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A review on field isolates of pigeon pox virus and their relationship with fowl pox virus strains

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Abstract

Avipoxviruses, particularly pigeon pox virus (PPV) and fowl pox virus (FPV), are important pathogens of poultry and pigeons that continue to impact avian health and productivity worldwide. This review synthesizes current knowledge on their etiology, morphology, transmission pathways, pathology, and molecular characteristics, with emphasis on field isolates and their relationship to vaccine strains. Both PPV and FPV produce similar gross and microscopic lesions, yet variations in virulence and pathogenicity are influenced by host species, genetic background, and environmental conditions. Advances in molecular detection have revealed significant genetic diversity, highlighting the circulation of multiple subclades and the emergence of novel variants, including those reported in India. Comparative analyses demonstrate that while conserved genomic regions are stable across isolates, antigenic variation in accessory genes limits the efficacy of existing vaccines and contributes to vaccine failures in the field. Epidemiological data confirm widespread prevalence of avipoxviruses in backyard systems, live bird markets, and wild bird reservoirs, where high vector density sustains transmission and creates opportunities for interspecies spillover. Despite vaccination being the cornerstone of control, conventional live attenuated vaccines often fail to provide full cross-protection against divergent field isolates, while pigeon-specific vaccines remain largely unavailable. Diagnostic limitations, underreporting, and weak surveillance systems further hinder effective management. Future directions emphasize the integration of molecular surveillance, whole-genome sequencing, and the development of recombinant or region-specific vaccines to address antigenic variation. Coupled with improved vector control, farm-level biosecurity, and community engagement, these strategies provide a roadmap for sustainable control of avipoxvirus infections. By linking field epidemiology, molecular insights, and vaccine innovation, this review underscores the need for a multi-dimensional approach to mitigate the economic and health impacts of pigeon and fowl pox viruses.

Keywords: Pigeon pox virus, fowl pox virus, avipoxvirus, field isolates, molecular characterization, vaccine strains, cross-protection, epidemiology

1. Introduction

Avipoxviruses (APVs) are double-stranded DNA viruses of the family *Poxviridae* that cause avian pox, a disease with a global distribution affecting a broad range of wild and domestic birds. Clinical forms range from cutaneous (“dry”) lesions on unfeathered skin to severe diphtheritic (“wet”) lesions of the oropharynx and upper respiratory tract; disease severity depends on the viral strain, host species and age, and environmental and management factors (van der Meer *et al.* 2022) ^[29]. Modern molecular epidemiology has revealed that APVs segregate into distinct clades and subclades, with fowl pox virus (FPV) typically clustering in subclade A1 while pigeon pox virus (PPV) and other avipoxviruses occupy related but distinct branches — a genetic architecture that helps explain differences in host range, virulence and vaccine cross-protection (Chacón *et al.* 2022) ^[10]. Recent field surveillance and sequencing efforts have demonstrated continued emergence of novel APV lineages and unexpected host-associations (for example, detection of APV DNA in mosquito vectors and novel clade E outbreaks in vaccinated flocks), emphasizing that avian pox remains dynamic and capable of producing economically important outbreaks despite vaccination efforts (van der Meer *et al.* 2022; Chacón *et al.* 2022) ^[29, 10]. Together, these findings justify renewed, integrative attention to APV ecology, genomic diversity, and the comparative biology of pigeon and fowl pox viruses-both to understand cross-species risk and to inform vaccine strategies and surveillance priorities.

1.1 Background of Avipoxvirus infections

Avipoxviruses infect hundreds of bird species worldwide and persist in environments through multiple transmission pathways: direct contact and fomite transmission, inhalation or ingestion of contaminated material (notably for diphtheritic forms), and mechanically via arthropod vectors such as mosquitoes and biting flies — vectors that can maintain local transmission cycles between wild birds, backyard flocks and commercial poultry (van der Meer *et al.* 2022; Yehia *et al.* 2023) [29, 32]. Molecular studies using conserved loci such as the P4b (major core protein) and DNA polymerase genes have become standard for typing field isolates and for reconstructing APV phylogenies; these analyses reveal both high within-clade similarity in core genes and regionally distinct strain clusters, with occasional vaccine-like sequences recovered from field cases — suggesting vaccine-derived strains, recombination, or incomplete vaccine-seed matching in some regions (Mohamed *et al.* 2024; Yehia *et al.* 2023) [18, 32]. Experimental and field pathogenicity studies show marked heterogeneity: some pigeon pox isolates produce severe disease in pigeons but little or no pathology in chickens, whereas other isolates and some fowl pox strains cause clinical disease across multiple galliform and columbiform hosts — underscoring that host specificity is not absolute and that cross-species transmission remains a practical risk (Sarker *et al.* 2021; Mohamed *et al.* 2024) [26, 18]. Advances in whole-genome sequencing and next-generation sequencing pipelines during the 2018-2025 period have substantially improved resolution for epidemiological tracing, revealing microevolutionary changes, occasional integration of host or retroviral elements that may affect virulence, and geographic spread patterns crucial for targeted control.

1.2 Economic and health importance in poultry and pigeons

Avian pox has measurable economic and welfare impacts across production systems: in commercial layers and breeders it can reduce egg production, increase feed conversion ratios, necessitate flock culling or replacement, and increase biosecurity and treatment costs; in backyard and smallholder settings the impacts include increased mortality among young birds, loss of marketable animals, and increased labor and medication costs — effects that can be particularly severe in low- and middle-income regions where poultry constitute a critical livelihood and nutrition source (Chacón *et al.* 2022; Muñoz-Gómez *et al.* 2025) [10, 19]. Beyond direct production losses, outbreaks impose indirect costs through surveillance and control measures, disruptions to trade and movement, and consumer confidence impacts when visible disease affects marketability; case reports and outbreak investigations in recent years document situations where vaccinated flocks still experienced economically significant outbreaks due to emergent clades or vaccine-mismatch issues, reinforcing the need for updated vaccine strategies and local strain surveillance (Chacón *et al.* 2022; Mohamed *et al.* 2024) [10, 18]. For pigeons, the disease is important both for hobbyist or commercial pigeon-keeping and for wild pigeon populations; severe cutaneous disease can impair flight and foraging, reduce market and racing performance, and cause mortality in nestlings, while pigeon reservoirs in urban or backyard contexts may seed infection to nearby poultry

(Mohamed *et al.* 2024; van der Meer *et al.* 2022) [18, 29]. Recent reviews on vaccine technologies and recombinant vector vaccines emphasize opportunities to reduce economic burden by improving vaccine seed selection, delivery platforms and thermostability, but they also stress that vaccine development must be coupled with surveillance to detect antigenic drift or novel clade emergence (Wang *et al.* 2024) [30].

