VETERINARIA ITALIANA
RIVISTA DI SANITÀ PUBBLICA VETERINARIA

Review

Highly pathogenic avian influenza A(H5N1) in domestic cats: epidemiology, clinical manifestations, and One Health implication

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Veterinaria Italiana, Vol. 62 No. 3 (2026) DOI: 10.12834/VetIt.4040.41131.2

Abstract

Historically considered accidental hosts of influenza A viruses, domestic cats (Felis catus) have gained attention during the global expansion of highly pathogenic avian influenza (HPAI) H5N1 clade 2.3.4.4b. In addition to traditional exposure via predation on infected wild birds, recent outbreaks have highlighted novel transmission routes, including contaminated commercial raw poultry products and unpasteurised milk. The susceptibility of cats has been linked to the abundant distribution of α-2,3-linked sialic acid receptors in both the respiratory tract and the central nervous system, while adaptive mutations such as PB2-E627K may enhance viral replication efficiency in mammalian hosts. Clinical disease is characterised by severe respiratory, neurological, and ocular manifestations, including acute bilateral amaurosis, and is associated with high mortality rates. Recent documentation of feline-associated zoonotic transmission highlights the importance of surveillance and infection control measures in veterinary settings. Early recognition of compatible clinical syndromes, combined with rapid molecular diagnosis and appropriate biosafety measures, is essential for case management and risk mitigation. This review summarises current insights into the epidemiology, pathogenesis, clinical presentation, diagnosis, and One Health implications of H5N1 infection in domestic cats.

Keywords

Influenza virus, Avian influenza, Domestic cats, One Health, zoonotic

Introduction

Historically, avian influenza A viruses (AIVs) have been recognised as highly species-specific pathogens, predominantly maintained in wild aquatic birds and domestic poultry populations (Webster et al., 1992). Felid infections were considered sporadic, self-limiting spillover events, with affected animals regarded as dead-end hosts. Feline cases reported during the early 2000s in Asia and during the 2006 European outbreaks were localised phenomena, almost exclusively associated with the ingestion of infected wild birds or contaminated poultry offal (Kuiken et al., 2004; Leschnik et al., 2007). However, the global epidemiological landscape changed with the emergence and rapid spread of highly pathogenic avian influenza A(H5N1) (HPAI H5N1) clade 2.3.4.4b (WHO, 2025). This lineage has demonstrated an expanded host range and an increased capacity to infect mammalian species. Consequently, the domestic cat (*Felis catus*) is now recognised as a highly susceptible host and a valuable sentinel species for HPAI H5N1 surveillance, as highlighted by recent reviews addressing H5N1 infection in cats

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(Bonilla-Aldana et al., 2025). These observations challenge previous assumptions regarding AIV host restriction and highlight the role of companion animals at the human-animal interface within the One Health framework (Borland et al., 2020). This narrative review summarises current knowledge of HPAI H5N1 infection in domestic cats, considering both early reports and recent cases associated with the clade 2.3.4.4b panzootic. The review focuses on epidemiology, exposure routes, clinical presentation, diagnostic approaches, biosafety measures and One Health implications. The literature was identified through searches in PubMed and Google Scholar, together with a review of official surveillance reports and guidance documents from animal and public health authorities, including WHO, WOAH, EFSA, ECDC, EURL and CDC. The search covered the period from 2004, when the first natural H5N1 infections in domestic cats were reported, to 2026. Search terms included combinations of “H5N1”, “highly pathogenic avian influenza”, “avian influenza”, “domestic cat”, “feline”, “felid”, “clade 2.3.4.4b”, “genotype B3.13”, “raw milk”, “neurological signs”, “ocular signs”, “diagnosis”, “biosafety” and “One Health”. Articles, surveillance reports and guidance documents were selected when they provided relevant information on H5N1 infection in domestic cats, mammalian spillover events, exposure routes, clinical or pathological findings, diagnostic procedures, biosafety measures or veterinary public health implications.

Virological features and mammalian adaptation

From a virological perspective, the aetiological agent belongs to the family *Orthomyxoviridae*, genus *Alphainfluenzavirus*, species *Alphainfluenzavirus influenzae* (ICTV, 2025). The virion is enveloped and containing a negative-sense, single-stranded RNA genome segmented into eight distinct RNA segments encoding both structural and non-structural proteins (Webster et al., 1992). This genomic organisation facilitates genetic reassortment when different influenza viruses co-infect the same host cell. Classification into specific subtypes is based on the antigenic properties of the two major surface glycoproteins: haemagglutinin (HA), which mediates viral attachment and entry, and neuraminidase (NA), which facilitates the release of newly formed virions from infected cells. To date, 18 HA subtypes and 11 NA subtypes have been identified, with highly pathogenic avian influenza viruses predominantly expressing the H5 or H7 HA subtypes (WHO, 2025). The virulence and systemic tropism of clade 2.3.4.4b viruses in cats are associated with adaptations involving both surface glycoproteins and the viral replication machinery. A key component of this host adaptation is receptor usage. While classical avian influenza viruses preferentially bind to sialic acid receptors linked to galactose via an α-2,3-glycosidic bond (α-2,3-Gal), which are highly abundant in the avian respiratory and gastrointestinal tracts (Ito et al., 1998), mammalian-adapted influenza viruses typically target α-2,6-linked sialic acid (α-2,6-Gal) receptors (Connor et al., 1994; Shinya et al., 2006). Importantly, the feline lower respiratory tract and central nervous system (CNS) express relatively high levels of α-2,3-Gal receptors (Chothe et al., 2025; Kuchipudi et al., 2021; Wang et al., 2013). This distribution may partly explain the susceptibility of cats to systemic infection without requiring a complete shift towards α-2,6 affinity (van Riel et al., 2007).

In addition to receptor binding dynamics, these viruses utilise adaptations in the polymerase complex to overcome species barriers. High-throughput sequencing of feline isolates has repeatedly identified key adaptive mutations within the polymerase basic protein 2 (PB2) subunit, most notably the E627K and D701N substitutions (Subbarao et al., 1993). These specific amino acid replacements effectively lower the optimal replication temperature threshold of the viral polymerase complex. Consequently, this allows the virus to bypass the restriction of the higher avian body temperature (~40°C) and replicate efficiently at the lower temperatures of the mammalian upper respiratory tract (~33–35°C), significantly enhancing replication kinetics and systemic spread in mammalian hosts (Hatta et al., 2001).

Epidemiology of H5N1 infection in domestic cats

The recent emergence of HPAI H5N1 clade 2.3.4.4b in feline populations can be described through three epidemiologically relevant phases. The first major epidemiological event occurred during the summer of 2023 with the outbreak in Poland, where domestic cats across geographically separated regions developed severe systemic disease. Because many affected animals lacked any direct history of wild bird contact, a foodborne exposure route through contaminated raw poultry products was suspected, indicating that indoor cats could be exposed through commercial feeding practices (Domańska-Blicharz et al., 2023). This foodborne transmission route became more evident during the subsequent US dairy farm epizootic, marking the second phase of this epidemiological progression and representing a significant shift in HPAI H5N1 ecology. Following the spillover of HPAI H5N1 genotype B3.13 into dairy cattle, resident farm cats ingesting contaminated unpasteurised raw milk developed severe systemic infections, with a reported mortality rate exceeding 50%, supporting raw milk as an important exposure route in affected dairy farm settings (Burrough et al., 2024).

