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A BRIEF REVIEW OF THE COLUMBID ALPHAHERPESVIRUS-1 INFECTION WORLDWIDE

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ABSTRACT

Pigeon Herpesvirus infection is a widespread disease of pigeons caused by Columbid alphaherpesvirus-1 (CoHV-1), a virus within the Herpesviridae family. This virus is detected worldwide in pigeons and other bird species, especially raptors (owls, falcons, hawks, and eagles). Columbid alphaherpesvirus-1 was first described in the USA, with records dating back to 1943. The main clinical signs of CoHV-1 infection are respiratory and nervous, in addition to nonspecific signs like diarrhoea, vomiting, depression, and anorexia. The virus contributes to young pigeon disease syndrome (YPDS), a condition that affects young pigeons and causes high mortalities. This study aims to provide information about the virus, its transmission, the clinical symptoms, and postmortem lesions in pigeons and other species, the developed diagnostic tools for virus detection, and the disease control methods. As well as this review has also utilized an approach to map CoHV-1-related research papers to understand its research scene and virus world distribution.

Keywords: Pigeon herpesvirus, Raptors, Inclusion body hepatitis, CoHV-1.

INTRODUCTION

Columbid alphaherpesvirus-1 (CoHV-1), also called Columbid herpesvirus-1 and pigeon herpesvirus-1 (Table 1), commonly infects pigeon populations and is classified within the Mardivirus genus (Alphaherpesvirinae, Herpesviridae) (H. Vindevogel and Duchatel, 1991; Swayne et al., 2013; Maclachlan and Dubovi, 2016). The earliest account of CoHV-1 dates back to 1943 in the USA, where it was found in US army pigeons (Smadel *et al.,* 1945). Then it has been isolated from Columbid and non-Columbid birds (Table 2) in many countries all over the world (Figure 1). Because it affects the liver and causes intranuclear inclusions in its tissue, this disease is known as inclusion body herpesvirus hepatitis in Columbid and non-Columbid birds (Bernabé Salazar *et al.,* 1994; Abdul-Aziz and Barnes, 2018; Raj and Jaime, 2019). Nervous and respiratory clinical signs are frequently observed in pigeons affected by Columbid alpha-herpesvirus-1 (CoHV-1). Infection with CoHV-1 in juvenile or immuno-compromised pigeons is associated with high rates of mortality and the occurrence of sudden death (Cornwell and Wright, 1970a; Zhao *et al.,* 2015; Gornatti-Churria *et al.,* 2023). These high mortality rates observed in squabs result in considerable economic losses for the pigeon industry, especially in the case of YPDS (Freick *et al.,* 2008).

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Table 1: Different names of CoHV-1 through the years.

NO.	virus name	references
1	*Intranuclear inclusion (I.N.I.) agent*	(Smadel *et al.,* 1945; Lehner *et al.,* 1967)
2	*Pigeon Herpes Virus*	(Harold J. C. Cornwell, 1968; H. J. C. Cornwell and Wright, 1970 a; Thompson et al., 1977)
3	*Pigeon Herpes Virus 1*	(Henri Vindevogel et al., 1980 a; H. U. Vindevogel et al., 1981)
4	*Pigeon Herpes Encephalomyelitis Virus*	(Tantawi and Hassan, 1982; Shalaby *et al.,* 1985)
5	*Columbid Herpesvirus 1*	(Ehlers *et al.,* 1999; Gailbreath and Oaks, 2008)
6	*Columbid alphaherpesvirus 1*	(Gornatti-Churria *et al.,* 2023; Nath *et al.,* 2023)

Table 2: Different bird species other than pigeons that have been previously documented with CoHV-1 infection.

	species	references
1	Doves	(Kunkle and Duhamel, 1991; David N. Phalen *et al.,* 2017)
2	Falcons	(Aini *et al.,* 1993; Gailbreath and Oaks, 2008; Raj and Jaime, 2019)
3	Owls	(D. N. Phalen et al., 2011; Rose et al., 2012)
4	Cooper's Hawks	(Pinkerton *et al.,* 2008)
5	Australian Hobby	(D. N. Phalen et al., 2011)
6	buzzard	(Grzegorz J. Woźniakowski et al., 2013)
7	goshawk	
8	Kestrel	
9	Herring Gull	
10	Grey Heron	
11	Hooded Crow	
12	Song Thrush	
13	Eagle	(Fischer *et al.,* 2022)

