Common viral and bacterial avian respiratory infections: an updated review

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ABSTRACT Many pathogens that cause chronic diseases in birds use the respiratory tract as a primary route of infection, and respiratory disorders are the main leading source of financial losses in the poultry business. Respiratory infections are a serious problem facing the poultry sector, causing severe economic losses. Avian influenza virus, Newcastle disease virus, infectious bronchitis virus, and avian pneumovirus are particularly serious viral respiratory pathogens. *Mycoplasma gallisepticum*, *Staphylococcus, Bordetella avium, Pasteurella multocida*, *Riemerella anatipestifer*, *Chlamydophila psittaci*, and *Escherichia coli* have been identified as the most serious bacterial respiratory pathogens in poultry. This review gives an updated summary, incorporating the latest data, about the evidence for the circulation of widespread, economically important poultry respiratory pathogens, with special reference to possible methods for the control and prevention of these pathogens.

Key words: avian respiratory pathogen, clinicopathological characteristic, disease control, poultry, vaccine

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INTRODUCTION

The poultry industry offers a solution for bridging the nutritional gap in many people's diets and providing a rich and economical source of animal protein (Salem and Attia, 2021; Abd El-Hack et al., 2022a; Abd El-Fatah et al., 2023). Nevertheless, respiratory diseases pose a significant threat to global health because of their high prevalence, a wide range of mortality and morbidities, the need for expensive treatments, and the impact on bird performance. Additionally, several of these infections have zoonotic significance, pose a risk to the public's health, and inflict significant economic harm (Jordan et al., 2018). These respiratory difficulties can lead to various infectious and management-related issues, including viral, bacterial, fungal, and parasitic infections. This review focuses on the most prevalent and dangerous avian respiratory tract infections caused by viruses and bacteria (Figure 1).

MOST COMMON AVIAN RESPIRATORY INFECTIONS CAUSED BY VIRUSES

Avian Influenza

Avian influenza (**AI**) viruses belong to the family orthomyxoviridae, which is a single-stranded, eight-segmented, negative sensed riboxynucleic acid (**−ssRNA**) viruses that encodes at least 10 viral proteins (Capua and Alexander, 2004). The family is categorized

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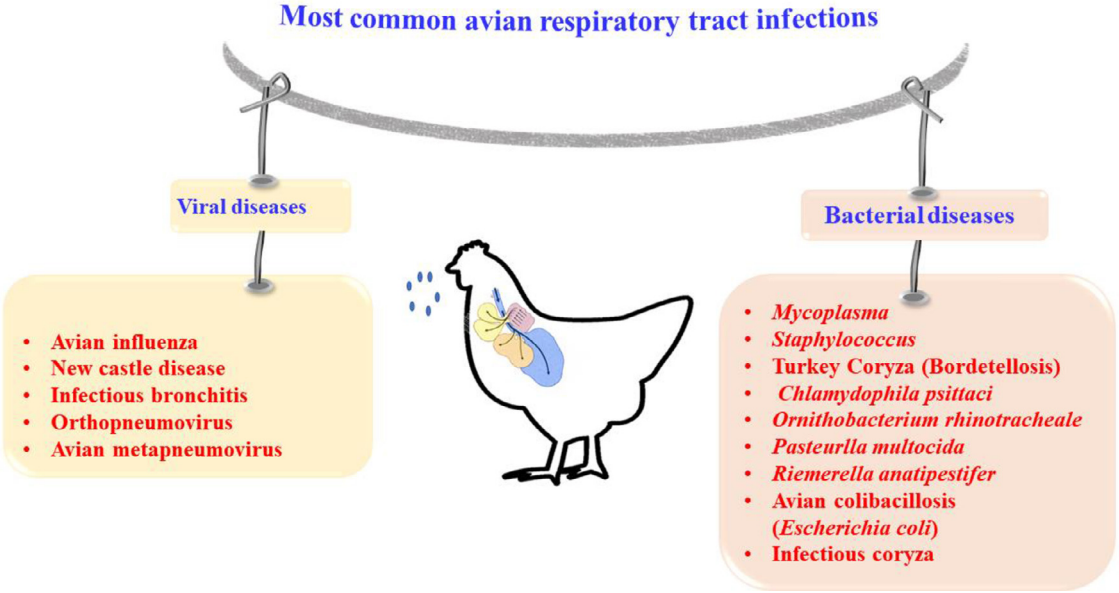