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This expansion in mammalian host range and geographical distribution highlights the need for continuous international surveillance, given that the virus now represents a persistent environmental and multi-species risk (EFSA, ECDC & EURL, 2024). The third phase is represented by clusters of HPAI H5N1 infection in domestic cats and by increasing attention to the possible occupational risk associated with close contact with infected animals. Between late 2024 and early 2025, retrospective active surveillance in Los Angeles County, California, identified clusters of domestic cats infected with HPAI H5N1 genotype B3.13 after exposure to commercially purchased raw animal products or raw milk. An additional report described multiorgan lesions in a cat associated with the consumption of recalled raw milk (Acevedo et al., 2026; Vaughan et al., 2026).

Transmission pathways and spillover events

The epidemiology of clade 2.3.4.4b in Europe has shifted significantly, marked by persistent environmental reservoirs and increasing spillover events into mammalian hosts (EFSA, ECDC & EURL, 2025). While historical outbreaks in companion animals were considered self-limiting, official multi-agency surveillance reports from the European Food Safety Authority (EFSA), the European Centre for Disease Prevention and Control (ECDC) and the European Union Reference Laboratory for Avian Influenza (EURL) documented an increase in HPAI H5N1 detections in domestic cats and wild carnivores across multiple European countries (EFSA, ECDC & EURL, 2025). This increase in cases occurred against a background of widespread mortality and continuous circulation of the virus in wild birds, associated with high environmental viral loads and increased mammalian exposure. Transmission dynamics within the European feline population depend chiefly on predation and scavenging of infected wild birds, with no significant link to commercial feed contamination. A notable sentinel event occurred in February 2026 in the Sigmaringen district of Germany, where an H5N1 outbreak in a small poultry holding resulted in severe clinical disease and high mortality among resident free-roaming cats (Dressler et al., 2026). Epidemiological investigations identified bite marks on poultry carcasses, supporting predation or scavenging as the most likely exposure route. Notably, serological testing showed that all surviving cats on the farm were positive for H5-specific antibodies, indicating widespread exposure within the feline group (Dressler et al., 2026). These recurring European feline cases highlight an expanding mammalian host range and a shifting viral ecology at the wildlife-companion animal interface. Because companion felids may be exposed in agricultural environments while living in close contact with humans, their susceptibility to HPAI H5N1 has potential implications for zoonotic risk assessment. Consequently, these localised outbreaks have led to adapted public health surveillance protocols; occupationally exposed veterinary first responders and owners within these European clusters are now integrated into coordinated One Health monitoring systems to detect early signs of mammalian adaptation or zoonotic spillover (Dressler et al., 2026; EFSA, ECDC & EURL, 2026). A chronological overview of major H5N1 infection events reported in domestic cats is provided in Table I, including early cases associated with clade 1 and clade 2.2 viruses and more recent cases associated with clade 2.3.4.4b. This overview highlights how the epidemiology of feline H5N1 infection has changed over time.

Predation and environmental exposure

The primary and historically documented route of transmission to domestic felids remains direct predatory behaviour and the consumption of infected avian tissues. Free-roaming or outdoor cats contract the virus through hunting and subsequent ingestion of infected wild birds or domestic poultry, a mechanism that exposes the oral and gastric mucosa to a high viral load (Kuiken et al., 2004). This classic pathway is supported by recent field data from European micro-outbreaks, where scavenging on infected carcasses in rural holdings was associated with acute, fatal infections in resident cats (Dressler et al., 2026). Beyond direct consumption of infected prey, environmental contamination has emerged as a potential indirect route of exposure. HPAI H5N1 viruses can remain infectious in aquatic environments and organic matrices for several weeks, depending on ambient temperature and moisture levels (Brown et al., 2009). This environmental persistence may allow indirect exposure to shared water sources or environments heavily contaminated with the faeces and secretions of migratory waterfowl, even in the absence of successful hunting or direct bird contact (EFSA, ECDC & EURL, 2024; Nazir et al., 2011).

Lactogenic transmission and the bovine-feline interface

A recently recognised transmission route was documented during the widespread spillover of genotype B3.13 into dairy cattle populations in the United States. On affected dairy farms, cats kept for pest control developed severe disease characterised by hyperacute systemic infection and rapid death (Burrough et al., 2024). Epidemiological investigations and tissue analyses supported unpasteurised raw milk as the main suspected exposure route. The virus

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replicates to high viral titres within the bovine mammary gland and is shed directly into the lipid fraction of the milk. Ingestion of contaminated raw milk may expose the oral and gastrointestinal mucosa to high viral loads, facilitating systemic infection in susceptible cats (Burrough et al., 2024). This lactogenic pathway represents a significant shift in influenza ecology, demonstrating that untreated agricultural products can serve as transmission vehicles between domestic mammals within shared farming ecosystems. It also raises biosecurity concerns regarding the possible introduction of this specific US cattle genotype into European dairy herds, where the close proximity of livestock and companion felids could favour similar spillover events to those observed in North America (EFSA AHAW Panel et al., 2025).

Horizontal shedding and zoonotic implications

Although the domestic cat has traditionally been viewed as an epidemiological “dead-end” host for avian influenza, experimental infection models have shown that H5N1-infected felids can shed virus through both respiratory and digestive routes, and transmission to naïve in-contact cats has been demonstrated (Kuiken et al., 2004). This capacity for horizontal transmission may be relevant in high-density settings such as animal shelters, rescue centres, or veterinary clinics. Domestic cats may act as epidemiological links in shared environments where atypical influenza viruses circulate. Serological data from Southern Europe reported exposure and seroconversion in companion felids against non-feline influenza lineages, suggesting exposure at the animal-human interface (Trombetta et al., 2026). These findings indicate that companion animals may be exposed to influenza viruses circulating in other host species, supporting their inclusion among relevant targets for One Health influenza surveillance. Furthermore, the high viral load present in the saliva, ocular discharge, and respiratory droplets of infected cats poses a potential zoonotic risk to humans in close contact. This concern is supported by the report of serological evidence of H5N1 infection in a veterinary professional following unprotected occupational exposure to infected domestic cats during clinical examination (Vaughan et al., 2026). These findings support managing suspected or confirmed infected cats as potential zoonotic sources requiring appropriate infection-control measures.

Pathogenesis and clinical manifestations

The pathogenesis of HPAI H5N1 in domestic felids is characterised by rapid, multi-organ systemic involvement, differing from the predominantly respiratory tropism typical of seasonal influenza viruses (Kuiken et al., 2004; Rimmelzwaan et al., 2006; van Riel et al., 2007). Following entry via the respiratory or gastrointestinal mucosa, the virus undergoes localised replication before crossing epithelial barriers to reach the cardiovascular and lymphatic systems. This systemic dissemination is facilitated by the broad tissue distribution of host cellular proteases capable of cleaving the polybasic haemagglutinin cleavage site characteristic of HPAI strains, allowing entry into a broad range of cell types (Rimmelzwaan et al., 2006).