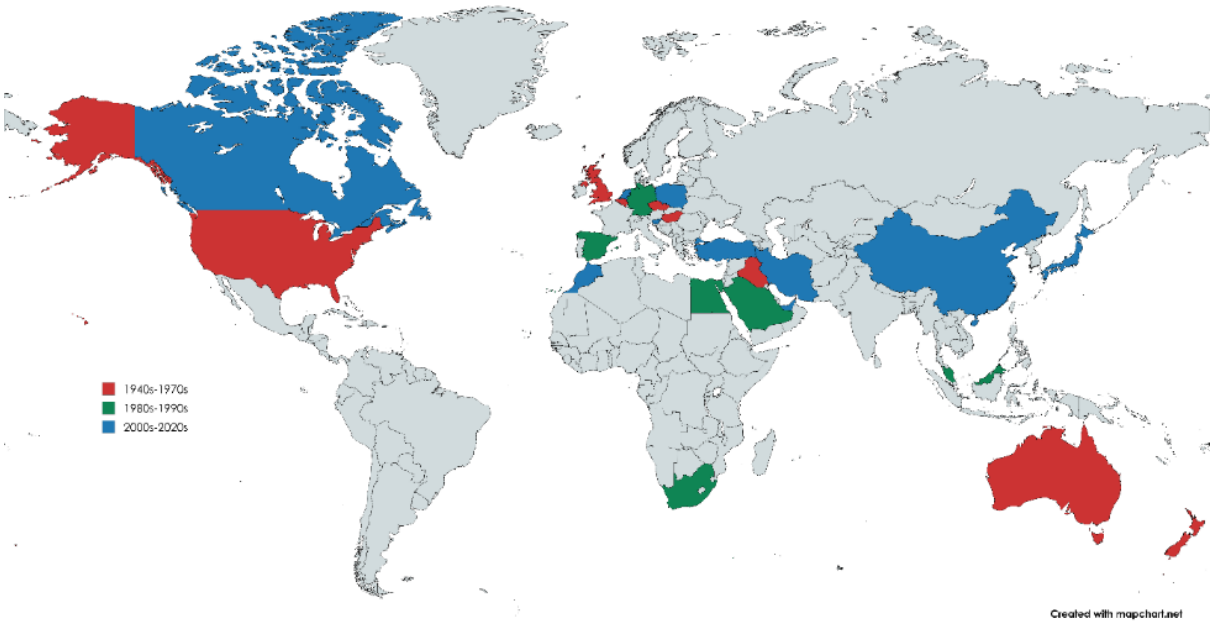

Figure 1: Reported detection of CoHV-1 in different countries across the world, colored according to the earliest retrieved record.

As the pigeon industry gains considerable importance in numerous countries, serious attention must be directed towards the diseases that threaten its productivity and sustainability, and among these diseases is CoHV-1 infection. This review aims to

provide a recent review about the Columbid alphaherpesvirus-1, presenting a comprehensive overview covering its etiology, epidemiology, transmission, clinical manifestation, diagnosis, vaccines, and prevention/control measures.

Causative agent

The *Herpesviridae family* is organized into the *Gammaherpesvirinae* subfamily, the *Alphaherpesvirinae* subfamily, and the *Betaherpesvirinae* subfamily. The Alphaherpesvirinae subfamily comprises five genera, including *Scutavirus, Varicellovirus, Simplexvirus, Iltovirus,* and *Mardivirus.* The genus Mardivirus itself is composed of six species: *Mardivirus Columbid alphaherpesvirus 1, Mardivirus Anatid alphaherpesvirus 1, Mardivirus Gallid alphaherpesvirus 2, Mardivirus Spheniscid alphaherpesvirus* 1, *Mardivirus Gallid alphaherpesvirus 3,* and *Mardivirus Meleagrid alphaherpesvirus 1* (Maclachlan and Dubovi, 2016; Simmonds *et al.,* 2024).

Columbid alphaherpesvirus-1 is characterized as an enveloped virus containing a single, linear molecule of double-stranded DNA within its core, which is further surrounded by a capsid and tegument. The protein capsid of CoHV-1 has a diameter of approximately 180 nm and is structurally composed of 162 elongated capsomers, specifically 150 hexons and 12 pentons (McMullin, 2020). The alpha-herpesvirus genome is characterized by two key segments: the unique-long (UL) and unique-short (US) regions. Herpesvirus genes are commonly classified into three functional groups: immediate early and early genes, which encode regulatory and replication proteins; late genes, responsible for structural proteins; and nonessential genes. The genes produced by these viral genes are probably crucial in the pathogenesis of herpesvirus infections. (Maclachlan and Dubovi, 2016). The replication cycle of CoHV-1, like all herpesviruses, involves a series of distinct steps. When the virus infects a cell, its DNA is liberated and subsequently migrates to the host cell's nucleus. This initiates the transcription of immediate-early genes, which code for regulatory proteins. Subsequently, the expression of early and then late genes occurs, leading to the production of various viral proteins (Whitley, 1996). The genome of CoHV-1 consists of 204,237 base pairs and has a guanine-cytosine content of approximately 61.5%. This genome encodes an estimated 130 genes (Guo *et al.,* 2017). The Columbid herpesvirus-1 (CoHV-1) genome shares class E structural features with the genomes of Falconid herpesvirus 1, Meleagrid herpesvirus 1, Gallid herpesvirus 2, and Gallid herpesvirus 3 (Guo *et al.,* 2017; McMullin, 2020). Sequencing and comparing a part of the polymerase gene from falcon, owl, and pigeon herpesviruses showed that their sequences were closely related(Gailbreath and Oaks, 2008). Based on the shared features, some researchers proposed that Strigid herpesvirus-1, Columbid herpesvirus-1, and Falconid herpesvirus-1 were, in fact, the same virus. They proposed using the name CoHV-1, suggesting that pigeons might be responsible for transmitting the virus to various birds of prey (Gailbreath and Oaks, 2008; Raj and Jaime, 2019). However, (Spatz *et al.,* 2014) showed that the UL43 gene exhibits clear differences between FaHV-1 and Columbid herpesvirus 1, even though the viruses share 99.4% identity in multiple sequence alignments. Conseq-uently, CoHV-1 and FaHV-1 belong to the same monophyletic group (Guo *et al.,* 2017). Also, some recent research indicates the presence of a herpesvirus (Strigid herpesvirus-1) distinct from CoHV-1 in owl species (Gleeson *et al.,* 2019; Žlabravec *et al.,* 2024).

