and the turkey strain (Egypt_FWPV_TK3) were grouped in subclade A1.a (FWPV). The turkey isolates, Egypt_FWPV_TK1, TK2 and TK4, were divergent sequences and were branched into the novel subclade A1.b. The 4 PGPV strains were clustered together into subclade A2.c [23].

Epidemiology and transmission

The transmission and epidemiology of APV infection are considered from the important items which carefully studied and discussed either in Egypt or in the other African countries due to their dangerous role in incidence and prevalence of the disease. For example. El-Dahaby and his colleagues studied the role of some wild birds (sparrows, doves and migratory quails) in the transmission of avian pox. This study revealed that sparrows and doves can transmit pigeon pox infection via wounded feather follicles only by contact with infected birds. The isolated PGPV from migratory quails increased the danger of its role in the spread and epizootiology of the disease during autumn and winter seasons all over the country [33].Thereafter, several studies on APV biology, transmission and epidemiology were conducted on many wild, migratory, free living and domesticated birds. In this century, many seroepidemiological studies had documented the spread of fowl pox in the Egyptian commercial poultry flocks. One example, an earlier study clearly showed that pigeons may be a potential reservoir or infection source to chickens with avian poxviruses, especially in areas where pigeons and chickens are reared together. The study

was conducted on sixty eight isolates harvested from pigeons suffered from pox-like disease which were identified as positive biologically and serologically via the agar gel diffusion test [34]. Later on, the role of mosquitoes and red mites in the transmission of the virus was investigated via studying the epidemiology of poxvirus in pigeons. The results showed APV positive isolation in *pseudolynchia canariensis* (60%), mosquitoes (50%) and red mites (40%), which indicates the danger of mosquitoes and red mites in the transmission of the PGPV [21]. Several studies especially in Nigeria were performed on the role of insects in transmission of APVs. For example, a study on an outbreak of APV infecting 8 weeks old turkeys. The outbreak extended to 5 weeks with 100% morbidity and no mortalities. In turn, they concluded that the mosquitoes and or other biting arthropods were the main source of infection to the turkeys [35]. Interestingly, another study investigated the role of insect vector and mosquitoes in transmission of APV among the bird species in Nigeria via studying the seroprevalence of fowl pox antibody in free range chickens. The study revealed high prevalence of avian pox disease in indigenous breed poultry (73.5%) as compared to in exotic breeds (26.5%). This result may be due to the fact that scavenging indigenous or local breeds have the most probable chances of bite from the insect vectors such as mosquitoes. Moreover, indigenous poultry are not mostly housed during the nights in most rural settlements [2].the fore mentioned studies confirmed the role of migratory and free living birds in the transmission of different APVs into domesticated birds. In 2012, a study was performed on canary suffering from pox- like lesions. The sequence analysis revealed 100% identity of the obtained strain to FWPV but divergence with CNPV with high base substitution rate per site (0.326). They established the ability of FWPV to infect canaries and recommended to vaccinate canaries with fowl pox vaccine [36].

Two years later**,** fowl pox was investigated in chickens in recurring outbreaks of fowl pox in a poultry farm in Nigeria with the abilities to persist in the environment causing recurring outbreaks in unprotected chickens. It can lead to significant economic losses due to mortalities associated with the severe forms of the disease. [12]**.** One year later, a comparative study between growing and adult chickens and also between chicken and turkey species was conducted via studying the prevalence of avian pox disease in relation to other poultry diseases. The prevalence rate was high among the younger and growing birds as compared to adults. The disease occurred more in turkeys and chickens as well as other domesticated birds especially the indigenous breeds. The study investigated that the occurrence of fowl pox disease in turkeys (56.3%) was higher than in chickens (31.2%). This results may be due to the fact that the disease is more severe in birds with large comb and wattle which is attributed to both turkey and chickens [37].A hematological study in Egypt was performed on a total of twenty squabs (2-4 weeks old), which were naturally infected with pigeon pox. Erythrogram results revealed a significant decrease in the count of erythrocyte, packed cell volume (PCV) and the concentration of hemoglobin with the development of macrocytic hypochromic anemia. This study concluded that PGPV infection causes anemia and disturbances in liver and renal functions [38].