2. Methodology

This review was conducted using a systematic approach to identify, evaluate, and synthesize scientific literature on pigeon pox virus (PPV) and fowl pox virus (FPV), with emphasis on field isolates, molecular characterization, and vaccine comparisons. A comprehensive literature search was performed across academic databases including Google Scholar, PubMed, Scopus, Web of Science, and open-access platforms such as ResearchGate. Keywords and Boolean combinations such as “pigeon pox virus,” “fowl pox virus,” “avipoxvirus,” “field isolates,” “molecular characterization,” “cross-protection,” and “vaccination” were employed to capture relevant articles. Publications between 2005 and 2025 were considered, ensuring coverage of both foundational and the most recent research. Priority was given to peer-reviewed journal articles, open-access reports, surveillance studies, and official reference works, while non-scientific sources were excluded. Reference lists of selected articles were further screened to identify additional relevant studies.

To maintain quality and consistency, each article was assessed for relevance, reliability, and novelty of contribution. Studies focusing on epidemiology, molecular techniques, vaccine efficacy, comparative pathology, and regional reports (including India) were prioritized. Information was organized thematically according to predefined review objectives: (1) documenting traditional and modern understandings of PPV and FPV, (2) evaluating genetic and antigenic diversity of field strains, (3) analyzing comparative pathology and immune responses, (4) examining limitations of existing vaccines, and (5) identifying future opportunities for integrated control. Extracted data were synthesized into narrative form under structured subheadings, ensuring that findings from different regions and authors were represented without over-reliance on any single study. This approach allowed a comprehensive, unbiased, and up-to-date review of the role of PPV and FPV in avian health.

3. Pigeon Pox Virus: An Overview

3.1 Etiology and morphology

Pigeon pox virus (PPV) is a member of the genus *Avipoxvirus*, in the subfamily *Chordopoxvirinae* of the family *Poxviridae*; like other avipoxviruses, it possesses a large, double-stranded linear DNA genome of approximately 270-300 kilobases, with a complex brick-or ovoid virion structure enveloped by a lipoprotein membrane, assembled and matured in the cytoplasm of avian host cells (Elshwihdi *et al.* 2025; Alaarajy & Almremdhay, 2025) [12, 6]. Morphologically, PPV causes proliferative lesions with epidermal hyperplasia, ballooning degeneration of keratinocytes, formation of large eosinophilic intracytoplasmic inclusion bodies (so-called Bollinger bodies), and thickening (acanthosis) of stratified squamous epithelium in skin and mucosal sites (Al Aarajy &

Almremdh, 2025; Mohamed *et al.* 2024) [6, 18]. Histopathologically, the characteristic features include vacuolar degeneration, keratinocyte necrosis, edema of epithelial layers, lymphoplasmacytic infiltration beneath the epithelial lesions, and overlying crust or scab formation in cutaneous lesions (Mohamed *et al.* 2024; Al Aarajy & Almremdh, 2025) [18, 6]. In many molecular studies, the core protein gene P4b is used as a conserved marker for identification and phylogenetic placement, and PPV field isolates tend to cluster in subclades of clade A (commonly A2, sometimes A1) when this gene is sequenced (Mohamed *et al.* 2024; Elshwihdi *et al.* 2025) [18, 12].

3.2 Transmission routes and host range

Transmission of pigeon pox virus is multifactorial. Arthropods such as mosquitoes and biting flies act as mechanical vectors, facilitating virus transfer from infected birds (via lesions or secretions) to susceptible pigeons, especially during warm, humid seasons when vector abundance is high (Al Aarajy & Almremdh, 2025; Mohamed *et al.* 2024) [6, 18]. The virus may also spread through direct contact — via contaminated surfaces, shared water or feed troughs, or through abrasions or wounds in skin, especially in unfeathered or lightly feathered regions (Mohamed *et al.* 2024) [18]. Inhalation or ingestion of virus particles from scabs or dust has been reported, and mucosal exposure (oral or conjunctival) may also lead to wet (diphtheritic) forms. Host range of PPV is largely pigeons (*Columba livia* and related pigeons), but cross infections or mixed infections in backyard settings with other birds (chickens, etc.) have been documented (Mohamed *et al.* 2024; Elshwihdi *et al.* 2025) [18, 12]. Molecular phylogeny shows that some PPV isolates are genetically close to fowl pox virus isolates (in certain core gene sequences), hinting at either shared ancestry or possible cross species spill-over under favorable ecological conditions (Mohamed *et al.* 2024) [18]. Factors influencing susceptibility include age (younger pigeons more severely affected), breed, nutritional status, immune status, and environmental stressors (e.g., heat, poor hygiene) (Al Aarajy & Almremdh, 2025) [6].

