

# CEVA HANDBOOK OF POULTRY DISEASES

## 9 FOWL POX

p/171



## FOWL POX

### DEFINITION

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Fowl Pox (FP) is a viral disease found in hens, turkeys and many other birds, characterised by cutaneous lesions on the featherless skin and/or diphtheritic lesions of mucous coats in the upper alimentary and respiratory tract. FP is encountered in either cutaneous or diphtheritic form or in both, or a combination of both.

## HISTORY AND SYNONYMS

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FP is one of the earliest described avian diseases, due its characteristic, easily-identifiable external lesions (Heusinger, 1844). Subsequent histological examinations for confirmation of tentative diagnoses have demonstrated the relationship between histological lesions and the structure of inclusion bodies (Bollinger, 1873; Borrel, 1904).

Convincing evidence associating inclusion bodies and poxvirus as etiological agents was provided by Woodruff & Goodpasture (1930).

In the mid-20<sup>th</sup> century, research focused on identifying the virus through preparation of an appropriate culture – ectoderm chorioallantoic membrane of embryonated chicken eggs (Cunningham 1966).

This technique is still a commonly-used method of identification. Electron microscopy development also provided a reliable diagnostic tool. The prevalence of avian poxvirus among wild birds was later reviewed (Bolte et al., 1999).

Nowadays, avianpox virus strains

are identified by molecular methods as gel-electrophoresis and PCR (polymerase chain reaction) analyses of mitochondrial DNA sequences (Schnitzlein et al. 1988). In recent times, avian poxvirus strains have been used extensively in the production of recombinant vaccines.

Various synonyms of the disease have emerge as this research has advances, and are still used in different regions worldwide: avian pox, bird pox, pox, poxvirus infection, avian diphtheria, contagious epithelioma, molluscum contagiosum, Gefluegelpocken (German), viruela aviar (Spanish), variole aviaire (French), boubas (Portuguese),

The FP virus is the prototype member of the genus *Avipox virus*. It infects chickens and turkeys. The term fowl pox initially comprised all avian poxvirus infections, but more recently has been used to designate disease in commercial bird species only.



## CHARACTERISTICS AND CLASSIFICATION OF THE PATHOGEN

FP is caused by an epitheliotropic DNA virus from the *Avipox* genus, the Poxviridae family. Several virus types (strains) exist: fowl pox virus, turkey pox virus, pigeon pox virus, canary pox virus etc., and differ pathogenically and immunogenically. Poxviruses are epitheliotropic, so the infections are characterised by transient epithelial hyperplasia, inflammation and necrosis. These viruses are very resistant to environmental factors and may persist for several months. The virions are inactivated at 60°C for 8 min. They are sensitive to chloramine, 2% sodium hydroxide, 0.01% formalin and 1% phenol.

It is generally believed that avian poxviruses are strictly host-specific, although some investigations have demonstrated that certain strains may be transmitted over species or even family frontiers (Adams et al., 2005; Jarmin et al., 2006). This is mainly a result of taxonomic considerations, as the classification of avian poxviruses is based on the host species from which the virus was originally recovered. A cutaneous form of poxvirus infection has, for example, been reported on a turkey farm. On the

basis of 100% identity of sequences of isolates, it was assumed that the probable source of infection was avian poxvirus-infected commercial layers in the vicinity. Furthermore, there was an exchange of workers and equipment among the farms, facilitating the spread of infections, as well as a high prevalence of fleas and mosquitoes, acknowledged vectors of poxviruses (Hess et al., 2011). FP virus is pathogenic for turkeys, while the pigeon poxvirus can affect crows but not chickens and turkeys (Zarkov, 2003).

FP is among the largest poxviruses, with dimensions of 330x280x200 nm. Broken skin serves as entry door for viruses. Once in the bloodstream, they provoke a primary viraemia, replicating in haematopoietic organs and concentrating in epithelial cells of the skin and mucous coats. A secondary contamination with bacterial microflora (*Staphylococcus spp.* in particular) is not uncommon. Skin damage may, however, progress to generalised infection incurring economic losses from increased mortality, weight loss and slaughterhouse culling (Hess et al., 2011). Virus-neutralising antibodies

have been demonstrated in FP viruses. Viruses infecting chickens, turkeys and pigeons are antigenically related. Canary and pigeon pox viruses are immunologically independent. Only members of genus *Avipoxvirus* from the subfamily Chordopoxvirinae (poxviruses of vertebrates) of the Poxviridae family infect non-mammalian hosts. Avian poxviruses are cytoplasmic DNA viruses. The variability in restriction enzyme profiles of viral DNA implies substantial differences in the family members' genomes. Cross infection research also suggests genetic differences among the viruses, reflected in the pathogenetic effects produced (lack of clinical disease, local pox lesions, local and generalised infections, generalised infection with fatal outcome) and the lack of cross-protection depending on the specific virus-host combination (Alfonso et al., 2000).