Experimental and pathological data indicate that HPAI H5N1 can disseminate systemically in cats and reach the central nervous system, where viral replication is associated with severe neurological disease (Butt et al., 2026; Chothe et al., 2025; Reperant et al., 2012; Rimmelzwaan et al., 2006). This haematogenous route of neuroinvasion explains the rapid onset of neurological signs, including ataxia, tremors, and blindness, which frequently culminate in fatal outcomes in affected animals. Extensive screening and pathological evaluations of companion and stray felids exposed during recent European outbreaks support neuroinvasion as an important contributor to acute mortality in the field (Duijvestijn et al., 2024). Alternatively, neuroinvasion can occur through retrograde axonal transport along the olfactory and trigeminal pathways following the ingestion or inhalation of viral particles (Reperant et al., 2012). Molecular and genomic analyses of recent mammalian spillover events have shown that adaptive mutations, such as the E627K substitution in the PB2 protein, may be detected in feline isolates and may enhance viral replication efficiency in mammalian tissues, including the central nervous system (Burrough et al., 2024; Domańska-Blicharz et al., 2023). Once within the neural parenchyma, the virus is associated with acute necrotising encephalitis and with the onset of severe neurological signs, including tremors, generalised seizures, severe ataxia, circling and rapid recumbency (Frymus et al., 2021).

Among the prominent clinical features of feline HPAI H5N1 infection, acute bilateral blindness (amaurosis) has emerged as an important diagnostic sentinel. Rather than a secondary feature of general systemic decline, this ocular involvement is a direct consequence of localised viral replication within the sensory structures. The virus gains entry to the eye either via the haematogenous route across the blood-retinal barrier or through direct anatomical extension along the optic pathway from the infected central nervous system (Reperant et al., 2012). Pathologically, this localised viral replication results in severe necrotising chorioretinitis and optic neuritis. The virus targets the neural retinal layers,

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causing rapid retinal degeneration, intraocular inflammation and acute dysfunction of the optic nerve (Frymus et al., 2021). Clinically, affected felids present with fixed, mydriatic pupils that completely lack pupillary light reflexes (PLR), a sign that frequently precedes or accompanies the onset of advanced neurological disease (Frymus et al., 2021; Guan et al.,2004). This clinical presentation provides evidence of systemic viral spread and may represent an important warning sign for veterinary clinicians when evaluating cats with acute neurological or ocular disease.

While neurotropic manifestations are clinically dominant, the respiratory tract remains a primary site of viral replication and severe damage. Pulmonary involvement is characterised by severe diffuse alveolar damage (DAD) and necrotising bronchiolitis (Korteweg et al., 2008). The virus targets type I and type II pneumocytes, leading to an extensive loss of alveolar architecture, intra-alveolar oedema, and the formation of hyaline membranes (Rimmelzwaan et al., 2006). This cellular destruction may trigger local and systemic immune dysregulation, commonly referred to as a "cytokine storm," characterised by the uncontrolled release of pro-inflammatory cytokines. Clinically, this manifests as acute respiratory distress syndrome (ARDS), with affected cats exhibiting severe dyspnoea, tachypnoea, and the production of a serosanguineous nasal discharge (Frymus et al., 2021; Kuiken et al., 2004).

Severe disease may progress to multi-organ dysfunction associated with widespread endotheliotropism and systemic microvascular injury. Beyond the brain and lungs, currently circulating clade 2.3.4.4b viruses can disseminate to multiple peripheral tissues. Pathological investigations in infected mammalian carnivores have demonstrated systemic viral distribution involving several organs, including the lungs, brain, liver, and other visceral tissues (Agüero et al., 2023; Burrough et al., 2024). The clinical progression can be rapid and is often associated with fatal outcomes in clinically affected cats; infected individuals may progress from localised respiratory shedding and pyrexia to visceral involvement, vascular damage and terminal recumbency, with fatal disease reported in naturally and experimentally infected felids (Thiry et al., 2007).

Diagnosis and differential diagnosis

Given the hyperacute clinical onset, multi-organ tropism and rapid disease progression of this virus in domestic felids, the immediate establishment of a structured in-clinic triage protocol is important for veterinary practitioners (Burrough et al., 2024; EFSA, ECDC & EURL, 2025; Thiry et al., 2009). The early clinical manifestations of currently circulating clade 2.3.4.4b strains are often non-specific and may overlap with those observed in several feline infectious and neurological diseases. Therefore, signs such as lethargy, anorexia, and fever alone are insufficient to support a clinical suspicion of H5N1 infection (Burrough et al., 2024; Chothe et al., 2025; Domańska-Blicharz et al., 2023; EFSA, ECDC & EURL, 2025).

Attention should be paid to the association between acute ocular signs and rapidly progressive neurological disease, especially bilateral amaurosis, fixed mydriatic pupils, absent pupillary light reflexes, tremors, ataxia, circling or seizures (Domańska-Blicharz et al., 2023; Frymus et al., 2021; Reperant et al., 2012; Thiry et al., 2009). When these findings occur together with pyrexia, respiratory distress or a compatible exposure history, H5N1 infection should be considered among the principal differential diagnoses (Chothe et al., 2025; Kuiken et al., 2004; Rimmelzwaan et al., 2006). This syndromic presentation may be particularly relevant in multi-cat environments or when the clinical history reveals contact with wild birds, poultry, raw animal products or unpasteurised milk (Burrough et al., 2024; Domańska-Blicharz et al., 2023; Dressler et al., 2026; Vaughan et al., 2026).

In the clinical setting, an important differential diagnosis is the neurological and ocular form of feline infectious peritonitis (FIP), which may present with chorioretinitis, uveitis, cranial nerve deficits, and progressive ataxia that can resemble some of the clinical manifestations observed in H5N1-infected cats (Addie et al., 2009; Pedersen et al., 2009; Vasinioti et al., 2026). However, dry FIP typically follows a subacute to chronic clinical course that develops over weeks to months and generally occurs as sporadic individual cases, in contrast to the rapid progression and high mortality reported during recent H5N1 outbreaks in domestic cats (Burrough et al., 2024; Domańska-Blicharz et al., 2023; Pedersen et al., 2009; Vasinioti et al., 2026). Similarly, acute or reactivated neurotoxoplasmosis caused by *Toxoplasma gondii* can induce severe localised myelitis, focal chorioretinitis, and central nervous system disturbances that may resemble influenza-associated encephalopathy, but it is not typically associated with severe necrotising diffuse alveolar damage or with clusters of fatal cases involving multiple animals, as reported in domestic feline populations exposed to currently circulating HPAI H5N1 genotypes (Chothe et al., 2025; Kuiken et al., 2004; Lindsay et al., 2010; Reperant et al., 2012).

Rabies should also be considered among the differential diagnoses in cats presenting with acute neurological signs. As in H5N1 infection, affected animals may exhibit behavioural abnormalities, ataxia, tremors, paralysis, and rapidly progressive neurological deterioration (Gunn-Moore, 2005). However, rabies is not typically associated with the

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severe respiratory disease, acute bilateral amaurosis, or epidemiological clustering that have been reported during recent H5N1 outbreaks in domestic cats (Burrough et al., 2024; Domańska-Blicharz et al., 2023; Gunn-Moore, 2005).

Other differential diagnoses include Aujeszky's disease (pseudorabies) and feline bornavirus infection. Although both conditions may cause severe neurological signs, including behavioural abnormalities and ataxia, pseudorabies is typically characterised by intense pruritus and rapid clinical deterioration, whereas feline bornavirus infection generally follows a more progressive clinical course. In contrast, H5N1 infection is frequently associated with concurrent respiratory involvement and severe pulmonary lesions in addition to neurological disease (Gunn-Moore, 2005; Lutz et al., 2015; Thiry et al., 2013).