3.3 Clinical manifestations in pigeons

Clinically, PPV in pigeons primarily manifests in two forms: a cutaneous (“dry”) form and a diphtheritic or mucosal (“wet”) form; often both forms may co-occur in severe infections (Alaarajy & Almremdh, 2025) [6]. The cutaneous form presents as nodular wart-like lesions on unfeathered skin—around the eyes, beak, legs, feet, toes—and sometimes on feathered skin. These lesions may start as small papules or pustules, which crust over and develop thick yellowish, brown or greyish scabs; ulceration or secondary bacterial infection may complicate the lesions (Alaarajy & Almremdh, 2025; Mohamed *et al.* 2024) [6, 18]. In the wet (diphtheritic) form the mucous membranes of the oral cavity, pharynx, larynx, conjunctiva may develop necrotic, fibrinous or cheesy exudative lesions, occasionally leading to difficulty swallowing, breathing, or even airway obstruction (Alaarajy & Almremdh, 2025) [6]. Systemic signs may include depression, decreased feed intake, weight loss, anorexia, sometimes pyrexia, and in younger or immunocompromised birds, increased mortality (Mohamed *et al.* 2024) [18]. Seasonal outbreaks are common (often summer) when vector activity is elevated; in recent field studies mortality in PPV-affected flocks ranges up to ~10-15% in backyard settings (Mohamed *et al.* 2024) [18].

4. Fowl Pox Virus: An Overview

4.1 Etiology and morphology

Fowl pox virus (FPV) is a large, enveloped, double-stranded DNA virus in the genus *Avipoxvirus* (family Poxviridae, subfamily Chordopoxvirinae) (Abosse, Wolde, & Feyisa, 2022; Zhu *et al.* 2025) [5, 33]. The virus has a linear genome of approximately 288-300 kilobase pairs (kbp) encoding more than 250 open reading frames (ORFs), many of which are involved in immune evasion, host interaction, or virulence (Elshwihdi, Kammon, & Asheg, 2025; Abosse *et al.* 2022) [12, 5]. Morphologically, FPV virions are brick- or ovoid-shaped, approximately 200-400 nm in length and 170-200 nm in width, with a dense central nucleoid, lateral bodies, and surrounded by an envelope; virion replication occurs entirely in the cytoplasm of infected host cells (Abosse *et al.* 2022; Zhu *et al.* 2025) [5, 33]. Histopathologic examination of cutaneous lesions shows epidermal hyperplasia, ballooning degeneration of keratinocytes, formation of large eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies), necrosis of epidermal layers, and sometimes secondary bacterial infections under scabs (Elshwihdi *et al.* 2025; Abosse *et al.* 2022) [12, 5].

4.2 Transmission and epidemiology in poultry

Transmission of FPV occurs through multiple routes: direct contact with infected birds, contact with contaminated surfaces (fomites), abrasions on skin, mechanical vectors such as mosquitoes and biting arthropods, and occasionally via inhalation or ingestion of virus particles or scab debris (Abosse *et al.* 2022; Zhu *et al.* 2025) [5, 33]. Epidemiologically, FPV is a worldwide disease affecting domestic chickens of all breeds and ages; prevalence tends to be higher in tropical and subtropical regions, especially where biosecurity is weak, backyard poultry systems predominate, and arthropod vectors are abundant (Abosse *et al.* 2022; Elshwihdi *et al.* 2025) [5, 12]. Recent molecular studies in Libya showed that FPV field isolates there were genetically highly similar (100% identity) to strains from Iraq, Iran, Brazil, and other countries, suggesting both long-distance movement of strains (via trade or migration of birds) and a lack of strong geographic barriers to FPV spread (Elshwihdi *et al.* 2025) [12]. Epidemiological data also show seasonal peaks in transmission, often corresponding with high vector activity and warm, moist conditions; mortality from outbreaks varies depending on whether the diphtheritic (wet) form is involved, flock immunity (vaccination status), and concurrent stressors (nutritional deficiency, concurrent infections) (Abosse *et al.* 2022) [5].

4.3 Clinical features and pathogenesis

Clinically, FPV causes two major forms in poultry: the cutaneous or “dry” form, in which wart-like proliferative lesions and scabs develop on featherless skin regions such as comb, wattles, eyelids, legs, and feet; and the diphtheritic or “wet” form, in which proliferative lesions or necrotic membranes occur in mucous membranes of the upper respiratory or digestive tracts (Abosse *et al.* 2022; Elshwihdi *et al.* 2025) [5, 12]. The incubation period is typically 4-20 days after exposure (Abosse *et al.* 2022) [5]. Pathogenesis begins with virus entry into skin or mucosal epithelium, replication in epidermal cells leading to hyperplasia and formation of Bollinger bodies, followed by cell death and scab formation; in mucosal infections there may be airway obstruction, impaired swallowing, or respiratory distress (Abosse *et al.* 2022) [5]. Virus spreads locally via epidermal

extension and systemically in severe cases; host immune response involves innate responses (macrophages, interferons) followed by cell-mediated immunity and humoral antibody production, but immune evasion genes in FPV (and in some field strains bridging into vaccine strains) can delay clearance and exacerbate lesions (Elshwihdi *et al.* 2025; Zhu *et al.* 2025) ^[12, 33]. Mortality is generally low in cutaneous cases but can be significant in diphtheritic forms or when secondary infections or stressors are present (Abosse *et al.* 2022) ^[15].

5. Comparative Pathology of Pigeon Pox and Fowl Pox Viruses (Updated with new references)

5.1 Gross and microscopic lesions

Recent investigations in Libya and Iraq have documented that pigeons and chickens infected with pigeon pox virus (PPV) or fowl pox virus (FPV) develop strikingly similar gross lesions: wart-like growths, scabs, nodules on unfeathered skin regions such as the beak, cere, eyelids, legs, and toes (Elshwihdi, Kammon, & Asheg, 2025; Faisal & Al-Azzawi, 2023) ^[12, 13]. In pigeon flocks, lesions can be solitary or in clusters, sometimes resembling cauliflower-like masses especially in regions of persistent infection or poor treatment (Faisal & Al-Azzawi, 2023) ^[13]. Microscopic examination reveals epidermal hyperplasia, acanthosis, ballooning degeneration of keratinocytes, and abundant eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies) in both species (Elshwihdi *et al.* 2025; Faisal & Al-Azzawi, 2023) ^[12, 13]. Dermal inflammatory infiltrates composed of lymphocytes, macrophages, and heterophils/neutrophils underlie the epithelial lesions. In chickens especially, secondary changes like necrosis under scabs and infiltration by opportunistic bacteria are more common, along with some fibrosis in chronic lesions (Elshwihdi *et al.* 2025) ^[12]. These data suggest while the tissue pathology is broadly homologous in PPV and FPV infections, the severity and extent of lesion progression differ between host species and with environmental / immunological conditions.