The FP virus genome contains 260 to 309 kbp double-stranded DNA. Replication occurs only in the cytoplasm, and takes in several stages. The first stage is attachment, penetration and deproteinisation. After attachment

to sensitive cells, the viruses penetrate the cytoplasm by fusion and unwrap. The DNA release occurs in two stages. Cell and viral enzyme (DNA/RNA polymerase)-mediated iRNA synthesis on a genomic region takes place. Early enzymes participating in genome unwrapping, DNA replication and synthesis of transcription components are produced. Transcription occurs during the subsequent stage. Middle and late iRNA are formed inside the cell. The middle iRNA is synthesized on maternal DNA and supports late RNA formation. Late RNA synthesis begins after DNA replication.

Next follows translation –synthesis of viral DNA, virus-specific enzymes and structural proteins. The process continues with the DNA replication stage, which requires mainly DNA/RNA polymerase. New viral DNA is produced. During the final stage, assembly and release of virions takes place in the cytoplasm. Mature infective virions leave the destroyed cell (Zarkov, 2003).

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## EPIDEMIOLOGY

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Infection is spread mechanically by dissemination of the virus through desquamation of infected crusts. Some mosquitoes and blood-sucking arthropods may also help disseminate the virus. Mosquitoes can remain infective for several weeks. The incubation period lasts from 4 to 10 days. The disease is spread slowly, and many weeks could pass between its first emergence and the appearance of a severe outbreak.

Geographically, avian poxviruses are omnipresent. They are more prevalent among bird populations in warm regions of the world. Even within a continent, these viruses tend to be concentrated in specific regions with a warm, humid climate. The disease may be encountered all year round and is often associated with increased humidity, deficiency states, moulting etc.

Most avian species are susceptible to one or several avian poxvirus strains. A report has demonstrated the susceptibility of 278 bird species from 70 families and 20 orders to the virus. It also states that avian pox has never been reported in tinamous (tinamiformes), loons (gaviiformes), nightjars (caprimulgiformes), or kingfishers (coraciiformes). Among commercial species, natural infection outbreaks are most commonly encountered

in hens, turkeys, pheasants, guinea fowl, pigeons. In identical conditions, young birds are more sensitive to the virus than adults.

A number of biotic and abiotic factors have been linked to the incidence and prevalence of avian pox – density of host population, interacting viral loads, vector counts etc. These factors determine to a large extent the character and the course of avian pox outbreaks. Diseased birds are the source of infection, which may spread via contact during transfer of personnel, equipment, feed etc. Blood-sucking insects as mosquitoes, flies, fleas or ticks (*Argas persicus*, *Dermanysus gallinae*) are possible disease vectors. The virus may survive for up to 7 months in mosquitoes. Among wild birds, sparrows are a reservoir of avian pox. They are able to spread the virus to farms located far away from the original outbreak. Entrance routes for infection include scratches on the skin, comb and wattles, and the mucosa of the mouth, oesophagus and skin follicles after feathers are plucked. Feather pecking (cannibalism) may also be a route of transmission of infection (Obreshkov et al., 1978).

## CLINICAL SIGNS AND PATHOLOGY

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The incubation period of natural infection is 4–10 days in chickens, turkeys and pigeons. The morbidity rate varies from several ill birds to infection of the entire flock when the virus is highly virulent and no prevention measures are taken. Birds affected by the cutaneous form are more likely to recover than those suffering from the diphtheritic form. The usual course of the mild cutaneous form is 3–4 weeks, but can be longer if complications arise. In affected breeder flocks, a drop in egg production and fertility are observed. The mortality in infected chickens and turkey flocks is usually low, but may increase in case of complications (Tripathy & Reed, 2008).

The cutaneous form is characterised by nodular lesions of the comb, wattles, eyelids, but lesions of oviduct and cloacal mucosae may also appear (Metz et al., 1985). The lesions represent crusted epithelial proliferations, red-brown in colour, with a wart-like growth, irregular surface and borders. Most lesions join together. Sometimes, single lesions may be located on feet, the beak or the eyelids. The size of single lesions varies from 2 mm to 2 cm. The emergence of multiple lesions on eyelids could be fatal, as reported in pheasant, quails and turkeys. The lesions interfere with

the eyesight of birds and prevent them from finding food (Forrester & Spalding, 2003).

Histologically, the cutaneous form of FP is characterised by marked hyperplasia and hypertrophy of the epithelium. The cytoplasm of pox-affected keratinocytes contains eosinophilic inclusion bodies (Bollinger bodies) with centrally-located vacuoles, which do not stain and which are presumed to be of dissolved lipid origin. An accompanying inflammatory reaction from heterophilic granulocytes or coccoid bacteria colonisation should also be detected.