Figure 1. Most common avian respiratory tract infections caused by viruses and bacteria.

into 3 main genera of influenza (types A, B, and C) and 2 unknown genera: isavirus and thogotovirus (Spackman and Suarez, 2008). AI viruses follow the genus influenza A—viral species that are abundant and can infect avian and other mammalian species (Capua and Alexander, 2004).

Influenza A viruses are subtyped based on the envelope proteins neuraminidase (**NA**) and hemagglutinin (**HA**). There are 9 subtypes (N1−N9) of NA and 16 subtypes (H1−H16) of HA (Capua and Alexander, 2004). AI A viruses tend to show antigenic changes in their surface glycoproteins (HA and NA) due to antigenic shifts and antigenic drifts (Wright and Webster, 2001). According to pathogenicity, the infection may be highly lethal "high pathogenic AI" (**HPAI**) and is thus a public health hazard with zoonotic importance. At the same time, less severe forms of AI have lower pathogenicity AI and are nonhighly pathogenic with lower impacts on poultry production, and these are called low pathogenic AI (**LPAI**) (Swayne, 2006).

AI viruses could be identified based on the intravenous pathogenicity index (**IVPI**) as highly pathogenic notifiable AI (**HPNAI**) in the European Union legislation, which defines HPNAI as "an infection of poultry caused by influenza A virus whose IVPI in 6-wk-old hens is greater than 1.2, or any infection with influenza A viruses of the H5 or H7 subtype for which nucleotide sequencing demonstrates the presence of numerous basic amino acids at the cleavage site of the HA" (Swayne, 2006).

HPAI Viruses AV H5N1 (fowl plague) is a highly pathogenic virus that causes severe economic losses and has been associated with an up to 100% mortality rate (Swayne, 2006). AV H5N1 is characterized clinically by septicemia with a variable degree of edema, hemorrhages, and necrosis in the skin and visceral organs. It may produce variable lesions in the visceral organs, hemorrhages on serosal and mucosal surfaces, necrotic foci within the parenchyma of visceral organs, circumscribed bluish ulcers in Peyer's patches, and pneumonia in the lungs (Swayne and Halvorson, 2008; Oliver et al., 2022).

Usually, HPAI viruses result in necrosis in the pancreas, spleen, heart, liver, and kidney, with the lungs usually exhibiting pneumonia and edema, and in some cases, the lungs are congested with hemorrhagic patches. Moreover, atrophy in the thymus and bursa, and spleen enlargement, may be observed (Swayne and Halvorson, 2008).

HPAI (H5N1) is enzootic and threatens the poultry sector in Egypt, causing severe monetary losses (Kandeil et al., 2016). H5N1 was reported for the first time in 2006 and continued at somewhat stable levels without new pandemics until 2008. During 2008, subclade 2.2.1.1 emerged and spread until early 2011 (Shehata et al., 2019). At the same time, another clade 2.2.1 viruses were changing. In 2013 and 2014, viruses merged within a new cluster, 2.2.1a (Shehata et al., 2019). Then, in 2014, HPAI H5N1 mutated at R140K in antigenic site A, and A86V in antigenic site E of the HA gene. Like other viruses in the new cluster, it was categorized as being within clade 2.2.1.2 (Arafa et al., 2016).