Transmission and infection

Horizontal transmission is the main transmission route for CoHV-1, but vertical transmission hasn't been detected (Vindevogel and Pastoret, 1980b). Direct contact between infected and susceptible pigeons is the main route of infection, especially in young pigeons. To a lesser

extent, the virus is also excreted with secretions of the respiratory tract, the conjunctiva, and the faeces (Kaleta and Docherty, 2007). It should also be noted that the trichomonas (Trichomonas gallinae), which frequently occurs in pigeons, can harbour the virus in the cell body and thus enable transmission via contaminated drinking water (Freick, 2005; McMullin, 2020). Adult pigeons survive as carriers after infection and may shed the virus intermittently (Vindevogel *et al.,* 1980a). Squabs can get infections from their parents through crop milk feeding (Vindevogel *et al.,* 1985). Maternal antibodies are the main defence mechanism of squabs, by which they can protect themselves from severe disease symptoms or death. Therefore, many squabs become asymptomatic carriers after CoHV-1 infection (Vindevogel and Pastoret, 1980b). This virus affects a wide variety of tissues, including the liver, gastrointestinal tract, spleen, pancreas, eye, conjunctiva, respiratory tract, air sacs, bone marrow, ear, kidneys, gonads, thymus, cloaca, bursa of Fabricius, brain, skin, heart, and thyroid gland (Gornatti-Churria *et al.,* 2023). The primary route of Columbid alphaherpesvirus-1 (CoHV-1) transmission to falcons, hawks, and owls is through the ingestion of infected pigeons as prey (Pinkerton *et al.,* 2008; Žlabravec *et al.,* 2022). Furthermore, these birds of prey are considered an important route in facilitating the spread of the virus between pigeon populations worldwide (Figure 2) (Woźniakowski *et al.,* 2013).

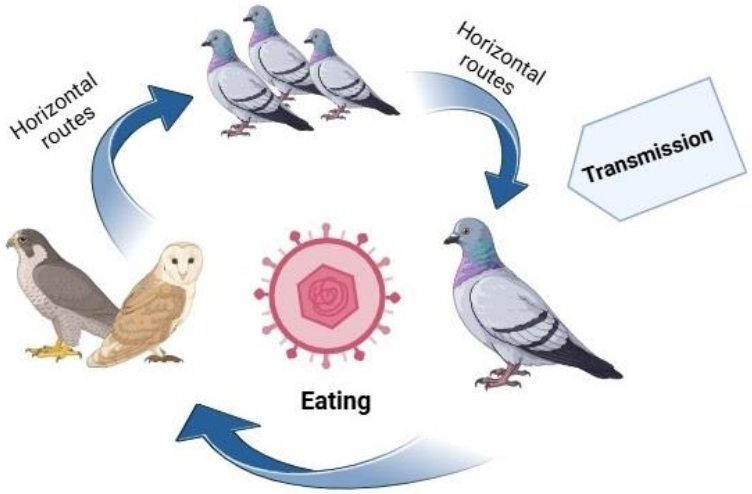

Figure 2: Diagram of the transmission of CoHV-1 between pigeons and raptors.

Epidemiology

First identified in the USA in 1943 (Smadel *et al.,* 1945), Columbid alphaherpesvirus-1 (CoHV-1) has since been identified in numerous countries across the globe: the United Kingdom (Cornwell, 1968), Czechia (Krupieka *et al.,* 1970), Australia (Boyle and Binnington, 1973; Nath *et al.,* 2023), Belgium (Vindevogel *et al.,* 1975; Vindevogel and Pastoret, 1981), Hungary (Vetesi and Tanyi, 1975), New Zealand (Thompson *et al.,* 1977), Iraq (Tantawi *et al.,* 1979), Egypt (Tantawi and Hassan, 1982; Abdellatif *et al.,* 2025), South Africa (Pollard and Marais, 1983), Saudi Arabia (Shalaby *et al.,* 1985), Spain (Carranza *et al.,* 1986), Germany (Schraishuhn, 1989; Freick *et al.,* 2008), Malaysia (Aini *et al.,* 1993), Japan (Yui *et al.,* 2008), Poland (Stenzel *et al.,* 2012; Woźniakowski *et al.,* 2014), Canada (Rose *et al.,* 2012), Netherlands (Hellebuyck *et al.,* 2017), Morocco (Lorenté, 2017), United Arab Emirates (Raj and Jaime, 2019), Italy (Giglia *et al.,* 2022), Slovenia (Žlabravec *et al.,* 2022), Turkey (Sahindokuyucu *et al.,* 2022), USA (Gornatti-Churria *et al.,* 2023), Iran (BehrouziNasab et al., 2024). Despite the previous studies mentioning (CoHV-1), its epidemiology is still poorly studied. This is because the research papers about this virus are little in describing the actual

epidemiology of the virus, especially with unrecorded field outbreaks of the virus.