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**Fig.1**

In most outbreaks, the cutaneous form is prevailing. The lesions vary according to the stage of development: papules, vesicles, pustules or crusts. The lesions are usually in the region of the head.



**Fig.2**

FP is common among pigeons Papules and crusts on the eyelids.



**Fig.3**  
Skin lesions in the cloacal area in a pigeon.



**Fig.4**  
FP lesions on the skin of the legs.

The diphtheritic form is distinguished with a typical stomatitis, with grey-whitish deposits on mucous coats of the oral cavity, the pharynx, the larynx, the upper parts of the trachea and the

oesophagus, the crop and the eyes. When the larynx and the trachea are affected, dyspnea and crackles are present. The severe stomatitis could cause disturbed feeding, emaciation and death.

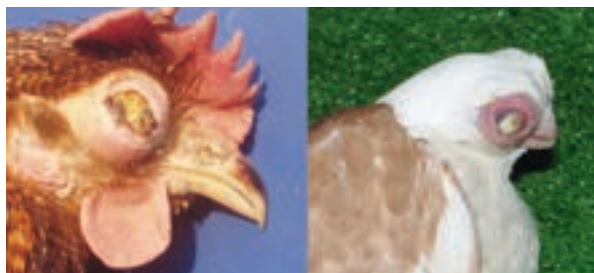


**Fig.5**

Diphtheritic lesions look like whitish or yellowish plaques that are deposited and grown on the mucous coats of the buccal and nasal cavities, the sinuses, the larynx, the pharynx, the trachea or the oesophagus (arrows).

In some instances, the skin and mucous coats are simultaneously affected. If a secondary bacterial infection is involved, the lesions undergo severe inflammation and

necrosis. Birds which survive the infection usually recover fully, but some may remain permanently blind or lose a toe (Forrester & Spalding, 2003).



**Fig.6**

Frequently, the conjunctival mucosa, injured by the pox virus, is an entrance door for additional contamination (*E. coli*, *Staphylococcus spp.* etc.) opening the way for complications

Outbreaks of the cutaneous form of fowl pox have been described in Brazil in broilers with unusual localisation of lesions in feathered parts of the body, mainly in the posterior dorsal and the external thigh regions. The disease resulted in serious economic losses from culled carcasses at the slaughterhouse due to dermatitis (Back et al., 1995). These low-incidence small lesions affecting

the comb and wattles in 5-month-old pox-vaccinated birds were described as another atypical form of fowl pox. The authors suggested that the clinical manifestation was due to a modified virus having survived in the host population. In this epidemiological model, the virus could survive for an indefinite period of time, thus rendering disease control more difficult (Tripathy et al., 1973).

## DIAGNOSIS

Diagnosis is not a challenge when the typical skin or diphtheritic lesions are present at predilection sites. Some difficulties may arise if gross lesions are scarce or appear on atypical sites (e. g. single small papules or crusts only on feet). To confirm the diagnosis, imprint preparations are prepared from lesions, stained with Wright or Gimenez solution for detection of Borrel elementary bodies. By means of conventional histological techniques, tissue cuts from skin or diphtheritic lesions could be also made to detect cytoplasmic inclusion bodies. Other methods for diagnosis confirmations include electron microscopy, immunohistochemistry etc.

For isolation and identification of the virus, inoculation of chorioallantoic membranes taken from 10–12-day-old chick embryos with poxvirus suspension from skin or diphtheritic lesions should be performed. After 5–7 days, a diffuse swelling and irregularly spread grey-whitish nodules appear on the embryonic membrane surface. Apart from chick embryos, avian poxviruses can be inoculated

in cell cultures from chicken and duck embryo fibroblasts, in which they induce a cytopathogenic effect (Tripathy & Reed, 2008). Restriction fragment length polymorphism (RFLP) analysis can also be used to compare field isolates and vaccinal strains of the fowl pox virus, but not as a routine diagnostic method (Schnitzlein et al., 1988; Ghildyaln). Some serological tests such as virus neutralisation (VN), agar gel immunodiffusion (AGID), passive haemagglutination and fluorescent antibody tests, as well as the enzyme-linked immunosorbent assay (ELISA), may also be utilised to detect specific humoral antibody response.

The diphtheritic form is distinguished with a typical stomatitis, with grey-whitish deposits on mucous coats of the oral cavity, the pharynx, the larynx, the upper parts of the trachea and the oesophagus, the crop and the eyes. When the larynx and the trachea are affected, dyspnea and crackles are present. The severe stomatitis could cause disturbed feeding, emaciation and death.