5.2 Virulence and pathogenicity differences

Studies from backyard birds in Libya (Elshwihdi *et al.* 2025) ^[12] indicate that FPV field isolates tend to produce more severe pathology and broader lesion distribution in chickens than similar PPV isolates do in pigeons. For example, FPV in chickens often leads not only to pronounced cutaneous lesions but also diphtheritic (wet) forms in mucosal tissues, especially under conditions of immunological stress or when concurrent infections are present. In contrast, PPV infections in pigeons more frequently remain localized in cutaneous form, with less frequent mucosal involvement. Mortality rates, though generally lower for cutaneous cases, are higher for FPV outbreaks involving diphtheritic lesions (Elshwihdi *et al.* 2025) ^[12]. Genetic studies support these differences: FPV isolates vary in virulence genes, immune evasion loci, and other accessory proteins, which correlate with observed pathology (Kim *et al.* 2022) ^[17]. Kim *et al.* (2022) ^[17] showed among 21 FPV isolates globally, those separated into a particular clade showed lower pathogenicity in chickens, and only specific isolates produced diphtheritic lesions, linking genetic subgrouping with phenotypic severity. Moreover, Faisal & Al-Azzawi (2023) ^[13] observed that pigeons tend to mount stronger local inflammatory

responses, which may limit lesion spread, whereas chickens sometimes show more systemic signs, depressed productivity, and greater mortality when FPV is involved, especially if vaccination is imperfect or absent.

6. Molecular Characterization of Pigeon Pox Virus Field Isolates

6.1 Genetic diversity and strain variation

Molecular characterization of pigeon pox virus (PPV) field isolates over the last decade has revealed appreciable genetic diversity across geographic regions and host systems, with implications for virulence, host range and vaccine efficacy. Sequencing studies that include either the conserved P4b core protein gene or larger genomic regions show that PPV isolates frequently cluster into distinct sublineages within the *Avipoxvirus* clade, and that some pigeon isolates are more closely related to fowl pox virus (FPV) isolates than to other pigeon isolates—evidence consistent with historical host-switching and local diversification (Sahu, 2023; Mohamed *et al.* 2024) ^[24, 18]. Whole-genome sequencing of an Indian pigeonpox isolate (the first complete genome from that region) demonstrated unique genomic features—insertions, deletions and single-nucleotide polymorphisms (SNPs) in several accessory genes implicated in host interaction and immune modulation—highlighting how regionally restricted genomic changes can arise that may alter pathogenicity or antigenic profile (Sahu, 2023) ^[24]. Regional surveys using P4b and other loci have also documented microevolutionary patterns within countries: for example, Iraqi and Egyptian studies found multiple PPV haplotypes circulating simultaneously, sometimes within the same outbreak, suggesting multiple introductions or in-situ evolution rather than a single point source (Jumaa, 2024; Abbiw, 2024) ^[15, 2]. Comparative analyses across recent studies indicate that while core replication genes are highly conserved, variation in genes encoding ankyrin repeat proteins, immune modulators and host range factors is where most inter-isolate differences concentrate—precisely the loci that may explain why some PPV isolates remain pigeon-restricted while others show cross-infection potential for galliform hosts. This body of work underscores the need for continued genomic surveillance (including whole-genome approaches when feasible) to detect emergent variants with altered virulence or vaccine escape potential and to inform rational selection of vaccine seed strains for local contexts.

6.2 Techniques for molecular detection and identification

Modern molecular diagnostics for PPV combine targeted PCR assays for routine detection with sequencing and genomic approaches for in-depth characterization. For frontline diagnostics, conventional PCR targeting the highly conserved 4b (P4b / fpv167) core protein gene remains the workhorse because of its reliability across avipoxviruses and its long history of use in comparative phylogenetics; many field investigations validate P4b amplicons by Sanger sequencing and BLAST comparison to assign isolates to clades (Abd El Hafez *et al.* 2021; Mohamed *et al.* 2024) ^[3, 18]. Real-time quantitative PCR (qPCR) assays using TaqMan probes have been developed for sensitive detection in tissue swabs, feathers and blood, enabling detection even in low-titer samples and supporting surveillance from live birds or environmental sampling (Baek *et al.* 2020) ^[17]. New

isothermal and rapid methods (for example MIRA/RPA-based or LAMP-style assays, and recent fluorescent-probe adaptations) aim to shorten turnaround time and allow near-point-of-care screening in field laboratories (Zhu *et al.* 2025)^[33]. For deeper characterization, Sanger sequencing of P4b and other loci (e.g., DNA polymerase, VLTf3) is commonly used for phylogenetic placement, but whole-genome sequencing (WGS) using short-read platforms or portable long-read devices (Oxford Nanopore MinION) is increasingly feasible and offers the resolution to detect recombination, accessory gene variation and SNPs associated with virulence (Sahu, 2023)^[24]; rapid WGS applications described in sequencing method studies). Several recent field studies successfully applied nanopore or combined short+long read strategies to obtain near-complete genomes directly from lesion material, bypassing prolonged culture on CAM (chick embryo chorioallantoic membrane) and reducing bias introduced by passaging (research on rapid WGS methods). Best practice now couples an initial sensitive qPCR screen (to confirm APV presence) with targeted Sanger sequencing for routine typing and WGS for isolates of special interest (vaccine failure, unusual pathology, or suspected cross-species events). Importantly, molecular workflows should include negative controls, confirmation by sequencing, and where possible, deposition of sequences in public databases to enable ongoing comparative surveillance and meta-analyses across regions.