HPAI H5N1 was highly zoonotically important in Egypt, with 292 human cases reported during 2006−2015 and a 34% case-fatality rate (Kandeil et al., 2016). Egypt recorded the largest number of human cases of the HPAI H5N1 virus in the world and was 1 of only 3 countries (Egypt, China, and Bangladesh) that recorded the LPAI H9N2 in humans (Naguib et al., 2019). In 2016, HPAI viruses of the H5N8 subtype originally from China were identified in birds in Egypt (Selim et al., 2013).

During 2016−2017, commercial broiler chickens with the H5N8 viruses suffered from respiratory problems and mortality. Sequence analysis of these H5N8 viruses indicated that they had clustered within the 2.3.4.4b clade (Shehata et al., 2019; Yehia et al., 2020). Multiple

introductions of different reassorted viruses came from the incursion of AI H5N8 in Egypt. In 2017, 3 genotypes were found in Egypt. The first genotype was a reassortment of PB2 and NP from H5N6 and H6N2 that had recently been detected in Italy. The second was a new reassortment of PB1 and NP genes from H6N8 or H1N1 and H6N2. Last, a reassortment occurred in the PA and the NP genes that had previously occurred in Germany and originated from H6N2 viruses (Yehia et al., 2018) and 3 other introductions of the H5N8 virus into fowl in Egypt (Salah Eldin et al., 2018).

Kandeil et al. (2017) recorded other reassortments in PB2, PB1, PA, and NP. M was from different Eurasian countries from other AI subtypes such as H3N8, H4N6, H2N3, H9N2, and H10N7. During 2017−2018, H5N8 viruses were detected in poultry farms where the birds exhibited respiratory problems. The mortality was high and was co-occurring with H9N2 viruses in 22 out of 39 poultry flocks in Egypt. The highest incidence of H5N8 viruses was reported in 2018. An investigation of 66.6% of poultry flocks in Egypt, and sequence analysis of H5N8 viruses consequently showed that the viruses had clustered within the 2.3.4.4 clade (Hassan et al., 2016). In addition, during this period of H5N8 infections, the HA gene included 2 new mutations in the antigenic sites, A and E. Identification of the HA nucleotide sequence ranged from 77 to 90% with various vaccine seeds, and all examined viruses were a reassorted strain of clade 2.3.4.4b. These viruses caused mutations specific to Egyptian strains rather than the original virus identified in 2017 (A/duck/Egypt/F446/2017), with a novel antiviral resistance marker, V27A, representing resistance to amantadine in the M2 protein of 2 strains (Yehia et al., 2020).

In 2019, a study was performed on 32 duck flocks, 4 broiler chicken flocks, and 1 turkey flock, all suffering from respiratory manifestations with moderate to high mortality raised in 2 Egyptian governorates. Real-time RT-PCR screening revealed the existence of HP H5N8 in 21 of the 37 investigated flocks, with mixed infection of H9N2 in 2 of them, while HP H5N1 was not observed (Yehia et al., 2018; Hassan et al., 2020). In 2018, HPAI (H5N2) Egyptian virus EG-VG1099 was detected and submitted to the gene bank database with accession number EPI1387245-52. This virus was closely correlated, the preposition to its HA gene segment, to the HPAI H5N8 viruses discovered in Europe between 2016 and 2017, with a nucleotide sequence identity 98% like the A/EurWig/NL-Greonterp/16015653-001/2016; the same identity of 98% similarity with A/duck/Egypt/F446/2017(H5N8) (Hagag et al., 2019).

Later in Egypt, a new reassorted HPAI (H5N2) virus was detected in 3 poultry flocks, the virus having the genome segments of a pigeon H9N2 AIV recorded in 2014, a nucleoprotein segment of chicken H9N2 from Egypt and HA derived from the 2.3.4.4b H5N8 virus clade (Hassan et al., 2020).