A structured clinical triage approach integrating characteristic ocular lesions, rapidly progressive neurological signs, and a thorough exposure history, particularly regarding contact with wild birds, raw poultry products, or unpasteurised milk, may facilitate the early recognition of suspected H5N1 cases. Early identification of potentially infected animals is important not only for timely diagnostic investigation but also for the implementation of appropriate infection-control measures within veterinary facilities and the protection of veterinary personnel potentially exposed to infected animals (Burrough et al., 2024; EFSA, ECDC & EURL, 2025; Thiry et al., 2009).

Following clinical suspicion, confirmation of H5N1 infection should be based on molecular testing, most commonly reverse-transcription quantitative PCR (RT-qPCR) assays targeting conserved influenza A matrix gene regions, followed by H5-specific testing, pathotyping and sequencing when appropriate (Hoffmann et al., 2007). In live cats, sample selection should be guided by the clinical presentation and exposure history. Nasal and oropharyngeal swabs are recommended in animals with respiratory signs or suspected recent exposure. Conjunctival swabs should be considered when ocular involvement is present, particularly in cats showing acute blindness, conjunctivitis, ocular discharge or other signs suggestive of ocular viral replication. Rectal swabs may be useful when gastrointestinal involvement or faecal shedding is suspected, although negative results from a single anatomical site should not exclude infection in strongly suspected cases. Whenever possible, sampling from more than one site may increase diagnostic sensitivity, particularly in animals with systemic or neurological disease (Mainenti et al., 2025; Schlachter et al., 2025; Thiry et al., 2009).

For post-mortem diagnosis, RT-qPCR should be performed on fresh tissues collected from organs showing gross or histopathological lesions. Given the marked neurotropism and pneumotropism of H5N1 in domestic cats, brain and lung should be prioritised, particularly in animals with neurological or respiratory signs. Additional tissues, including the trachea, spleen, liver, kidneys, heart, intestines and other affected organs, may be collected when systemic infection is suspected. Samples should be submitted in accordance with national biosafety and transport requirements, and diagnostic laboratories should be contacted before shipment when HPAI H5N1 is suspected (Acevedo et al., 2026; Mainenti et al., 2025). Histopathology and immunohistochemistry using antibodies against influenza A viral antigens remain important complementary tools for confirming active tissue infection and to distinguishing systemic viral replication from passive contamination or incidental viral carriage (Acevedo et al., 2026; Kuiken et al., 2004; Reperant et al., 2012; Schlachter et al., 2025; van Riel et al., 2007).

Biosafety considerations in veterinary practice

Following initial syndromic identification, biosafety measures should be adapted to the level of clinical suspicion, the available infrastructure and the procedures that need to be performed. In referral centres or veterinary hospitals with adequate facilities, cats with suspected HPAI H5N1 infection should ideally be managed in a dedicated isolation area, preferably with negative-pressure ventilation or high-efficiency particulate air (HEPA) filtration. Access should be restricted to trained personnel using appropriate personal protective equipment (PPE), including a fit-tested FFP2/FFP3 or N95 respirator, protective eyewear or a face shield, a fluid-resistant long-sleeved gown and disposable gloves. Dedicated equipment should be used whenever possible, and aerosol-generating procedures should be avoided unless clinically necessary and performed with appropriate respiratory protection (Domańska-Blicharz et al., 2023; Stull et al., 2018; Williams et al., 2015).

In general veterinary practice, where negative-pressure rooms or specialised isolation facilities may not be available, minimum practical measures should still be implemented as soon as H5N1 infection is suspected. The animal should be placed in a separate room away from other patients and clients, handling should be limited to essential procedures, and the number of exposed staff members should be kept as low as possible. Non-urgent procedures should be postponed until appropriate PPE is available or the case has been discussed with the competent veterinary authority or diagnostic laboratory. At a minimum, staff involved in examination or sampling should use disposable gloves, protective outerwear, eye protection and suitable respiratory protection according to national guidance. Strict hand

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hygiene should be practised before and after animal contact.

Environmental management should include the removal of organic material before disinfection, because secretions, faeces and other biological material may reduce disinfectant efficacy. Surfaces, cages, instruments and contaminated areas should be disinfected using products active against enveloped viruses, such as sodium hypochlorite, potassium peroxymonosulfate or accelerated hydrogen peroxide, following the manufacturer's recommended dilution and contact time. Materials contaminated with respiratory, ocular or faecal secretions should be handled as potentially infectious. Suspected or confirmed cases should be reported according to national regulations, and sample shipment or referral should be coordinated with the diagnostic laboratory and competent authorities (De Marco et al., 2021; EPA, 2023; Khalil et al., 2023; Sehulster et al., 2003; Suarez et al., 2003; Williams et al., 2015).

One Health implications

Given the zoonotic and transboundary nature of HPAI H5N1, suspected or confirmed infections in domestic cats should be promptly reported to the competent veterinary and public health authorities in accordance with national regulations and international surveillance frameworks (EFSA, ECDC & EURL, 2025; European Commission, 2020; WOAH, 2025). Domestic cats could be integrated into H5N1 surveillance systems through both event-based and targeted approaches. In general veterinary practice, event-based surveillance should rely on the prompt recognition and reporting of compatible clinical presentations, particularly acute neurological disease, ocular involvement, severe respiratory signs, or unexplained clusters of fatal disease in cats with a history of exposure to wild birds, poultry, raw poultry products, raw pet food, or unpasteurised milk.

Targeted surveillance may be considered in high-risk settings where feline exposure is more likely. These include poultry holdings affected by HPAI H5N1, dairy farms with confirmed or suspected H5N1 infection in cattle, shelters or multi-cat environments located in areas with intense wild bird mortality, and households or colonies with known exposure to raw animal products or contaminated environments. In these contexts, domestic cats may act as sentinels of local viral circulation or cross-species spillover. Surveillance should not be limited to laboratory confirmation of individual feline cases, but should also collect information on exposure history, feeding practices, contact with livestock or wild birds, clinical outcome, and possible human exposure.

A practical One Health surveillance pathway should include early notification by veterinarians, coordinated sampling and testing through authorised diagnostic laboratories, and timely communication of results to animal health and public health authorities. When H5N1 infection is confirmed or strongly suspected in a cat, epidemiological investigation should assess possible links with infected birds, dairy cattle, raw milk, raw pet food, or other contaminated animal products. Public health follow-up may include monitoring owners, veterinary personnel, and other close contacts for respiratory or ocular symptoms, according to national guidance (CDC, 2026). Integrating feline diagnostic results with wildlife, poultry, livestock and human health data could improve the early detection of spillover events and support more timely risk assessment at the animal-human interface.