Clinical and pathological manifestations

a) In the pigeons:-

Clinical manifestation of CoHV-1 infection in pigeons is variable and not very specific (Marlier and Vindevogel, 2006). The first proof of the virus came from birds presenting a clinical picture similar to that of psittacosis (Cornwell *et al.,* 1970 b). The highest susceptibility to CoHV-1 infection is observed in young pigeons and immunocompromised adult pigeons (Vindevogel and Pastoret, 1980b). Cases of sudden death without prior clinical signs are also reported in pigeons, especially after weaning at 4-5 weeks of age (Bernabé Salazar *et al.,* 1994). The clinical signs are respiratory symptoms (Figure 3) such as rhinitis, Epiphora, conjunctivitis, and sneezing, up to severe dyspnea, which is associated with damage to the upper respiratory system (Cornwell *et al.,* 1970b; Ritchie *et al.,* 1994; Freick *et al.,* 2008). In chronicity, complications with other infections, such as Trichomonas gallinae or bacterial infections, may occur, resulting in sinusitis and severe dyspnea with diphtheroid lesions in the pharynx (McMullin, 2020; Gornatti-Churria *et al.,* 2023). Other clinical signs are reported, such as hypo-vigilance, depression, anorexia, and digestive signs with vomiting/regurgitation, and diarrhea (Figures 3 and 4) (Marlier and Vindevogel, 2006; Zhang *et al.,* 2015). Neurological deficits are also detected (Figure 3) (Tantawi *et al.,* 1979; Tantawi, 1981; Vindevogel *et al.,* 1985).

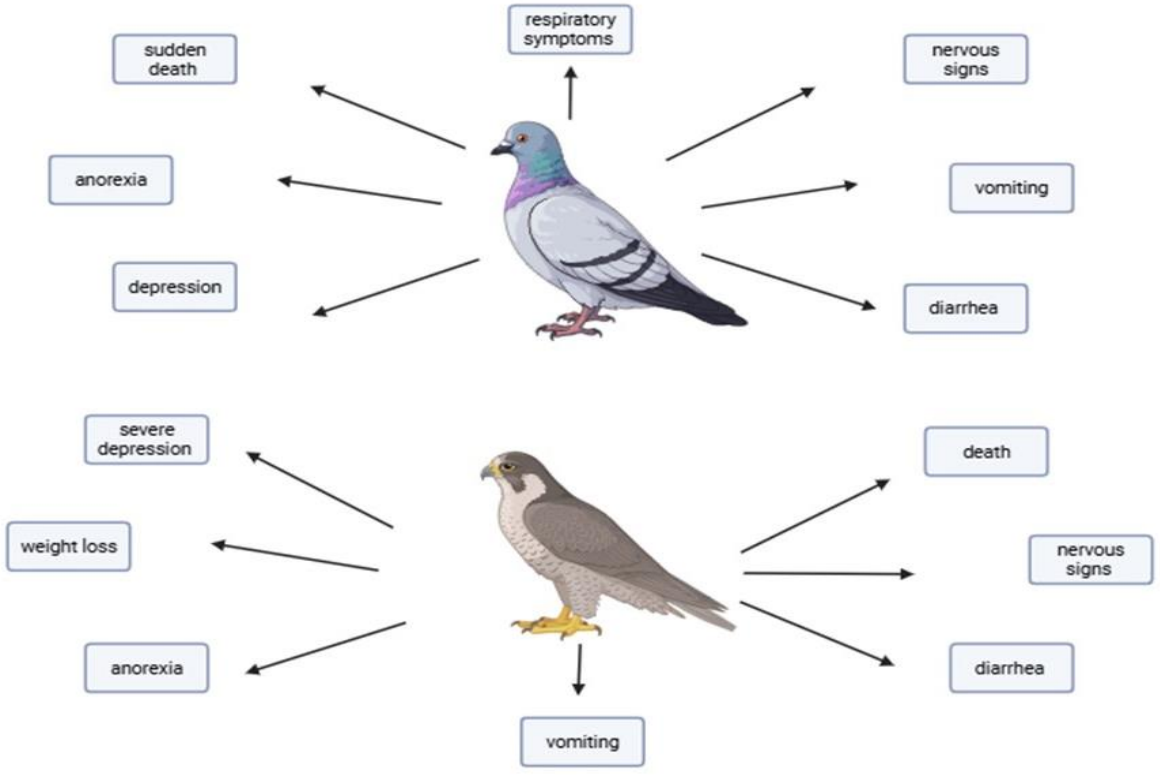

Figure 3: Diagram demonstrating the clinical signs in pigeons and falcons.

b. In falcons and other birds:

The clinical signs presented in the genus Falco are also not very specific and of variable intensity. The bird of prey generally presents a period of severe depression and weight loss, anorexia, associated with non-specific digestive signs (diarrhoea, vomiting, regurgitation), and nervous signs are also detected(Raj and Jaime, 2019). Respiratory system involv-

ement directly linked to CoHV-1 has not been demonstrated in falcons. This is often the result of secondary bacterial or fungal infections (Tarello, 2011). At the end of this clinical phase, the falcons die in almost 100% of cases, 4 to 6 days post-infection (Figure 3) (Raj and Jaime, 2019)

CoHV-1 infection has been detected with nonspecific symptoms in bird species like hawks, owls, buzzards, eagles, and sparrows. Sometimes, sudden death may occur (Tantawi *et al.,* 1979; Pinkerton *et al.,* 2008; Woźniakowski *et al.,* 2013; Žlabravec *et al.,* 2021a; Žlabravec *et al.,* 2021b; Fischer *et al.,* 2022)

Gross Lesions

a. In pigeons:

In the acute stage of infection, the mucous membranes of the proximal part of the gastrointestinal tract and the proximal part of the respiratory tract become congested and inflamed. Subsequently, small ulcerations and necrotic foci may evolve in these mucous membranes (Figure 4). In critical cases, diphtheritic membranes may develop (Figure 4), especially with mixed infections like with Trichomonas gallinae (Callinan *et al.,* 1979). In many cases, small ulcerated areas in the glandular stomach and around the beak angle may be found (Callinan *et al.,* 1979). After viremia, necrosis may be seen in the hepatic tissue (Figure 4). Splenomegaly and hepatomegaly, as well as ulcers in the intestine covered by diphtheroid membranes, may be observed in some cases (Lorenté, 2017). Histopathologically, necrosis was detected in the splenic tissues, renal tissue, pancreas, lungs, nasal mucosa, and the proventriculus (Abdul-Aziz and Barnes, 2018; Gornatti-Churria *et al.,* 2023). If the infection is complicated by secondary infections, the whole respiratory tract may be implicated, leading to obstruction of the trachea by caseous material, and some pigeons may show airsacculitis, sinusitis, and pericarditis (Vindevogel *et al.,* 1980a; Vindevogel, 1981; Lorenté, 2017; McMullin, 2020).

b. In falcons and other birds:

The lesion picture described in falcons and other raptors is similar, including hepatomegaly, splenomegaly, a multifocal necrotizing area in the liver and spleen, white nodules in the gonads, congestion with white caseous necrotic lesions in the small intestine, cloacal bursa, thyroid gland, thymus, brain, renal tissue, and pancreatic tissue (Pinkerton *et al.,* 2008; Rose *et al.,* 2012; Phalen *et al.,* 2017; Raj and Jaime, 2019).

Microscopic lesions

a. In pigeons:

In CoHV-1 infection, the epithelial lining of the mouth, pharynx, larynx, trachea, and salivary glands undergo necrosis. Subsequently, ulcers may form at sites of extensive necrosis (Vindevogel and Pastoret, 1981). Examination reveals necrosis, degeneration, and intranuclear inclusions within neighbouring epithelial cells, accompanied by mononuclear cell infiltration in the subjacent connective tissue (Vindevogel and Pastoret, 1981). The trachea exhibits significant damage, characterized by the loss of cilia on its cells alongside an overgrowth of the epithelial lining (hyperplasia). A mild inflammatory response is also evident, with heterophils present within the epithelium and the tracheal passage. Notably, the mucus-producing glands are largely destroyed (Cornwell *et al.,* 1970b; Marlier and Vindevogel, 2006; Zhao *et al.,* 2015). As the condition progresses, a consistent finding is hepatitis with localized areas of cell death (necrotic foci), and the presence of intranuclear inclusion bodies within numerous liver cells (Vindevogel and Pastoret, 1981; Lorenté, 2017; Gornatti-Churria *et al.,* 2023). These inclusions are mainly eosinophilic Cowdry type inclusions (Figure 4) (Vindevogel *et al.,* 1975). Fibrin-rich heterophils and some macrophages typically cover the liver capsule. Kidney lesions show tubular necrosis, hyperemia, mild lymphocyte and heterophil infiltration. The spleen exhibits small necrotic foci and numerous macrophages in the medulla (

Vindevogel and Pastoret, 1981; Zhang *et al.,* 2015). Some of the necrotic foci are formed of degenerated renal parenchyma, lymphocytes, and macro-phages. Intranuclear inclusions are also found at the margins of the necrotic foci in the renal tissue (Cornwell and Wright, 1970a; Vindevogel *et al.,* 1975; Zhao *et al.,* 2015; Lorenté, 2017). Occasionally, herpesvirus lesions are found in the brain (Cornwell *et al.,* 1970b). Lesions have also been illustrated in the pancreas by the detection of diffuse interstitial infiltration of mononuclear cells and Eosinophilic inclusions (Cornwell *et al.,* 1970b; Callinan *et al.,* 1979)