## DIFFERENTIAL DIAGNOSIS

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The lesions caused by cutaneous FP are too specific to be mistaken for symptoms of another disease. However in certain complicated cases, when panophthalmitis had occurred following eyeball infection, certain other conditions such as *E.coli* septicaemia or vitamin A

deficiency should be ruled out. Due to the specificity of lesions affecting the oral mucosa, the pharynx, the larynx and the upper trachea, the diphtheritic form of FP should be differentiated from laryngotracheitis and trichomoniasis.

## PREVENTION AND CONTROL

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### Biosecurity

If pox outbreaks are linked with virus transmission by vectors, containment measures should be focused on reducing the vector population. In case of mosquitoes, the aim should be direct reduction of adults or larvae. The access of vectors to birds could be controlled by mesh screens.

### Vaccines

Vaccination is indicated in regions where the pox is endemic or has been previously diagnosed.

Commercially available vaccines are produced using pigeon poxvirus, attenuated fowl

poxvirus and vector vaccines which could protect against pox.

Such vaccines are derivatives of chick embryo or chick cell cultures. A passive immunity may be passed on to the progeny of remitted or recently vaccinated parents. This passive immunity is reliable until 2–3 weeks of age, and after its depletion the chicks should be vaccinated. The fowl pox vaccine is applied using the wing web stab method. With this recommended method of application, the vaccine is safe. The birds should be checked 7–19 days after vaccination for presence of a yellowish crust at the injection site. 50–100 birds should be tested. Vaccination is considered successful when over

95% of birds tested exhibit a positive result.

The time for vaccination is determined according to the degree of FP exposure, the type of chickens or turkeys subject to vaccination and the current management procedures. It is generally recommended to apply FP vaccines to chickens between 8 and 17 weeks of age and to turkeys between 10 and 22 weeks of age. The best protection is achieved when vaccinated birds are allowed to develop immunity prior to exposure to poxvirus. Good management practices are recommended to restrict the potential exposure to the virus for at least 3 weeks after vaccination. Birds should not be immunised 4 weeks before the beginning or during the egg laying period.

Poxviruses have some unique properties allowing a reliable expression of foreign genes. This way, a remarkable variety of genes coding for specific antigenic proteins have been introduced into the FP genome. The FP viral genome is able to harbour a sufficient amount of foreign DNA without reduction of virus infectivity. Physiological and biological traits of the FP virus present several advantages for its use as an expression vector. The vaccinal

virus causes a mild local infection in a narrow spectrum of hosts, affecting only avian species. Due to its large size, genes from multiple pathogens can be inserted into its genome to produce polyvalent vaccines (Tripathy & Reed, 2008). Efficient vaccines should generate humoral immunity and produce protective antibodies, as well as to provide a long-lasting cell-mediated T-cell response. Recombinant poxviruses are able to induce T-helpers, cytotoxic T-lymphocyte (CTL) and high antibody titers. Furthermore, some other properties of poxviruses make them attractive candidates for development as rational vaccinal vectors –high level of gene expression, capacity for insertion of multiple genes and cytoplasmic gene expression (Beukema et al., 2006).

Poxvirus promoters are essential elements of vectored vaccine design. The FP virus replicates in the cytoplasm using its own transcription mechanism. The level and the time of gene expression are regulated via early, intermediate or late promoters. The early promoters guide gene expression before viral DNA replication, followed by intermediate and late promoters which are subsequently activated. The choice of a promoter apparently influences the level of

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heterologous gene expression. In a tolerant host, the gene expression driven by intermediate and late promoters could yield protein levels more than 10 times higher than that controlled by the early promoter, due to the relative abundance of newly synthesised viral DNA and transcription factors following DNA replication. Regardless of the low intrinsic activity, early promoters have been used in recombinant avian poxviruses for induction of heterologous gene expression prior to the occurrence of virus-provoked cytotoxic effects and abortion replication events encountered in mammalian cells (Beukema et al., 2006).

Using appropriate molecular techniques, recombinant fowl pox or pigeon pox virus vaccines have been created with the capacity to produce proteins from the genes of

poultry pathogens. Among these antigens are the haemagglutinin of the avian influenza virus, protein and enzyme (hemagglutinin neuraminidase) of the Newcastle Disease virus, glycoprotein B of the Marek's Disease virus, the viral protein (VP) 2 of infectious bursal Disease virus, and the nucleoprotein of IBV. In many cases, foreign genes of avian pathogens inserted into the avian poxvirus genome are expressed, and synthesised proteins induce the expression of specific immunity against the respective agent (Tripathy & Reed, 2008).

The potential role of the FP virus in the rational design of vectored vaccines is already implemented to produce the currently available commercial vaccines against avian influenza, Newcastle Disease and infectious laryngotracheitis.

## TREATMENT

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No specific treatment exists. To prevent complications caused by secondary infections, some broad-spectrum antibiotics and vitamins may be used.

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