Conclusion and future perspectives

HPAI H5N1 infection in domestic cats has gained increasing relevance during the current panzootic, particularly because of the expanding host range of clade 2.3.4.4b viruses and the occurrence of feline cases linked to different exposure pathways. Domestic cats should therefore be considered in H5N1 risk assessment, especially in contexts involving contact with infected birds, contaminated animal products or affected livestock environments (Burrough et al., 2024; Domańska-Blicharz et al., 2023; Dressler et al., 2026; EFSA, ECDC & EURL, 2025; Vaughan et al., 2026). To mitigate the risk of further outbreaks in companion animals and subsequent zoonotic spillover, the implementation of rapid, in-clinic syndromic triage represents an important component of early detection and risk mitigation strategies. Veterinarians play a key role in the early recognition of suspected H5N1 infections, as the prompt identification of characteristic ocular and neurological manifestations may support timely infection-control measures and contribute to reducing the risk of zoonotic exposure. Strengthening this approach requires heightened clinical vigilance, access to rapid molecular diagnostics, and the consistent implementation of infection-prevention and biosafety measures within veterinary facilities (Burrough et al., 2024; Chothe et al., 2025; Stull et al., 2018). Delaying isolation until laboratory confirmation may increase the risk of hospital-associated transmission and occupational exposure among veterinary staff. A One Health approach is essential for the surveillance and management of H5N1. Improved monitoring of domestic cats, particularly in peri-agricultural and dairy-associated environments, together with effective information exchange between veterinary, environmental, and public health authorities, may contribute to the early detection of emerging outbreaks (EFSA, ECDC & EURL, 2026; WHO, 2025; WOAH, 2026). Future research should focus on the

How to cite: Salvaggiulo et al. Highly pathogenic avian influenza A(H5N1) in domestic cats: epidemiology, clinical manifestations, and One Health implication. Veterinaria Italiana, Vol. 62 No. 3 (2026) DOI: 10.12834/VetIt.4040.41131.2

development of effective vaccination strategies for susceptible mammalian hosts and on the continued monitoring of genetic markers associated with mammalian adaptation. Strengthening surveillance activities and maintaining close collaboration between veterinary and public health sectors will be important for improving outbreak detection and mitigating the impact of future H5N1 incursions.

Year	Country	H5N1 clade/genotype	Exposure route/source	Level of evidence	Main findings	Reference
2004	Thailand	Clade 1	Contact with or ingestion of infected poultry	Probable	First documented natural H5N1 infection in domestic cats; severe systemic disease and high mortality.	Kuiken et al., 2004
2006	Austria	Clade 2.2	Exposure to infected wild birds in an affected area	Probable	First European feline cases; evidence of subclinical infection in exposed cats.	Leschnik et al., 2007
2022	France	Clade 2.3.4.4b	Predation or scavenging of infected wild birds	Probable	First confirmed feline case reported during the current global H5N1 panzootic in Europe.	Briand et al., 2023
2023	Poland	Clade 2.3.4.4b	Contaminated raw poultry products	Suspected	Multiregional outbreak involving indoor and outdoor cats; high mortality and evidence of mammalian-adaptive mutations.	Domańska-Blicharz et al., 2023
2023	Netherlands	Clade 2.3.4.4b	Exposure in areas with intense H5N1 circulation in wild birds	Undetermined / evidence of exposure only	Serological and virological evidence of H5N1 exposure among domestic and stray cats.	Duijvestijn et al., 2024
2024	United States	Clade 2.3.4.4b genotype B3.13	Ingestion of unpasteurised milk from infected dairy cattle	Probable	Fatal outbreaks in farm cats; identification of a bovine-associated feline exposure pathway.	Burrough et al., 2024
2024–2025	United States, California	Clade 2.3.4.4b genotype B3.13	Raw animal products and/or unpasteurised milk, depending on the cluster investigated	Suspected or probable	Cluster of infections in indoor cats; occupational exposure of veterinary personnel documented.	Vaughan et al., 2026
2026	Germany, Sigmaringen	Clade 2.3.4.4b	Predation or scavenging of infected poultry	Probable	Simultaneous outbreak in poultry and domestic cats; all surviving cats seropositive; One Health investigation involving exposed humans.	Dressler et al., 2026

Table I. Chronological overview of major HPAI H5N1 infection events reported in domestic cats worldwide (2004–2026), including viral clade or genotype, reported exposure route, level of evidence, and epidemiological significance.

Ethical approval

Not applicable. This article is a review of existing literature and does not involve any new studies with human participants or animals performed by the authors.

Conflict of interest

The authors declare no conflicts of interest.

Author Contributions

Conceptualisation: A.S., N.D. ; Writing – original draft preparation: A.S.; Writing – review and editing: I.P., S.B., M.C., G.G., M.D., N.D.; Visualisation: A.S.; Supervision: N.D.; All authors have read and agreed to the published version of the manuscript and are accountable for all aspects of the work.

Funding

This research received no external funding.

References

Acevedo, H. D., Beeler, E., Crossley, B., Armién, A. G., Henderson, E. E., Asin, J., Middleton, J., & Uzal, F. A. (2026). Salpingitis and multiorgan lesions caused by highly pathogenic avian influenza A(H5N1) virus in a cat associated with consumption of recalled raw milk in California. Journal of veterinary diagnostic investigation : official publication of the American Association of Veterinary Laboratory Diagnosticians, Inc, 10406387251413563. Advance online publication. https://doi.org/10.1177/10406387251413563.

Addie, D., Belák, S., Boucraut-Baralon, C., Egberink, H., Frymus, T., Gruffydd-Jones, T., Hartmann, K., Hosie, M. J., Lloret, A., Lutz, H., Marsilio, F., Pennisi, M. G., Radford, A. D., Thiry, E., Truyen, U., & Horzinek, M. C. (2009). Feline infectious peritonitis. ABCD guidelines on prevention and management. Journal of feline medicine and surgery, 11(7), 594–604. https://doi.org/10.1016/j.jfms.2009.05.008.

Agüero, M., Monne, I., Sánchez, A., Zecchin, B., Fusaro, A., Ruano, M. J., Del Valle Arrojo, M., Fernández-Antonio, R., Souto, A. M., Tordable, P., Cañás, J., Bonfante, F., Giussani, E., Terregino, C., & Orejas, J. J. (2023). Highly pathogenic avian influenza A(H5N1) virus infection in farmed minks, Spain, October 2022. Euro surveillance : bulletin Europeen sur les maladies transmissibles = European communicable disease bulletin, 28(3), 2300001. https://doi.org/10.2807/1560-7917.ES.2023.28.3.2300001.

Bonilla-Aldana, D. K., Bonilla-Aldana, J. L., Acosta-España, J. D., & Rodriguez-Morales, A. J. (2025). Highly Pathogenic Avian Influenza H5N1 in Cats (Felis catus): A Systematic Review and Meta-Analysis. Animals, 15(10), 1441. https://doi.org/10.3390/ani15101441.

Borland, S., Gracieux, P., Jones, M., Mallet, F., & Yugueros-Marcos, J. (2020). Influenza A Virus Infection in Cats and Dogs: A Literature Review in the Light of the "One Health" Concept. Frontiers in public health, 8, 83. https://doi.org/10.3389/fpubh.2020.00083.

Briand, F. X., Souchaud, F., Pierre, I., Beven, V., Hirchaud, E., Hérault, F., Planel, R., Rigaudeau, A., Bernard-Stoecklin, S., Van der Werf, S., Lina, B., Gerbier, G., Eterradossi, N., Schmitz, A., Niqueux, E., & Grasland, B. (2023). Highly Pathogenic Avian Influenza A(H5N1) Clade 2.3.4.4b Virus in Domestic Cat, France, 2022. Emerging infectious diseases, 29(8), 1696–1698. https://doi.org/10.3201/eid2908.230188.

Brown, J. D., Goekjian, G., Poulson, R., Valeika, S., & Stallknecht, D. E. (2009). Avian influenza virus in water: infectivity is dependent on pH, salinity and temperature. Veterinary microbiology, 136(1-2), 20–26. https://doi.org/10.1016/j.vetmic.2008.10.027.

Burrough, E. R., Magstadt, D. R., Petersen, B., Timmermans, S. J., Gauger, P. C., Zhang, J., Siepker, C., Mainenti, M., Li, G., Thompson, A. C., Gorden, P. J., Plummer, P. J., & Main, R. (2024). Highly Pathogenic Avian Influenza A(H5N1) Clade 2.3.4.4b Virus Infection in Domestic Dairy Cattle and Cats, United States, 2024. Emerging infectious diseases, 30(7), 1335–1343. https://doi.org/10.3201/eid3007.240508.