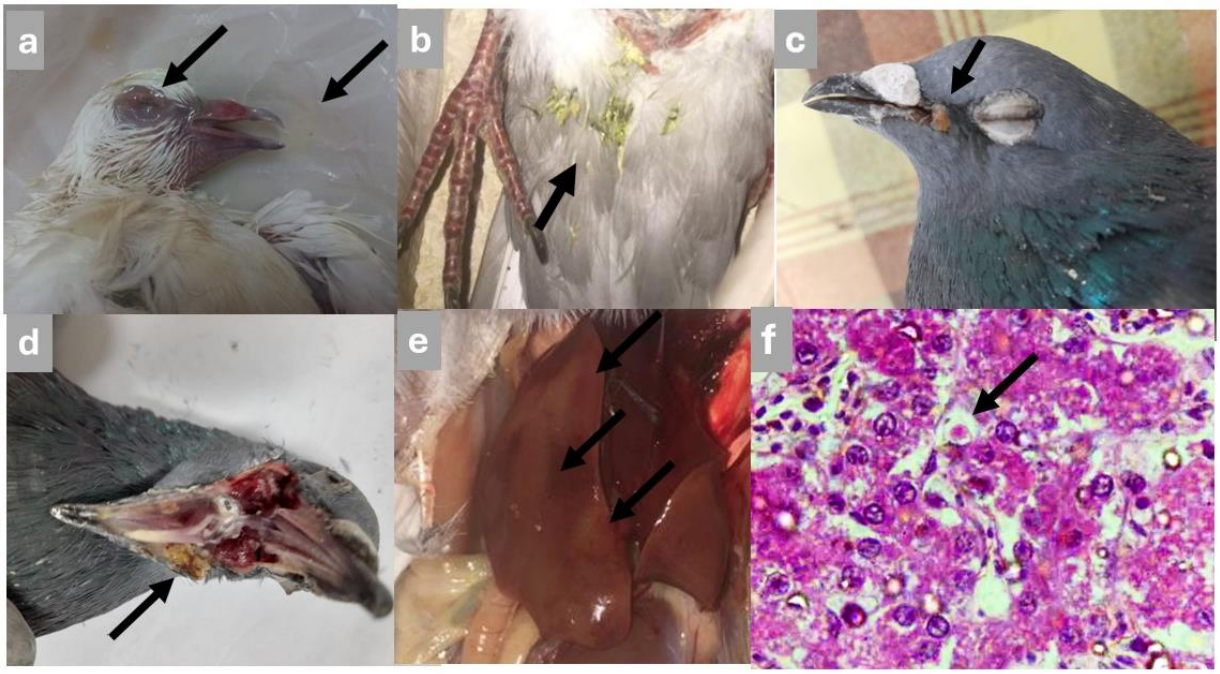

Figure 4: Some clinical manifestations of CoHV-1 infection in pigeons: a) Pigeon with vomiting and eye issues, b) Pigeon with diarrhea, c) Pigeon shows an ulceration in the mouth commissure, d) Pigeon shows a diphtheritic membrane in the oropharynx, e) Necrosis in the liver of a pigeon, f) Eosinophilic intranuclear inclusion in hepatocytes (x 100).

b. In Falcons and other raptors:
Microscopically, the liver displays multiple areas of necrosis with karyolysis and fragmentation. Within hepatocytes, the intranuclear inclusions are eosinophilic and display a distinct central inclusion surrounded by a clear, pale (hypochromatic) halo, pushing the chromatin to the periphery (Ward *et al.,* 1971; Mozos *et al.,* 1994; Gailbreath and Oaks, 2008; Rose *et al.,* 2012). These necrotic areas and eosinophilic inclusions can also be found in the splenic tissue, intestines, bone marrow, renal tissue, and gonads (Mozos *et al.,* 1994; Pinkerton *et al.,* 2008; Raj and Jaime, 2019). Necrotic foci surrounded by cells with inclusions were sometimes reported in other organs not showing macroscopic lesions: heart, thymus, bursa Fabricius, parathyroids, thyroid, pancreas, cecal lymphoid tissue and intestinal lymphoid tissue (Gailbreath and Oaks, 2008; Pinkerton *et al.,* 2008; Phalen *et al.,* 2011; Lorenté, 2017).

Diagnosis

Before molecular biology techniques development, CoHV-1 infections were primarily identified and diagnosed through histological analysis and electron microscopy, which revealed intranuclear inclusions in infected tissues (Cornwell and Wright, 1970a; Tantawi *et al.,* 1979; Abd-El-Motelib *et al.,* 1994; Bernabé Salazar *et al.,* 1994). To date, histopathological examination remains the confirmatory diagnostic modality, predicated on the visual identification of eosinophilic

intranuclear inclusions within the tissue microenvironment of necrotic lesions (Lorenté, 2017; Gornatti-Churria *et al.,* 2023; Phalen, 2024).

CoHV-1 can be isolated from a range of sample types, including throat swabs, organs, and faeces (Raue *et al.,* 2005). Viral isolation can be performed using various methods, including tissue culture. Columbid alphaherpesvirus-1 replicates efficiently in chicken embryo fibroblasts (CEF), where cytopathic effects (rounding, multinucleated syncytia with intranuclear inclusions, and eventual lysis) are noticeable within 12 hours and pronounced by 24 hours post-inoculation. Although other cell types, such as chicken embryonic kidney, duck embryo fibroblasts, pigeon embryo fibroblasts, and chicken embryo hepatic cells, can also support viral growth, the resulting cytopathic effects vary from each other (Cornwell and Wright, 1970a; Vindevogel and Pastoret, 1981; Woźniakowski *et al.,* 2013; Zhang *et al.,* 2015; McMullin, 2020).