7. Comparison with Fowl Pox Virus Field and Vaccine Strains

7.1 Cross-protection studies

Cross-protection between pigeon pox virus (PPV) and fowl pox virus (FPV) has been investigated intermittently in both experimental and field settings, and results point to a nuanced picture in which antigenic relatedness does not guarantee reliable heterologous protection under field conditions. Early classical cross-challenge studies and more recent vaccine-failure investigations indicate that some FPV vaccine strains can afford partial protection against certain PPV isolates, while other PPV field isolates remain capable of causing disease in vaccinated chickens or evade immunity sufficiently to produce clinical signs (Radwan, 2020; Mohamed *et al.* 2024)^[21, 18]. For instance, molecular surveillance in Egypt by Mohamed and colleagues (2024)^[18] documented isolates from pigeons with P4b sequences that were 100% identical to a local FPV vaccine seed (fowlpox/VSVRI/Egypt), yet field outbreaks still occurred in flocks vaccinated with that strain — suggesting that identity in a conserved locus (P4b) does not necessarily equate to protective antigenic equivalence across the whole genome. Experimental immunization trials using cell-culture adapted and CAM-derived vaccine seeds also show variable outcomes: some heterologous challenge experiments resulted in reduced lesion severity and lower virus shedding but not sterilizing immunity (Okafor, 2025 preprint; Radwan, 2020)^[20, 21]. These mixed results are likely explained by (a) antigenic differences concentrated in accessory proteins and surface antigens not covered by conservative locus comparisons, (b) differences in vaccine take and host immune status, and (c) field factors such as coinfections and high vector pressure that can overwhelm partial immunity (Kim *et al.* 2022; Yang, 2024)^[17, 1]. Practically, this means cross-protection can be partial and strain-dependent: homotypic (same-strain) vaccination

remains the most reliable protection, and where heterologous vaccines are used, their performance should be validated by local challenge or field-effectiveness studies. The implication for surveillance is clear: vaccine efficacy assessments should go beyond a single gene (e.g., P4b) and incorporate clinical endpoints and whole-genome comparisons to better predict cross-protective potential (Mohamed *et al.* 2024; Kim *et al.* 2022)^[18, 17].

7.2 Antigenic and genomic similarities/differences

At the genomic level, FPV and PPV share a conserved core involved in replication and morphology, but differ substantially in accessory genes implicated in host range, immune modulation and virulence. Comparative genomic surveys and phylogenetic analyses (including full genome studies and multi-locus sequencing) reveal that while the P4b gene and other replication core genes are highly conserved across avipoxviruses, genes coding for ankyrin repeat proteins, immune-modulatory secreted proteins, and certain surface proteins show the bulk of inter-isolate diversity (Kim *et al.* 2022; Sahu, 2023)^[17, 24]. Whole genome studies, for example the Indian PPV genome report (Sahu, 2023)^[24] and multiple FPV genome comparisons (Elshwihdi *et al.* 2025)^[12], identify insertions/deletions and SNPs in accessory ORFs that plausibly alter antigenic surfaces or immune-evasion capacity. Antigenically, serological cross-reactivity is often observed in crude antigen tests (e.g., complement fixation or agar gel immunodiffusion), but neutralization assays and protection studies reveal more limited functional cross-neutralization — indicating that serologic reactivity overestimates protective antigenic similarity (Abbiw, 2024; JWPR surveillance report, 2024)^[2, 16]. Importantly, recent field work has highlighted cases where nucleotide identity in P4b or other conserved markers reached 100% between a vaccine seed and field isolates (Mohamed *et al.* 2024)^[18], yet clinical disease in vaccinated flocks still occurred — reinforcing that whole-genome antigenic differences, post-translational modifications, or expression differences (e.g., truncated immunogenic proteins) may account for vaccine escape. The take-home message is that genomic similarity in conserved loci is a useful first screen for relatedness, but comprehensive antigenic mapping and genome-wide analyses are required to understand functional immunity and to predict vaccine match.

7.3 Implications for vaccine development

The genomic and antigenic evidence together point toward several practical directions for vaccine development. First, vaccine seed selection should incorporate whole-genome comparisons and, where possible, cross-neutralization data rather than relying solely on conserved gene identity; local strains with demonstrated divergence in accessory genes may require updated or regionally matched vaccine seeds (Kim *et al.* 2022; Elshwihdi *et al.* 2025)^[17, 12]. Second, modern vaccine platforms — including cell-culture adapted live attenuated vaccines, vectored recombinant vaccines and thermostable formulations — present opportunities to improve breadth of protection and field performance (Okafor, 2025 preprint; Zhu *et al.* 2025)^[20, 33]. Recombinant vaccines that express multiple antigenic determinants or that target conserved immunogenic proteins across avipoxviruses could help broaden cross-protection, while platforms that improve mucosal immunity may better

prevent diphtheritic disease. Third, vaccine evaluation must be iterative and context-specific: field trials, post-vaccination surveillance with molecular typing, and challenge studies against locally circulating PPV/FPV isolates should be routine components of vaccine roll-out (Tseng, 2024; JWPR, 2024)^[28, 16]. Finally, integrated control that complements vaccination with vector management, improved biosecurity, and targeted education for backyard flock owners will reduce pressure on vaccines and lower the probability of vaccine-escape selection. Taken together, the literature supports a transition from “one-size-fits-all” FPV vaccines to more evidence-based, genomically informed vaccine strategies coupled with active genomic surveillance to detect and respond to emergent antigenic variants.

8. Immune Response and Cross Immunity

8.1 Host immune response in pigeons and poultry

The immune response to avipoxvirus infections in both pigeons and poultry reflects the complex interplay between innate defenses, cell-mediated immunity, and humoral antibody production. Following natural infection, local innate responses are initiated by epithelial cells and macrophages at the lesion site, leading to interferon release, recruitment of heterophils/lymphocytes, and localized inflammation (Tripathy & Reed, 2008; Dey *et al.* 2019)^[27, 11]. Cellular immunity plays a decisive role: cytotoxic T lymphocytes (CD8+) and T-helper responses are essential for clearing virus-infected keratinocytes and limiting lesion spread (Giotis & Skinner, 2019)^[14]. Studies in chickens experimentally infected with FPV showed strong induction of pro-inflammatory cytokines such as IL-1 β , TNF- α , and IFN- γ within days of infection, correlating with lesion regression (Abdallah *et al.* 2022)^[4]. In pigeons, recent work highlights that local mucosal and cutaneous immune responses are often robust, sometimes containing lesions more effectively than in chickens, which may explain why diphtheritic forms of pigeon pox are less frequent (Faisal & Al-Azzawi, 2023)^[13]. Antibody responses, while detectable, are generally considered secondary in importance compared to cellular responses, but neutralizing antibodies can prevent reinfection with homologous strains. Notably, host genetics and age influence outcomes: younger birds often show more severe disease and delayed cellular responses, while breed differences in chickens affect lesion severity and viral shedding (Giotis & Skinner, 2019; Abdallah *et al.* 2022)^[14, 4].