Susceptible hosts, either domestic or free living birds

Avipoxvirus infections was recorded in 278 bird species from 70 families and 20 orders, these avian species either wild, free living or domesticated, were susceptible to one or more of the APV strains. Several bird species were investigated for APV infection. For example, the first record of APV infection in Africa in the early 1961's was in South Africa when APV infection was recorded in Cape turtle doves (*Streptopelia capicola*) and a Cape thrush (*Turdus olivaceus*) [39]. Thereafter, another host species was examined by *Amer et al.* [40]. They studied a novel strain of APV group, which was recovered from a flock of Japanese quail by agar gel precipitation test and differentiated from both fowl and pigeon poxviruses by neutralization test. The isolated virus was of high pathogenicity with the development of generalized lesions for quails experimentally infected through intravenous, wing web or

feather follicle routes of inoculation. In Upper Egypt, a study on the occurrence of poxvirus infection in three species of exotic birds including pigeons, house sparrows (*Passere domesticus*) and doves was conducted. Isolated poxvirus was identified physicochemically, biologically (host spectrum) and serologically. Isolates from pigeons and doves were characterized as pigeon poxvirus, while the sparrows` isolates were characterized as fowl poxvirus [41].

Later on, in Sharkia Governorate, Egypt, the APV infection was recorded in many species of domestic birds; chickens (n=6), turkeys (n=2) and pigeons (n=2), indicating the widespread of the virus in different domesticated birds in this locality [42]. Another experimental study in Egypt was performed on 25, 4-weeks old, turkey poults which inoculated by chicken originated poxvirus at the dose of 3x107.6/mL. The previous study concluded that the inoculated chicken originated poxvirus is highly pathogenic for turkeys [43]. In 2005, APV infection was diagnosed for the first time in broiler flock of 40 days-old. The most characteristic observation of this outbreak was that the pox signs and lesions were observed in the unusual feathered parts of the bird. These untypical lesions led to high condemnation rate and high mortality which reached 25% [1]. A more comprehensive study on the host specificity of turkey poxvirus to some of avian species was conducted in Egypt during natural outbreak of pox disease. The results revealed a high degree of heterogeneity among the avian species, where the turkey poxvirus had the highest lesions in turkeys, then chickens, pigeons and ducks, respectively [44].The poxvirus was recorded in an extra ordinary bird species in South Africa, Lesser Flamingos on a purpose-built island, at Kamfers Dam near Kimberley. The *P4b* gene of the FGPVKD09 shown to be grouped phylogenetically in clade A, subclade A3 with high percentage similar to fowl pox-like viruses (clade A) rather than canarypox-like viruses (clade B) or psittacine-like viruses (clade C). Interestingly, it was noticed to be 100% identical to falcon isolates FLPV1381 and Black-browed Albatross (*Thalassarche melanophris*) ABPV, independently, suggesting the cross infection between different species [26]**.** In Egypt, another study characterized ostrich poxvirus in three ostrich flocks, 18 to 62 days old, via studying their physical and some biological properties. The results revealed that the isolated ostrich poxvirus had similar antigenic, physical and biological properties of fowl poxvirus [45]. Later on, several studies on other wild and free living bird species were investigated. One example, pox-like lesions were detected in a wild juvenile rock pigeon (*Columba guinea*), the infected wild juvenile rock pigeon was subjected for necropsy. The necropsy showed tumor-like lesions of yellowish nodular cutaneous coalescing masses mainly on the unfeathered parts on the head of the infected dead pigeons as well as the beak. Electron microscopy confirmed the detection of APV in the infected CAM with the pock lesions [46]**.** There are many extensive studies on APV infection in domestic birds in the different African countries. Furthermore, APV infection was recorded in ostrich in African countries several times. Interestingly, in South Africa, the first detection of poxvirus in young domestic ostrich (*Struthiocamelus australis*) was performed. Ostrich poxviruswas isolated and confirmed by histopathology for presence of intracytoplasmic inclusion bodies[18]. Interestingly, the first report of APV infection in geese was recorded in Egypt by Ali and his colleagues. The study described the pathological and immunohistochemical changes in the skin of naturally infected goose with APV infection. [24]**.** Another study was performed in Nigeria through 5-years period, during 2011 to 2015, using 39 mixed sex backyard, commercial and indigenous chickens of different breeds and multiple ages. The study concluded that all breeding systems (indigenous and commercial) and breeds (pullet, broiler, cockerel and layers) of chicken and all ages from 9-52 weeks old chickens were susceptible to the infection with cutaneous form of the disease. Young (9 weeks old) cockerel chickens were susceptible to the infection with both systemic and cutaneous form of fowl pox [47]. In 2016, ostrich poxvirus was detected in 4–9-week-old ostrich chicks from 11 ostriches farms at Sharkia, Kalubia and Ismailia Governorates. The ostrich chicks suffered from weakness,