Butt, S. L., Rani, R., Nooruzzaman, M., Demeter, E. A., de Oliveira, P. S. B., Hitchener, G. R., & Diel, D. G. (2026). Hematogenous neuroinvasion and genotype-dependent transmission of influenza A H5N1 viruses in the cat host. bioRxiv [Preprint]. https://doi.org/10.64898/2026.02.21.707182.

CDC. (2026). Interim Recommendations for Prevention, Monitoring, and Public Health Investigations of Highly Pathogenic Avian Influenza A(H5N1) Virus Infections in People. Centers for Disease Control and Prevention, February 2026. https://www.cdc.gov/bird-flu/prevention/hpai-interim-recommendations.html.

Chothe, S. K., Srinivas, S., Misra, S., Nallipogu, N. C., Gilbride, E., LaBella, L., Mukherjee, S., Gauthier, C. H., Pecoraro, H. L., Webb, B. T., Pipas, J. M., Ramasamy, S., & Kuchipudi, S. V. (2024). Marked neurotropism and potential adaptation of H5N1 clade 2.3.4.4.b virus in naturally infected domestic cats. Emerging microbes & infections, 14(1), 2440498. https://doi.org/10.1080/22221751.2024.2440498.

Connor, R. J., Kawaoka, Y., Webster, R. G., & Paulson, J. C. (1994). Receptor specificity in human, avian, and equine H2 and H3 influenza virus isolates. Virology, 205(1), 17–23. https://doi.org/10.1006/viro.1994.1615.

De Marco, M. A., Delogu, M., Facchini, M., Di Trani, L., Boni, A., Cotti, C., Graziosi, G., Venturini, D., Regazzi, D.,

Ravaioli, V., Marzadori, F., Frasnelli, M., Castrucci, M. R., & Raffini, E. (2021). Serologic Evidence of Occupational Exposure to Avian Influenza Viruses at the Wildfowl/Poultry/Human Interface. Microorganisms, 9(10), 2153. https://doi.org/10.3390/microorganisms9102153.

Domańska-Blicharz, K., Świętoń, E., Świątalska, A., Monne, I., Fusaro, A., Tarasiuk, K., Wyrostek, K., Styś-Fijoł, N., Giza, A., Pietruk, M., Zecchin, B., Pastori, A., Adaszek, Ł., Pomorska-Mól, M., Tomczyk, G., Terregino, C., & Winiarczyk, S. (2023). Outbreak of highly pathogenic avian influenza A(H5N1) clade 2.3.4.4b virus in cats, Poland, June to July 2023. Euro surveillance : bulletin Europeen sur les maladies transmissibles = European communicable disease bulletin, 28(31), 2300366. https://doi.org/10.2807/1560-7917.ES.2023.28.31.2300366.

Dressler, A., Wagner-Wiening, C., Tegtmeyer, B., Haag-Milz, S., Demattio, B., Dürrwald, R., Harder, T., Salditt, A., & Köster, J. (2026). Highly pathogenic avian influenza A(H5N1) in poultry and domestic cats and occupational exposure among veterinary and other first responders, Germany, February 2026. Euro surveillance : bulletin Europeen sur les maladies transmissibles = European communicable disease bulletin, 31(17), 10.2807/1560-7917.ES.2026.31.17.2600293. https://doi.org/10.2807/1560-7917.ES.2026.31.17.2600293.

Duijvestijn, M. B. H. M., Schuurman, N. N. M. P., Vernooij, J. C. M., van Leeuwen, M. A. J. M., van den Brand, J. M. A., Wagenaar, J. A., van Kuppeveld, F. J. M., Egberink, H. F., de Haan, C. A. M., & Verhagen, J. H. (2024). Highly pathogenic avian influenza (HPAI) H5 virus exposure in domestic cats and rural stray cats, the Netherlands, October 2020 to June 2023. Euro surveillance : bulletin Europeen sur les maladies transmissibles = European communicable disease bulletin, 29(44), 2400326. https://doi.org/10.2807/1560-7917.ES.2024.29.44.2400326.

European Food Safety Authority, European Centre for Disease Prevention and Control, European Union Reference Laboratory for Avian Influenza, Alexakis, L., Fusaro, A., Kuiken, T., Mirinavičiūtė, G., Ståhl, K., Staubach, C., Svartström, O., Terregino, C., Willgert, K., Delacourt, R., Goudjihounde, S. M., Grant, M., Tampach, S., & Kohnle, L. (2024). Avian influenza overview March-June 2024. EFSA journal. European Food Safety Authority, 22(7), e8930. https://doi.org/10.2903/j.efsa.2024.8930.

European Food Safety Authority, European Centre for Disease Prevention and Control, European Union Reference Laboratory for Avian Influenza, Buczkowski, H., Ducatez, M., Fusaro, A., Gonzales, J. L., Kuiken, T., Mirinavičiūtė, G., Ståhl, K., Staubach, C., Svartström, O., Terregino, C., Willgert, K., Alarcón, E., & Kohnle, L. (2025). Avian influenza overview September-November 2025. EFSA journal. European Food Safety Authority, 23(12), e9834. https://doi.org/10.2903/j.efsa.2025.9834.

EFSA Panel on Animal Health and Welfare (AHAW), Alvarez, J., Boklund, A. E., Dippel, S., Dórea, F., Figuerola, J., Herskin, M. S., Michel, V., Miranda Chueca, M. Á., Nannoni, E., Nielsen, S. S., Nonno, R., Riber, A. B., Stegeman, J. A., Ståhl, K., Thulke, H. H., Tuyttens, F., Winckler, C., Bortolami, A., Ducatez, M., … Gervelmeyer, A. (2025). Risk of infection of dairy cattle in the EU with highly pathogenic avian influenza virus affecting dairy cows in the United States of America (H5N1, Eurasian lineage goose/Guangdong clade 2.3.4.4b. genotype B3.13). EFSA journal. European Food Safety Authority, 23(12), e9801. https://doi.org/10.2903/j.efsa.2025.9801.

EPA. (2023). U.S. Environmental Protection Agency: Registered antimicrobial products with label claims for avian influenza A viruses (List M). EPA Office of Pesticide Programs, 1–8. https://www.epa.gov/pesticide-registration/epas-registered-antimicrobial-products-effective-against-avian-influenza.

European Commission. (2020). Commission Implementing Regulation (EU) 2020/2002 of 7 December 2020 laying down rules for the application of Regulation (EU) 2016/429 of the European Parliament and of the Council with regard to disease notification and reporting of listed diseases. *Official Journal of the European Union*, L 412, 1–28. https://eur-lex.europa.eu/eli/reg_impl/2020/2002/oj/eng.

European Food Safety Authority (EFSA), European Centre for Disease Prevention and Control (ECDC), European Union Reference Laboratory for Avian Influenza (EURL), Barbezange, C., Buczkowski, H., Ducatez, M., Fusaro, A., Gonzales, J. L., Kuiken, T., Mirinavičiūtė, G., Ståhl, K., Staubach, C., Svartström, O., Terregino, C., Willgert, K., Alarcón, E., & Kohnle, L. (2026). Avian influenza overview December 2025-February 2026. EFSA journal. European Food Safety Authority, 24(3), e10015. https://doi.org/10.2903/j.efsa.2026.10015.