LPAI Viruses LPAI H9N2 is a common avian infection that circulates in the field. Despite H9N2 not being as devastating as HPAI H5, it contributes significantly to the monetary losses experienced by the poultry industry (Peacock et al., 2019). H9N2 belongs to the influenza A viruses (family; orthomyxoviridae) and contains 8 segments of negative-sense RNA (Szewczyk et al., 2014). H9N2 encodes 10 main viral proteins, with HA and NA playing the major roles in immunogenicity (Bouvier and Palese, 2008). Two main branches of H9N2 viruses are commonly circulating worldwide and are named after their Eurasian distribution as the western and eastern branches (Peacock et al., 2019). The Eurasian branch includes the G1-like, Y280-like, G9-like, BJ94-like, Y439-like, and Korean-like clades/lineages (Guo et al., 2000; Kim, 2018).

Variants/sublineages under each lineage are diverse and not highly cross-protective (Peacock et al., 2019). The G1-like H9N2 viruses are further divided into 4 distinct groups designated A, B, C, and D. Importantly, the G1-like clade is the most prevalent H9N2 in the Middle East, with a notable circulation of groups A and B (Fusaro et al., 2011; Naguib et al., 2017). Respiratory illness and reduced egg production are the most observed symptoms accompanying H9N2 infection (Nagy et al., 2017). Nevertheless, the infection could be asymptomatic with no obvious clinical signs, especially in quail and broilers flocks (Nagy et al., 2017).

Some H9N2 LPAI strains can cause high mortalities. This could be attributed to the effect of secondary pathogenic infections (Alexander, 2000). In other instances, the virus's origin has played a role in its pathogenicity. The H9N2 isolated from broiler farms displayed higher mortality rates and increased pathogenicity compared to the virus isolated from layer farms (Eladl et al., 2019). This report represents an exceptional case in Egypt, where the H9N2 virus exhibited strong pathogenic symptoms. Effects of coinfections associated with H9N2 were found to aggravate the pathogenicity of H9N2 (Umar et al., 2017). Certain pathogens were found to enhance the replication and pathogenicity of H9N2 as the infectious bronchitis virus (**IBV**), infectious laryngotracheitis virus (**ILT**), *Escherichia coli, Ornithobacterium rhinotracheitis*, and *Chlamydia psittaci* (Kishida et al., 2004; Pan et al., 2012; Jaleel et al., 2017; Chu et al., 2020).

Notably, reports describing the effect of IBV and ILT coinfections focus on the live vaccines of both IBV and ILT (Haghighat-Jahromi et al., 2008; Arafat et al., 2018). This has great significance for the field as these live vaccines are routinely administered to poultry flocks, and this would act as a risk factor when considering the high prevalence of H9N2. Another explanation for the economic losses caused by H9N2 is the immunosuppression of poultry flocks. This could interfere with the effectiveness of vaccinations against other pathogens (Ellakany et al., 2018; Belkasmi et al., 2020).

Furthermore, this increases the influence of secondary pathogens or the postvaccinal reaction on flock performance (Kishida et al., 2004; Umar et al., 2017; Arafat et al., 2018; Chu et al., 2020). The main trigger for immunosuppression by H9N2 is the virus' pathologic effect on the primary and secondary lymphoid organs,

including degeneration of the bursa and thymus and splenic congestion (Qiang and Youxiang, 2011). Significant decreases in CD^{4+} and CD^{8+} T cells have also been observed during H9N2 infections (Kwon et al., 2008; Qiang and Youxiang, 2011). Egypt is endemic to the G1 lineage (Kandeil et al., 2017; Nagy et al., 2017).

The state of endemicity has been established since 2012, and the first isolates from 2011 to 2014 showed a phylogenetic distribution with subclade B of the G1 lineage (Monne et al., 2013; Naguib et al., 2017). This does not mean Egypt was completely free of the virus before this time; previous introductions of H9N2 were reported (Li et al., 2020). It was proposed that back-and-forth transmission of the virus in the Middle East was frequent (Li et al., 2020). Two reports documented the presence of a reassorted H9N2 that contained 5 internal genes from Eurasian AIV subtypes (Kandeil et al., 2017; Samir et al., 2019).