8.2 Role of vaccination and heterologous protection

Vaccination remains the cornerstone of avipoxvirus control in poultry, typically using live attenuated fowl pox virus vaccines propagated in embryonated eggs or cell culture. These vaccines stimulate both humoral and cellular immunity, with the characteristic “take reaction” at the wing-web inoculation site serving as evidence of immune priming (Tripathy & Reed, 2008; Radwan, 2020)^[27, 21]. However, cross-protection between FPV vaccines and PPV field isolates is inconsistent. While some degree of heterologous immunity occurs, several field reports have documented disease outbreaks in vaccinated flocks, highlighting that vaccine-induced immunity may not fully neutralize divergent strains (Mohamed *et al.* 2024; Kim *et al.* 2022)^[18, 17]. Antigenic mismatch, variability in host immune responses, and inadequate vaccine coverage are contributing factors. In pigeons, formal vaccination against

PPV is uncommon, but studies suggest that exposure to FPV vaccines provides only partial protection, insufficient to prevent cutaneous lesion development after challenge with heterologous PPV strains (Radwan, 2020)^[21]. Advances in recombinant vaccine technologies, such as vectored vaccines expressing immunogenic FPV proteins or chimeric constructs incorporating conserved epitopes across avipoxviruses, are being explored to broaden heterologous protection (Wang *et al.* 2024)^[30]. Ultimately, effective control requires region-specific vaccine strain selection informed by molecular surveillance, coupled with good management and vector control, to reduce the selective pressure for vaccine escape.

9. Epidemiological Significance (Updated with other scientists)

9.1 Prevalence in different regions (including Indian research)

Recent epidemiological studies reveal broad variation in the prevalence of Avipoxvirus (APV) infections across regions, influenced strongly by bird husbandry type (backyard vs commercial), climate, vector presence, and surveillance effort. For example, molecular and pathological screening in backyard birds in Egypt (Mohamed *et al.* 2024)^[18] found APV infection highly common among pigeons and chickens with visible scabs or nodular lesions, especially during the hotter months when vectors thrive. In Europe, a study of asymptomatic wild birds in Italy detected APV DNA in 43.33% of birds sampled (Bertelloni *et al.* 2022)^[8], indicating widespread subclinical circulation (without visible lesions).

Focusing on India: Sahu *et al.* (2020)^[24] studied domestic chickens and pigeons in the Eastern Indian state of Odisha across 2010-2018, reporting morbidity rates of 18-19.2% in chickens and 16.9-23% in pigeons (based on clinical lesion surveys plus molecular confirmation). Furthermore, the first complete genome characterisation of an Indian pigeonpox virus isolate (Pur-Od-4b/01/Ind) was published in 2025, confirming that APV is not only present but genetically distinct in Indian pigeon populations and likely endemic (Sahu *et al.* 2025)^[25]. These Indian studies underscore that APV infections are persistent in mixed backyard settings, with seasonal peaks aligning with vector activity, and highlight under-recognized but consistent burden in both pigeons and chickens.

9.2 Risk of interspecies transmission

Evidence for interspecies transmission (spillover) of APVs has been strengthened in recent years by field, genomic, and experimental studies. Molecular phylogenetic comparisons show that APV strains from pigeons sometimes cluster closely with strains from chickens in conserved genes, suggesting recent or ongoing spillover or shared environmental exposure (Sahu *et al.* 2025; Sahu *et al.* 2020)^[25, 10]. For example, the Odisha study (Sahu, 2020)^[10] identified APV subclades A1 in chickens and A3 in pigeons, indicating that different avipoxvirus strains circulate in both species in the same region, raising the possibility that viral strains may cross species under suitable conditions.

Wild bird surveillance adds to the concern: an Italian study (Bertelloni *et al.* 2022)^[8] found APV DNA in a large fraction of wild birds (without visible lesions), particularly aquatic birds of Anseriformes, suggesting that wild and domestic birds may share virus reservoirs and transmission

pathways, potentially facilitating spillover in both directions. Experimental and observational studies (outside India) have further indicated that mechanical vectors (mosquitoes, flies) and contaminated environment can bridge species; though data specifically for pigeon-to-chicken transmission in India are limited, the genetic findings plus shared environments (live bird markets, mixed backyard rearing) strongly suggest risk is non-trivial.

10. Challenges and Gaps in Current Research

10.1 Limitations of existing vaccines

Despite decades of use, current fowl pox virus (FPV) vaccines — mainly live attenuated strains propagated in embryonated chicken eggs or cell culture — face several important limitations. Field reports from different regions consistently note outbreaks in vaccinated flocks, suggesting incomplete protection against circulating strains. For example, outbreak investigations in Egypt and Libya found vaccinated flocks with clinical disease, and molecular analysis revealed that field strains were genetically distinct in accessory genes compared with vaccine seeds, potentially explaining immune escape (Elshwihdi *et al.* 2025; Mohamed *et al.* 2024) [12, 18]. Similarly, in India, Sahu (2023, 2025) [24, 25] reported that pigeonpox virus (PPV) isolates circulating in Odisha and other regions shared partial genetic similarity with vaccine strains but contained unique mutations in immunomodulatory proteins, raising concerns about cross-protection and long-term efficacy. Another issue is that most FPV vaccines were developed decades ago and are not updated regularly to reflect genetic changes in field strains (Kim *et al.* 2022) [17]. In pigeons, no dedicated commercial vaccines are widely available, and use of FPV vaccines provides only partial, inconsistent heterologous protection (Radwan, 2020) [21]. Adverse reactions such as severe “take reactions” or localized necrosis, though rare, also limit farmer acceptance in some settings. Moreover, logistical challenges — cold chain requirements, multi-dose schedules, and limited thermostability — reduce vaccine effectiveness in rural or resource-limited regions (Wang *et al.* 2024) [30]. Collectively, these factors highlight the urgent need for updated, region-specific vaccines, possibly using recombinant platforms or polyvalent formulations incorporating conserved epitopes across avipoxviruses.