European Parliament, & Council. (2009). Regulation (EC) No 1069/2009 of the European Parliament and of the Council of 21 October 2009 laying down health rules as regards animal by-products and derived products not

intended for human consumption. *Official Journal of the European Union*, L 300, 1–33. https://eur-lex.europa.eu/eli/reg/2009/1069/oj/eng.

Frymus, T., Belák, S., Egberink, H., Hofmann-Lehmann, R., Marsilio, F., Addie, D. D., Boucraut-Baralon, C., Hartmann, K., Lloret, A., Lutz, H., Pennisi, M. G., Thiry, E., Truyen, U., Tasker, S., Möstl, K., & Hosie, M. J. (2021). Influenza Virus Infections in Cats. Viruses, 13(8), 1435. https://doi.org/10.3390/v13081435.

Guan, Y., Poon, L. L., Cheung, C. Y., Ellis, T. M., Lim, W., Lipatov, A. S., Chan, K. H., Sturm-Ramirez, K. M., Cheung, C. L., Leung, Y. H., Yuen, K. Y., Webster, R. G., & Peiris, J. S. (2004). H5N1 influenza: a protean pandemic threat. Proceedings of the National Academy of Sciences of the United States of America, 101(21), 8156–8161. https://doi.org/10.1073/pnas.0402443101.

Gunn-Moore D. (2005). Infectious diseases of the central nervous system. The Veterinary clinics of North America. Small animal practice, 35(1), 103–vi. https://doi.org/10.1016/j.cvsm.2004.09.003.

Hatta, M., Gao, P., Halfmann, P., & Kawaoka, Y. (2001). Molecular basis for high virulence of Hong Kong H5N1 influenza A viruses. Science (New York, N.Y.), 293(5536), 1840–1842. https://doi.org/10.1126/science.1062882.

Hoffmann, B., Harder, T., Starick, E., Depner, K., Werner, O., & Beer, M. (2007). Rapid and highly sensitive pathotyping of avian influenza A H5N1 virus by using real-time reverse transcription-PCR. Journal of clinical microbiology, 45(2), 600–603. https://doi.org/10.1128/JCM.01681-06.

International Committee on Taxonomy of Viruses (ICTV). (2025). ICTV taxonomy browser. Retrieved June 12, 2026, from https://ictv.global/taxonomy.

Ito, T., Couceiro, J. N., Kelm, S., Baum, L. G., Krauss, S., Castrucci, M. R., Donatelli, I., Kida, H., Paulson, J. C., Webster, R. G., & Kawaoka, Y. (1998). Molecular basis for the generation in pigs of influenza A viruses with pandemic potential. Journal of virology, 72(9), 7367–7373. https://doi.org/10.1128/JVI.72.9.7367-7373.1998.

Khalil, A. M., Esaki, M., Okuya, K., & Ozawa, M. (2023). Stability of the Virucidal Activity of Commercial Disinfectants against Avian Influenza Viruses under Different Environmental Conditions. Pathogens (Basel, Switzerland), 12(12), 1382. https://doi.org/10.3390/pathogens12121382.

Korteweg, C., & Gu, J. (2008). Pathology, molecular biology, and pathogenesis of avian influenza A (H5N1) infection in humans. The American journal of pathology, 172(5), 1155–1170. https://doi.org/10.2353/ajpath.2008.070791.

Kuchipudi, S. V., Nelli, R. K., Gontu, A., Satyakumar, R., Surendran Nair, M., & Subbiah, M. (2021). Sialic Acid Receptors: The Key to Solving the Enigma of Zoonotic Virus Spillover. Viruses, 13(2), 262. https://doi.org/10.3390/v13020262.

Kuiken, T., Rimmelzwaan, G., van Riel, D., van Amerongen, G., Baars, M., Fouchier, R., & Osterhaus, A. (2004). Avian H5N1 influenza in cats. Science (New York, N.Y.), 306(5694), 241. https://doi.org/10.1126/science.1102287.

Leschnik, M., Weikel, J., Möstl, K., Revilla-Fernández, S., Wodak, E., Bagó, Z., Vanek, E., Benetka, V., Hess, M., & Thalhammer, J. G. (2007). Subclinical infection with avian influenza A (H5N1) virus in cats. Emerging infectious diseases, 13(2), 243–247. https://doi.org/10.3201/eid1302.060608.

Lindsay, S. A., Barrs, V. R., Child, G., Beatty, J. A., & Krockenberger, M. B. (2010). Myelitis due to reactivated spinal toxoplasmosis in a cat. Journal of feline medicine and surgery, 12(10), 818–821. https://doi.org/10.1016/j.jfms.2010.05.002.

Lutz, H., Addie, D. D., Boucraut-Baralon, C., Egberink, H., Frymus, T., Gruffydd-Jones, T., Hartmann, K., Horzinek, M. C., Hosie, M. J., Lloret, A., Marsilio, F., Pennisi, M. G., Radford, A. D., Thiry, E., Truyen, U., Möstl, K., & European Advisory Board on Cat Diseases (2015). Borna disease virus infection in cats: ABCD guidelines on prevention and management. Journal of feline medicine and surgery, 17(7), 614–616. https://doi.org/10.1177/1098612X15588452.

Mainenti, M., Siepker, C., Magstadt, D. R., Gauger, P., Baum, D., Petersen, B., Aubrey, T., Sett, K., & Burrough, E. R. (2025). Distribution of lesions and detection of influenza A(H5N1) virus, clade 2.3.4.4b, in ante- and postmortem

samples from naturally infected domestic cats on U.S. dairy farms. Journal of veterinary diagnostic investigation : official publication of the American Association of Veterinary Laboratory Diagnosticians, Inc, 37(1), 27–35. https://doi.org/10.1177/10406387241300464.

Nazir, J., Haumacher, R., Ike, A. C., & Marschang, R. E. (2011). Persistence of avian influenza viruses in lake sediment, duck feces, and duck meat. Applied and environmental microbiology, 77(14), 4981–4985. https://doi.org/10.1128/AEM.00415-11.

Pedersen, N. C., Liu, H., Dodd, K. A., & Pesavento, P. A. (2009). Significance of coronavirus mutants in feces and diseased tissues of cats suffering from feline infectious peritonitis. Viruses, 1(2), 166–184. https://doi.org/10.3390/v1020166.

Reperant, L. A., van de Bildt, M. W., van Amerongen, G., Leijten, L. M., Watson, S., Palser, A., Kellam, P., Eissens, A. C., Frijlink, H. W., Osterhaus, A. D., & Kuiken, T. (2012). Marked endotheliotropism of highly pathogenic avian influenza virus H5N1 following intestinal inoculation in cats. Journal of virology, 86(2), 1158–1165. https://doi.org/10.1128/JVI.06375-11.

Rimmelzwaan, G. F., van Riel, D., Baars, M., Bestebroer, T. M., van Amerongen, G., Fouchier, R. A., Osterhaus, A. D., & Kuiken, T. (2006). Influenza A virus (H5N1) infection in cats causes systemic disease with potential novel routes of virus spread within and between hosts. The American journal of pathology, 168(1), 176–364. https://doi.org/10.2353/ajpath.2006.050466.

Schlachter, A. D., Bruno-McClung, E., Díaz-Delgado, J., Odom, M., Banyard, A. C., & Núñez, A. (2025). Feline high pathogenicity avian influenza H5N1 infection: past and present. Journal of felinemedicine and surgery, 27(10), 1098612X251370695. https://doi.org/10.1177/1098612X251370695.