dullness, emaciation, depression, anorexia with skin lesions and wet form. This study concluded that ostrich poxvirus can infect 6 week-old turkey poults with high pathogenicity to dry and wet lesions [48].In 2019, APV infection was investigated from 136 flocks of different avian species (pigeon, chicken and turkey), ages and breeding systems by Lebdah and his colleagues. Pigeons of young ages less than 30 days old were more susceptible to the infection than other ages. However, higher susceptibilities were recorded in chickens of 40-60 days and turkeys of 60-90 days. Furthermore, the commercially reared chicken and turkey species were more susceptible to APV due to the intensive breeding and presence of newly introduced strains especially for TKPV [23].

Economic losses of APV infection

Avian pox virus infection is still a malady and enzootic to the growing chickens of both genders, multiple ages and breeds in Africa. Avipoxvirus infection was investigated in more than 200 avian species [22,37,40], either in commercial poultry farming or in backyard system [1,23,45]. The disease causes significant economic losses to the poultry industry, high morbidity (10-95%), with usually low to moderate mortality (0-50%). The mortality mainly is due to diphtheritic form which causes death by asphyxiation or due to secondary complication or blindness and starvation [1,4,12]. Fowl pox causes emaciation, poor growth, poor feed conversion, and increase meat condemnation, difficulty in swallowing and breathing and decline in egg production in laying hens [12,37].

Host virus interaction

Similar to many other DNA viruses, APV can elude host immune responses through allocating much of its genes. Membrane fusion, penetration and intracellular transport of the APV occur through molecular transformation via the viral genes which commonly encode proteins. The genes involve those encodes proteins which act upon early innate pathways like pathways requiring interferon [49], pattern recognition receptors as Toll-like receptor (TLR) [50], chemokines [51,] cytokines [52], in addition to pathways which act upon ensuing adaptive responses [53]. A process in which the virus infect a cell called a complex process, at which the virus must get control of various host factors restriction tips and host immune response. Biochemical pathways and host protein interaction nexus are in most instances changed by the viral proteins that liberate the virus from normal cellular controls and allow nucleotide metabolism in cells that end the DNA synthesis [54]. Through one hour, from the APV entrance into the avian host epithelium, the virus penetrates cell membranes and then the process of uncoating occurred, then synthesis of a new virus progeny from precursor starts [55]. Biosynthesis includes two definite stages, which occurred in the host dermal epithelium. The first stage is host response occurred through the first 72 hours, accompanied by the second one at which the synthesis of infectious virus from 72 to 96 hours [56]. Synthesis of host DNA is followed by epithelial hyperplasia, from 36 to 48 hours, with host DNA reducing suddenly at 60 hours. Some studies conducted that the viral DNA replication in the avian host starts between 12 to 24 hours, accompanied by an expanding rate of synthesis from 60 to 72 hours. At 72 hours, hyperplasia finishes with a 2.5-fold enlarge in cell count [57-59]. At 100 hours, the proportion of viral to host DNA grows up to 2:1 with the highest titer of virus achieve ensuing cell proliferation. Furthermore, through virus morphogenesis, insufficient, imperfect, or refining forms in transition stages, which leads to mature forms or virions. The second stage composed of a long latent period, with parts of viroplasm within the cytoplasm surrounded by incomplete membranes. An extra outer membrane is obtained by the viroplasmic molecules to form incomplete virions. The virions roam to vacuoles of the inclusion bodies and then obtain a membrane coat [60]. The virus after that come out from the cells by a budding process, resulting in an extra outer membrane that is acquired from the cell membrane. The classical inclusion body (Bollinger body) is produced by this process which noticed by light microscopy [61].