10.2 Difficulties in diagnosis and control

Diagnosis and control of avipoxvirus infections remain difficult due to overlapping clinical presentations, subclinical infections, and limited access to molecular testing in many regions. Clinically, both PPV and FPV cause nodular and diphtheritic lesions that can resemble other avian diseases such as trichomoniasis, candidiasis, or bacterial stomatitis, complicating field diagnosis (Giotis & Skinner, 2019) [14]. Histopathology (Bollinger bodies) provides supportive evidence but requires laboratory infrastructure. Molecular diagnostics such as PCR and sequencing are increasingly available, but their uptake in low-resource settings remains limited (Abd El Hafez *et al.* 2021; Baek *et al.* 2020) [3, 7]. In India and many tropical countries, reliance on visual diagnosis leads to underreporting or misclassification, and asymptomatic carriers (wild birds, pigeons) further complicate epidemiology (Bertelloni *et al.* 2022) [8]. Control is also hindered by ecological and management factors: high vector populations, backyard poultry systems with mixed species,

and close contact between wild and domestic birds maintain continuous transmission cycles (Sahu, 2020; Bertelloni *et al.* 2022) [23, 8]. Biosecurity interventions such as vector control, strict separation of species, and improved hygiene are difficult to enforce in smallholder systems, while live bird markets act as amplifiers of transmission (JWPR, 2024) [16]. Furthermore, the slow pace of whole-genome sequencing in many endemic regions limits the ability to monitor antigenic drift or detect vaccine escape variants in real time. The combination of diagnostic gaps, environmental complexity, and limited genomic surveillance creates major challenges for timely detection and effective control of avipoxvirus outbreaks.

11. Future Perspectives

11.1 Need for molecular surveillance

Sustained, systematic molecular surveillance is foundational to detect emerging avipoxvirus variants, monitor vaccine-match, and guide targeted control measures. Recent studies demonstrate the practical benefits of coupling routine PCR screening with sequencing: field surveillance in backyard birds repeatedly uncovers multiple co-circulating haplotypes and occasional vaccine-like sequences that would be missed by clinical observation alone (Mohamed *et al.* 2024) [18]. Whole-genome sequencing (WGS), including affordable nanopore platforms, now enables rapid, near-real-time typing from lesion material without lengthy culture steps, revealing microevolutionary changes in accessory genes linked to host range and virulence (rapid WGS pipelines and field applications). Vector surveillance — for example detection of avipoxvirus DNA in mosquito pools — provides early warning of local transmission potential and can identify ecological hotspots for intervention (van der Meer *et al.* 2022) [29]. For countries such as India, where mixed backyard systems and live bird markets sustain complex transmission networks, an actionable surveillance backbone should combine (1) sentinel sampling in backyard flocks and pigeons, (2) qPCR screening for APV presence, (3) targeted Sanger sequencing of P4b for rapid clade calls, and (4) routine WGS of representative isolates uploaded to public databases to enable regional and global comparisons. Establishing such pipelines will require investments in basic sequencing infrastructure, training, standardized protocols and data-sharing agreements, but the return on investment is high: timely detection of divergent or vaccine-escape strains allows for rapid vaccine seed updates, targeted vaccination campaigns, and vector control measures before large outbreaks occur.

11.2 Potential of recombinant vaccines

Recombinant vaccine platforms present a promising route to broaden protective immunity against diverse avipoxviruses and to overcome several limitations of traditional live attenuated vaccines. Reviews and experimental studies demonstrate that FPV can be used as a viral vector to express heterologous antigens (e.g., NDV, influenza HA) and that recombinant constructs can enhance immunogenicity while enabling multivalent protection within a single product (Wang *et al.* 2024; Zhao *et al.* 2020) [30]. Recombinant fowlpox constructs have also shown utility as delivery systems that elicit robust cell-mediated immunity — the arm most critical for clearing poxvirus infections — and can be engineered to express conserved antigenic determinants from multiple APV strains to

improve cross-protection (Boyle, 2017; Wang *et al.* 2024)^[9, 30]. Emerging approaches such as virus-like particles (VLPs), thermostable formulations and adjuvant optimization further expand options for field-friendly vaccines that perform well in low-resource settings where cold-chain and multiple dosing are challenging (Raji *et al.* 2024). However, recombinant vaccine rollout must be guided by genomic surveillance (to select conserved epitopes) and by rigorous field trials to confirm heterologous protection against circulating PPV and FPV lineages; regulatory pathways and cost considerations will also shape adoption in smallholder systems.

11.3 Integrated disease management strategies

Vaccination alone will not eliminate avipoxvirus disease; integrated strategies that combine vaccination with vector control, biosecurity improvements, and community engagement are essential. Vector management (reducing mosquito and biting-fly breeding sites, insecticidal measures around coops) reduces mechanical transmission risk and lowers pressure on vaccines, while basic biosecurity (separating species, improving feed/water hygiene, restricting movement of birds and equipment) prevents local amplification in backyard contexts (Giotis & Skinner, 2019; recent biosecurity studies)^[14]. Digital tools and real-time reporting systems—coupled with diagnostic hubs that provide qPCR confirmation and sequencing—can enable rapid outbreak response and evidence-based targeting of resources (real-time surveillance frameworks). For India and similar settings, extension programs tailored to backyard keepers and market vendors (practical guidance on lesion recognition, timely reporting, simple wing-web vaccination techniques, and environmental sanitation) are cost-effective interventions that synergize with technological advances. Finally, One Health approaches that integrate wild bird and vector surveillance will improve early detection of cross-species spillover risks and support ecosystem-level interventions that protect both poultry livelihoods and avian biodiversity.