Isolation and propagation of CoHV-1 are feasible in embryonating chicken eggs through inoculation of the chorioallantoic membrane, with the subsequent production of characteristic pocks (Cornwell and Wright, 1970a; Tantawi, 1981; Lorenté, 2017; Sahindokuyucu *et al.,* 2022).

Techniques like uniplex PCR (Raue *et al.,* 2005), multiplex PCR (Freick *et al.,* 2008), and loop-mediated isothermal amplification (LAMP)(Woźniakowski *et al.,* 2014) were developed for virus diagnosis. The common primer pair used for PCR detection of CoHV-1 is PiHV-s (GGGACGCTCT-GATTAAGGAAT), PiHV-as (CTTG-GTGATCAGCAGCAGCTTG), which is used for amplification of a part of the Polymerase gene (242 base pairs) (Raue *et al.,* 2005). Serological techniques, such as virus-neutralization or indirect immuno-fluorescence methods, were also used for the diagnosis of the virus (Vindevogel, 1981; Swayne *et al.,* 2013).

The genome's first complete sequencing was published in China by (Guo et al., 2017), Who gave an exact infection detection method. Although the sequencing of CoHV-1 is the most exact identification method ever used, it is inappropriate for diagnostics due to its complexity, expense, and its huge genome length. Genomic analysis revealed that CoHV-1 exhibits a larger genome size relative to other avian alphaherpesviruses and is positioned within a monophyletic clade alongside FaHV-1 (Guo *et al.,* 2017).

Differential Diagnosis

The differential diagnosis of CoHV-1 with viral infections includes avian paramyxo-virus type 1 (APMV1) and pox virus infections. The diphtheritic membrane of CoHV-1 is loosely adhered and doesn't cause ulceration when removed, but in pox infection, the diphtheritic membrane is intensely adhered and causes bleeding and ulceration when removed (Marlier and Vindevogel, 2006). A hemagglutination inhibition test can be used to differentiate between CoHV-1 and (APMV1) (McMullin, 2020). Infections with *Chlamydia*, *Salmonella*, or *Trichomonas gallinae* can cause a clinical picture similar to that induced by CoHV-1 and may be mixed with it as a concurrent infection (Table 3) (Freick, 2005; Gornatti-Churria *et al.,* 2023).

Treatment, Prevention, and Control

Environmental management and hygiene rules must be implemented and improved. The treatment of mixed infections must be applied to achieve successful support treatment (Marlier and Vindevogel, 2006). Attempts to prevent CoHV-1 infection using trisodium phosphonoformate and acycloguanosine were unsuccessful (Schwers et al., 1981; Henri Vindevogel; Pastoret;Aguilar-Setien, 1982; Thiry et al., 1983). Vaccinating against CoHV-1 can reduce both the spontaneous re-excretion of the virus and its transmission. However, the vaccines couldn't stop pigeons' latent infection and virus shedding from

immunosuppressed birds (Henri Vindevogel; Pastoret;Leroy, 1982). Noteworthy, there is a commercial herpesvirus inactivated vaccine for pigeons in European countries (McMullin, 2020).

Table 3: Differential diagnosis of CoHV-1.

No.	Disease/ Condition	Key Clinical Signs	Key Lesions /Findings	Diagnostic Tools	references
1	*Columbid Herpesvirus-1 (CoHV-1)*	Young pigeons: Depression, anorexia, conjunctivitis, nasal discharge, dyspnea, oral ulcers, vomiting, diarrhoea, and sudden death. Adults: Often subclinical.	The liver and spleen display necrotic foci, while diphtheritic membranes are noted in the oropharynx and nasopharynx. Intranuclear inclusions are evident in liver cells and epithelial cells	PCR for viral DNA, virus isolation, and histopathology (identification of inclusion bodies).	(Lorenté, 2017)
2	*Paramyxovirus (PMV-1)*	Neurological signs (torticollis, tremors, paralysis), diarrhoea, vomiting, increased thirst, and sometimes respiratory signs.	Non-specific lesions may see visceral haemorrhages. Histopathology of the brain and other tissues may show non-suppurative encephalitis	Virus isolation, PCR for viral RNA, serology (HI test).	(Tong *et al.,* 2024)
3	*Chlamydia psittaci* (Ornithosis/Psittacosis)	Respiratory signs (sneezing, nasal discharge, conjunctivitis), lethargy, anorexia, diarrhea, and sometimes neurological signs.	Air sacculitis, hepatomegaly, splenomegaly, and fibrinous exudate on serous membranes. The presence of elementary bodies is observed in impression smears or tissue sections.	PCR for bacterial DNA, culture, serology (ELISA, CF test), cytology (impression smears).	(Tomasz Stenzel *et al.,* 2014)
4	*Trichomoniasis* ("Canker")	Yellowish-white cheesy plaques in the mouth and pharynx, difficulty swallowing, and weight loss.	Caseous lesions in the oral cavity, pharynx, esophagus, and sometimes internal organs.	Direct microscopic examination of oral swabs or lesions (motile trichomonads), and culture.	(Gornatti-Churria *et al.,* 2023; Jaafar, 2023)
5	*Salmonellosis (Salmonella Paratyphoid)*	Clinical signs can include depression, anorexia, diarrhoea (frequently greenish), vomiting, and weakness. Neurological involvement, manifesting as tremors or paralysis, may also be observed, and sudden death is a possibility.	Affected birds can show an enlarged, congested liver and spleen, enteritis, and sometimes caseous plugs in the intestines, along with swollen joints.	Key diagnostic methods include culture-based identification of Salmonella bacteria and PCR to identify specific Salmonella genes.	(Georgiades and Iordanidis, 2002)
6	*Pox* (Pigeon Pox)	Pigeon Pox manifests in two main forms. The cutaneous form is identified by wart-like growths on the exposed skin of the head, legs, and vent. The diphtheritic, or wet form, is characterized by white plaques in the oral cavity, pharynx, and trachea, frequently associated with conjunctivitis and breathing difficulties.	Cutaneous: Raised, scabby lesions on the skin. Diphtheritic: Yellowish white, raised plaques on mucous membranes.	Visual examination of lesions, histopathology (intracytoplasmic inclusions; Bollinger bodies), PCR	(Mohamed *et al.,* 2024)