A novel genotype with a different NP gene (obtained from the Egyptian 2010 viruses) was also reported (Kandeil et al., 2016). In general, 3 major LPAIVs H9N2 have evolved and are circulating in Egypt (Li et al., 2020). The simultaneous circulation of the low pathogenic H9N2 subtype with the H5N8 subtype in Egyptian farms increases the odds that novel reassorted viruses will be generated. This was already documented (Hagag et al., 2019; Hassan et al., 2020), and the evolutionary pressures on the viruses could have been aggravated by the possible introduction of H5N6 and the reemergence of the HPAI H5N1. This makes Egypt one of the most important hot spots for the evolution of AI viruses, especially considering the geographical dimension that many birds' migratory routes across the country. Notably, the Egyptian H9N2 virus' evolution has generated several genetic changes that enhance its transmission to mammals (Kim, 2018).

In terms of prevention and control of AI, it may be difficult to declare freedom from AI diseases due to continuous viral replications in host animals leading to the disease's "silent spread" (Swayne and Halvorson, 2008). There is no single effective prevention and control measure for AI, and many strategies are often combined to achieve these 2 objectives (Spackman and Suarez, 2008). These strategies should include the following steps: biosecurity measures and management practices to minimize AIVs (Capua and Morangon, 2006); awareness of the public health risk; updated surveillance studies helping to certify a country as free of AI as well as to identify an infected location (Dufour, 2001); notification of the authorities, culling, and hygienic elimination of infected birds (Swayne, 2006); vaccination to reduce host susceptibility to the infection to limit or prevent disease spread (Swayne et al., 2000).

Many AI vaccines offer protective value according to the following categories: inactivated whole AIV vaccines, *in vitro* expressed HA vaccines, and *in vivo* expressed HA vaccines (Swayne et al., 2000). Suboptimal use of vaccines would drive antigenic drift and clade replacement (Park et al., 2011; Kim, 2018; Peacock et al., 2019). This would eventually lead to the continuous circulation of AIV in the field and additional opportunities for viruses to evolve and generate novel reassortments (Iqbal et al., 2009).

Consequently, virus pathogenicity could be enhanced (Lau et al., 2016; Eladl et al., 2019) or its zoonotic potential could be increased (Peacock et al., 2017; Sealy et al., 2018). Nanotechnology has recently started generating new tools for poultry disease diagnosis and vaccine preparation, with promising results (Yousry et al., 2020; Abd El-Ghany et al., 2021). The mostly used vaccines against the discussed avian respiratory pathogens are summarized in Table 1.

Newcastle Disease

Newcastle disease virus (**NDV**), alone or accompanied by other viruses or bacteria, is the major etiology of respiratory illness in chicken farms, resulting in high mortality rates (Radwan et al., 2013; Samy and Naguib, 2018). ND is a serious avian pathogen that costs the avian sector billions of dollars worldwide (Alexander, 2000; Cheng et al., 2022). ND is caused by avian avulavirus type-1 (Russell, 1988), which is in the genus avian paramyxovirus-1 (**APMV-1**, a single-stranded RNA genome of negative polarity, encoding 6 structural proteins (Chambers et al., 1986; Wilde et al., 1986).

From 5′ to 3′ end, these include the genes for the nucleocapsid-protein (**NP**), phosphoprotein (**P**), matrix protein (**M**), fusion protein (**F**), hemagglutinin-neuraminidase (**HN**), and large RNA-dependent polymerase (**L**) (Locke et al., 2000; Moharam et al., 2019). The F-protein is the common determinant for the virulence of avulavirus because it is a reliable indicator of variations in tissue tropism in each strain. The recognition pattern of cellular proteases and subsequent cleavage into 2 biologically active fragments of the F-protein is determined by variations in a stretch of amino acids at the C-terminus of the F2 protein and the first amino acid motif at the N-terminus of the F1 protein ($aa^{112-117}$). Low-virulent NDV strains include only 2 basic amino acids in the recognition motif and leucine at the F1 protein's N-terminus (^{112}G-R/K-Q-G-R-L^{117}). Trypsin-like proteases found only in the respiratory and intestinal tract break this motif inside the F0 of NDV extracellularly (Moharam et al., 2019).