12. Conclusion

Avipoxvirus infections, particularly pigeon pox virus and fowl pox virus, continue to pose significant challenges to avian health, productivity, and disease management worldwide. Comparative evidence highlights both the shared pathology and distinct biological features of pigeon and fowl pox viruses, underscoring the complexity of their host-pathogen interactions. Molecular studies demonstrate considerable genetic diversity among field isolates, with

region-specific variations that influence virulence, host range, and vaccine effectiveness. Although conventional vaccines have provided a degree of control in poultry, their limitations against emerging or divergent strains, coupled with the absence of widely available pigeon-specific vaccines, remain critical obstacles. The epidemiological significance of these viruses lies not only in their persistence within domestic flocks but also in their circulation among wild birds and vectors, which maintain a continuous risk of interspecies transmission. Diagnostic challenges, reliance on clinical signs, and inadequate surveillance infrastructures exacerbate underreporting and hinder timely response. The integration of molecular surveillance, next-generation sequencing, and regionally adapted vaccines, alongside improved vector management and farm-level biosecurity, will be central to sustainable disease control. Looking ahead, the future of avipoxvirus management lies in a holistic approach that combines advances in recombinant vaccine technology, genomic monitoring, and community-based disease management strategies. Strengthening these efforts will not only reduce economic losses in poultry and pigeon industries but also minimize the risk of viral adaptation and cross-species spread. In conclusion, tackling pigeon and fowl pox viruses demands a multi-pronged strategy that bridges laboratory research, field surveillance, and practical interventions, ensuring resilient poultry systems and healthier avian populations.

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14. Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this review article. This work was carried out purely for academic and research purposes, and no financial or personal relationships have influenced its content.

Table 1: Epidemiological Studies on Pigeon and Fowl Pox Virus (2005-2025)

Author(s), Year	Country /Region	Host species	Method(s) Used	Key Findings
Sahu, 2020 ^[23]	India (Odisha)	Chickens & pigeons	PCR (P4b), sequencing	Identified subclades A1 (chickens) and A3 (pigeons); morbidity 17-23%; seasonal outbreaks during monsoon.
Mohamed <i>et al.</i> 2024 ^[18]	Egypt	Backyard chickens & pigeons	PCR, histopathology, sequencing	Detected both FPV and PPV in field outbreaks; some strains identical to vaccine seed by P4b, yet outbreaks occurred.
Elshwhidi <i>et al.</i> 2025 ^[12]	Libya	Chickens & pigeons	PCR, sequencing, histopathology	Field isolates genetically similar to strains from Iraq/Iran; confirmed vaccine failure in some flocks.
Bertelloni <i>et al.</i> 2022 ^[8]	Italy	Wild birds	PCR (P4b)	APV DNA in 43.33% of asymptomatic wild birds, highlighting hidden reservoir potential.
Sahu <i>et al.</i> 2025 ^[25]	India	Pigeons	Whole-genome sequencing	First complete genome of Indian PPV isolate (Pur-Od-4b/01/Ind); revealed SNPs in immunomodulatory genes.

Table 2: Comparative Pathology of PPV and FPV

Author(s), Year	Host species	Gross Lesions	Microscopic Lesions	Key Observations
Faisal & Al-Azzawi, 2023 ^[13]	Chickens & pigeons	Wart-like nodules, scabs on unfeathered skin	Ballooning degeneration, Bollinger bodies	PPV lesions more proliferative in pigeons; FPV lesions ulcerative with bacterial invasion.
Giotis & Skinner, 2019 ^[14]	Chickens	Comb, wattles, eyelids lesions	Hyperplasia, necrosis, inclusions	FPV pathology well-documented; diphtheritic lesions lead to higher mortality.
Elshwihdi <i>et al.</i> 2025 ^[12]	Pigeons & chickens	Nodular lesions in both hosts	Hyperplasia, necrosis, inclusions	Vaccine-like strains still caused pathology in field cases.

Table 3: Molecular Characterization and Detection

Author(s), Year	Gene/Method	Host/Region	Findings
Abd El Hafez <i>et al.</i> 2021 ^[3]	PCR (P4b)	Pigeons, Egypt	Reliable detection of PPV from skin lesions; confirmed circulating field isolates.
Baek <i>et al.</i> 2020 ^[7]	qPCR (TaqMan)	Wild birds	Sensitive detection of avipoxvirus DNA in feathers/blood samples.
Sahu, 2023 ^[24]	Whole-genome sequencing	India (pigeon)	Genome revealed unique SNPs and indels in immune-modulatory genes, suggesting adaptation.
Kim <i>et al.</i> 2022 ^[17]	Genomic analysis	FPV global isolates	Identified diversity in ankyrin repeat and immune-evasion genes correlating with pathogenicity.
Zhu <i>et al.</i> 2025 ^[33]	MIRA assay	Avipoxvirus isolates	Developed rapid, field-adaptable detection system.

Table 4: Vaccine Studies and Cross-Protection

Author(s), Year	Vaccine type	Host species	Findings
Radwan, 2020 ^[21]	Egg & cell culture FPV vaccines	Chickens	Provided partial heterologous protection; outbreaks still reported.
Mohamed <i>et al.</i> 2024 ^[18]	FPV vaccine strain comparison	Chickens/pigeons	Identical P4b to vaccine strain detected in outbreaks; vaccine not fully protective.
Wang <i>et al.</i> 2024 ^[30]	Recombinant vector vaccines (review)	Poultry	Highlighted potential of recombinant FPV vaccines expressing multiple epitopes.
Sahu <i>et al.</i> 2025 ^[25]	Genome vs vaccine strain analysis	Pigeons, India	Field isolate genetically distinct from vaccine strains; indicated need for local vaccine seeds.

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