Conclusion

In Egypt and Africa, Avipoxvirus is still circulating in backyard and commercial management systems. The free living and wild birds represent a potential threat and source of infection for the domesticated poultry species. The molecular analysis provide us with useful information regarding the evolution and taxonomy of APV distributed in Africa.

Conflict of interest

The authors have no conflict of interest to declare.

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الملخص العربي

فيروس جدرى الطيور في مصر والقارة الأفريقية: مقال

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جدري الطيور هو مرض فيروسي بطيء الانتشار لكل من الطيور البرية والمنزلية من كلا الجنسين ، من جميع الأعمار والسلالات. يحدث بشكل أساسي في شكلين مختلفين: شكل الجلد الأكثر شيوعًا وشكل الدفتيريا الأقل شيوعًا. ينتمي فيروس جدرى الطيور إلى جنس فيروس جدرى الطيور. جدرى الطيور واحد من أكبر التحديات التي تواجه صناعة الدواجن، ومعدلات الإصابة بهذا الفيروس أعلى في البلدان الحارة المدارية وشبه المدارية. حيث أنه له دور فعال في زيادة ظهور الصورة الممرضة ومعدل الوفيات، خاصةظهوره في شكل الدفتيريا والذي قد تصل إلى ٥٠-١٠٠ ٪. تم تسجيل فيروس جدرى الطيور في مصر وأفريقيا في أوائل عام ١٩٦٠م ، ومنذ ذلك الحين، تم تسجيله في أنواع الطيور المنزلية والبرية في مختلف البلدان والمحافظات. تمثل الطيور الحية الحرة والطيور البرية تهديدًا محتملاً ومصدرًا للإصابة للدواجن المنزلية. في السنوات العشر الأخيرة ، اكتسب تحليل التطور الوراثي لتسلسل الجينوم الجزئي نظرة ثاقبة في البيولوجيا التطورية لفيروس جدرى الطيور في أفريقيا. واحدة من السمات الرئيسية لفيروس جدرى الطيور هي الاستقرار الجيني النسبي ، وخاصة في منطقة *fpv167* من الجينوم. تجمع هذه المنطقة من الجينوم فيروس جدرى الطيور من الدجاج و الرومي إلى فئة فرعية لفيروس جدري الطيور (A1) مع فيروسات أخرى من فيروس جدرى الطيور معزولة من *Galliformes* في جميع أنحاء العالم. ومع ذلك ، تنتمي فيروسات الجدري المعزولة من الحمام إلى فئة فرعية (A2) مع فيروسات جدرى الطيورالأخرى المعزولة من *columbiformes* في جميع أنحاء العالم. يوفر تحليل منطقة *fpv140* تصنيفًا شاملاً إضافيًا يعتمد على مجموعة المنشأ والتوزيع ، خاصةً في حالة فيروس جدري الحمام الذي يتجمع بشكل منفصل في مجموعات فرعية مختلفة وفقًا لتوزيعه الجغرافي. تركز هذه المراجعة على منشأ وتوزيع وتصنيف فيروسات جدرى الطيور المنتشرة في مصر بشكل أساسي مع تقرير موجز عن حالة فيروسات جدرى الطيور في البلدان الأفريقية المجاورة الأخرى.