Economic importance

The economic importance of CoHV-1 is mainly related to its impact on pigeon breeding and racing industries, as well as the health of wild bird populations:

- The commercial rearing of pigeons for meat production represents a recognized industry in many nations across the globe (Mia *et al.,* 2022; Adawy and Abdel-Wareth, 2023). Pigeon racing is a popular competitive sport with a global following (Deckers and Pezzetta, 2023; Mostert and Donaldson, 2024). CoHV-1 can cause significant morbidity and mortality, especially in young pigeons, leading to considerable financial losses for breeders and racing enthusiasts. Outbreaks can result in decreased flock sizes, reduced race performance, and the costs of veterinary care (Gornatti-Churria *et al.,* 2023).

- Infection in wild raptors, such as falcons and owls, often results in high mortality rates. This can have ecological consequences and potentially impact ecotourism related to these species (Raj and Jaime, 2019; Žlabravec *et al.,* 2022).

It's important to note that the economic importance of CoHV-1 is not as broadly significant as diseases affecting major livestock or poultry species. However, it can have considerable financial consequences for individuals and industries directly involved with pigeons and can impact the health and conservation of wild bird populations (Patrick *et al.,* 2022).

CONCLUSION

This review provides information about CoHV-1 virus transmission, diagnosis, and control, as well as the clinical manifestation of the disease. In addition to providing data on the virus distribution in the world. However, the epidemiology of CoHV-1, as well as its actual economic impact, remains poorly studied. The research papers about this virus are limited. Further studies should be conducted to determine the CoHV-1 in all countries to assess its economic impact, its epidemiology, identify the traditional control measures, and for effective vaccination development.

CONFLICT OF INTEREST

There is no conflict of interest regarding the authorship or publication of this article.

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مراجعة موجزة لعدوى فيروس هربس الحمام ألفا – ١حول العالم

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تعتبر عدوى فيروس الهربس في الحمام مرضًا واسع الانتشار يصيب الحمام ويُسببه فيروس هربس الحمام ألفا – ١ (CoHV-1)، وهو فيروس ينتمي إلى عائلة الفيروسات الهربسية .(Herpesviridae) تم اكتشاف هذا الفيروس في جميع أنحاء العالم في الحمام وأنواع أخرى من الطيور، وخاصة الطيور الجارحة (البوم والصقور والنسور). تم وصف فيروس هربس الحمام ألفا -١لأول مرة في الولايات المتحدة الأمريكية، بسجلات تعود إلى عام ١٩٤٣. تتمثل العلامات السريرية الرئيسية لعدوى CoHV-1 بأعراض تنفسية وعصبية، بالإضافة إلى علامات غير محددة مثل الإسهال والقيء والاكتئاب وفقدان الشهية. يُساهم الفيروس في الإصابة بمتلازمة مرض صغار الحمام (YPDS) ، وهي حالة تصيب صغار الحمام وتسبب معدلات وفيات عالية. تهدف هذه الدراسة إلى تقديم معلومات حول الفيروس وانتقاله والأعراض والصفه التشريحية التي تُصيب الحمام والطيور الاخرى بعد النفوق، والأدوات التشخيصية المطورة للكشف عن الفيروس، وطرق مكافحة المرض. كما اعتمدت هذه المراجعة أيضًا على نهج لرسم خريطة للأبحاث المتعلقة بفيروس CoHV-1 لفهم طبيعة الابحاث المتعلقة به والتوزيع العالمي للفيروس.

الكلمات المفتاحية: فيروس الهربس في الحمام، الطيور الجارحة، التهاب الكبد ذو الأجسام الشاملة،CoHV-1 .