Sehulster, L., Chinn, R. Y., CDC, & HICPAC (2003). Guidelines for environmental infection control in health-care facilities. Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR. Recommendations and reports : Morbidity and mortality weekly report. Recommendations and reports, 52(RR-10), 1–42. https://www.cdc.gov/mmwr/preview/mmwrhtml/rr5210a1.htm.

Shinya, K., Ebina, M., Yamada, S., Ono, M., Kasai, N., & Kawaoka, Y. (2006). Avian flu: influenza virus receptors in the human airway. Nature, 440(7083), 435–436. https://doi.org/10.1038/440435a

Stull, J. W., Bjorvik, E., Bub, J., Dvorak, G., Petersen, C., & Troyer, H. L. (2018). 2018 AAHA Infection Control, Prevention, and Biosecurity Guidelines. Journal of the American Animal Hospital Association, 54(6), 297–326. https://doi.org/10.5326/JAAHA-MS-6903.

Suarez, D. L., Spackman, E., Senne, D. A., Bulaga, L., Welsch, A. C., & Froberg, K. (2003). The effect of various disinfectants on detection of avian influenza virus by real time RT-PCR. Avian diseases, 47(3 Suppl), 1091–1095. https://doi.org/10.1637/0005-2086-47.s3.1091.

Subbarao, E. K., London, W., & Murphy, B. R. (1993). A single amino acid in the PB2 gene of influenza A virus is a determinant of host range. Journal of virology, 67(4), 1761–1764. https://doi.org/10.1128/JVI.67.4.1761-1764.1993.

Thiry, E., Zicola, A., Addie, D., Egberink, H., Hartmann, K., Lutz, H., Poulet, H., & Horzinek, M. C. (2007). Highly pathogenic avian influenza H5N1 virus in cats and other carnivores. Veterinary microbiology, 122(1-2), 25–31. https://doi.org/10.1016/j.vetmic.2006.12.021.

Thiry, E., Addie, D., Belák, S., Boucraut-Baralon, C., Egberink, H., Frymus, T., Gruffydd-Jones, T., Hartmann, K., Hosie, M. J., Lloret, A., Lutz, H., Marsilio, F., Pennisi, M. G., Radford, A. D., Truyen, U., & Horzinek, M. C. (2009). H5N1 avian influenza in cats. ABCD guidelines on prevention and management. Journal of feline medicine and surgery, 11(7), 615–618. https://doi.org/10.1016/j.jfms.2009.05.011.

Thiry, E., Addie, D., Belák, S., Boucraut-Baralon, C., Egberink, H., Frymus, T., Gruffydd-Jones, T., Hartmann, K., Hosie, M. J., Lloret, A., Lutz, H., Marsilio, F., Möstl, K., Pennisi, M. G., Radford, A. D., Truyen, U., & Horzinek, M. C. (2013). Aujeszky's disease/pseudorabies in cats: ABCD guidelines on prevention and management. Journal of feline medicine and surgery, 15(7), 555–556. https://doi.org/10.1177/1098612X13489211.

Trombetta, C. M., Fiori, A., Falsini, A., Pellegrini, F., Le Poder, S., Eichenbaum, A., Cardona, V., Oliva, J., Meyer, G., Muzyka, N., Muzyka, D., Montomoli, E., di Martino, B., Ducatez, M. F., Lanave, G., Martella, V., & Camero, M. (2026). Multicenter Serologic Investigation of Influenza D Virus in Cats and Dogs, Europe, 2015-2024. Emerging infectious diseases, 32(2), 293–295. https://doi.org/10.3201/eid3202.251164.

van Riel, D., Munster, V. J., de Wit, E., Rimmelzwaan, G. F., Fouchier, R. A., Osterhaus, A. D., & Kuiken, T. (2007). Human and avian influenza viruses target different cells in the lower respiratory tract of humans and other mammals. The American journal of pathology, 171(4), 1215–1223. https://doi.org/10.2353/ajpath.2007.070248.

Vasinioti, V. I., Lucente, M. S., Catella, C., Buonavoglia, C., Decaro, N., Pratelli, A., & Capozza, P. (2026). Feline Infectious Peritonitis: A Challenging Diagnostic and Therapeutic Labyrinth. Animals : an open access journal from MDPI, 16(1), 128. https://doi.org/10.3390/ani16010128.

Vaughan, A., Joyce, A., Traub, E., Jae, M., Beeler, E., Paiva, E., Ananian, K., Holiday, C., Jefferson, S., Richardson, J., Munna, C., Chan, C., Scott, T., Kojima, N., Seneviratne, T., Mellis, A., Olsen, S. J., Green, N., Feaster, M., Terashita, D., … de St Maurice, A. (2026). Serologic Evidence of Highly Pathogenic Avian Influenza A(H5N1) Virus Infection in a Veterinary Professional Exposed to an Infected Domestic Cat - Los Angeles County, California, December 2024-January 2025. MMWR. Morbidity and mortality weekly report, 75(17), 215–220. https://doi.org/10.15585/mmwr.mm7517a1.

Wang, H., Wu, X., Cheng, Y., An, Y., & Ning, Z. (2013). Tissue distribution of human and avian type sialic acid influenza virus receptors in domestic cat. Acta veterinaria Hungarica, 61(4), 537–546. https://doi.org/10.1556/AVet.2013.030.

Webster, R. G., Bean, W. J., Gorman, O. T., Chambers, T. M., & Kawaoka, Y. (1992). Evolution and ecology of influenza A viruses. Microbiological reviews, 56(1), 152–179. https://doi.org/10.1128/mr.56.1.152-179.1992.

Williams, C. J., Scheftel, J. M., Elchos, B. L., Hopkins, S. G., & Levine, J. F. (2015). Compendium of Veterinary Standard Precautions for Zoonotic Disease Prevention in Veterinary Personnel: National Association of State Public Health Veterinarians: Veterinary Infection Control Committee 2015. Journal of the American Veterinary Medical Association, 247(11), 1252–1277. https://doi.org/10.2460/javma.247.11.1252.

WOAH. (2023). Rabies (infection with rabies virus and other lyssaviruses). In Manual of diagnostic tests and vaccines for terrestrial animals (Chap. 3.1.18). WOAH. https://www.woah.org/fileadmin/Home/eng/Health_standards/tahm/3.01.18_RABIES.pdf.

WOAH. (2025). World Organisation for Animal Health: Terrestrial Animal Health Code – Chapter 10.4: Infection with high pathogenicity avian influenza viruses. WOAH Paris, 112–124. https://www.woah.org/fileadmin/Home/eng/Health_standards/tahc/current/en_chapitre_avian_influenza_viruses.htm.

WOAH. (2026). World Organisation for Animal Health: High pathogenicity avian influenza (HPAI) - Situation Report 80. WOAH Global Avian Influenza Surveillance Data, February 2026. https://www.woah.org/app/uploads/2026/04/hpai-report-80.pdf.

World Health Organization (WHO). (2025). Antigenic and genetic characteristics of zoonotic influenza viruses and development of candidate vaccine viruses for pandemic preparedness. *Weekly Epidemiological Record*. https://cdn.who.int/media/docs/default-source/influenza/who-influenza-recommendations/vcm-sh-2025/c.-26-september-2025-antigenic-and-genetic-characteristics-of-zoonotic-influenza-a-viruses-and-development-of-candidate-vaccine-viruses-for-pandemic-preparedness.pdf.