Virulent NDV strains, on the other hand, have multibasic amino acids at the F2 protein's C terminus and phenylalanine at the F1 protein's N terminus (^{112}R/G/KRQ/K-K/R-RF^{117}). F0 of vNDV is cleaved intracellularly by ubiquitous furin-like proteases found in most host tissues (Bergfeld et al., 2017). Both mesogenic and velogenic NDV strains have this multibasic recognition pattern. As a result, determining the cleavage location does not distinguish between mesogenic and velogenic proteins. When evaluated by intracerebral pathogenicity index (**ICPI**), several pigeon-type paramyxovirus (**PPMV1**) strains with a proteolytic cleavage site (Dortmans et al., 2010) mimicking a virulent pathotype were shown to have low pathogenicity (Bergfeld et al.,

Table 1. The mostly used vaccines against common avian viral and bacterial respiratory pathogens.

Respiratory pathogens	Vaccine	Types of vaccine	Route of administration	Precautions	Impact	References
Avian influenza (AI)	Homologous (H5N1)	Inactivated	Injection	Sentinel birds should be kept unvaccinated to achieve differentiating infected from vaccinated animals (DIVA) approach. Needs 2−3 booster doses in layers and breeders	Provides a protective immunity to broiler, layers, and breeders flocks	Swayne et al. (2007); Peyre et al. (2009)
Avian influenza (AI)	Heterologous (H5N2, H7N2, H7N3)	Inactivated	Injection	Apply differentiating infected from vaccinated animals (DIVA) approach	Provides a protective immunity to broiler, layers, and breeders flocks	Swayne et al. (2007); Peyre et al. (2009)
Avian influenza (AI)	Recombinant fowl pox	Live	Injection	Repeated annually (single dose per year)	Provides long-term protection of up to 24 wk, post a single dose. No protection was observed if the birds were previously infected with fowl pox virus	Swayne et al. (2007); Peyre et al. (2009)
Avian influenza (AI)	Recombinant (Reverse genetics H5N1)	Live	Injection	Needs 2−3 booster doses in layers and breeders	Provides immunity against avian influenza	Swayne et al. (2007); Peyre et al. (2009)
Avian influenza (AI)	Recombinant avian influenza/Newcastle disease (e.g., H5/ND)	Live	Eye drops	Repeated every 4 mo in breeders and layers	Provides immunity against avian influenza and Newcastle disease	Swayne et al. (2007); Peyre et al. (2009)
Newcastle disease (ND)	Genotype I (I-2,V4, PHY-LMV42, Ulster)	Live	Mass (spray, aerosol, drinking water) or individual (eye drop)	Protection onset (IgY, IgM, IgA) during 2−3-wk postvaccination	Provokes strong cell-mediated immune response, but impacted by maternal derived antibodies levels	Kapczynski et al. (2012); Dai et al. (2015)
Newcastle disease (ND)	Genotype II (LaSota, B1, VG/GA, Clone 30)	Live	Mass (spray, aerosol, drinking water) or individual (eye drop)	Protection onset (IgY, IgM, IgA) during 2−3-wk postvaccination	Provokes strong cell-mediated immune response, but impacted by maternal derived antibodies levels	Kapczynski et al. (2012); Dai et al. (2015); Dimitrov et al. (2017)
Newcastle disease (ND)	Genotype I or II	Inactivated	Injection	Stimulates the production of (IgY, IgM) and the protection onset during 3−4-wk postvaccination	Provides humoral immune response and it was also impacted by the level of maternal antibodies	Kapczynski et al. (2012); Dai et al. (2015); Dimitrov et al. (2017)
Newcastle disease (ND)	Recombinant (fowl pox virus and herpes virus of turkeys were used as vector)	Live	*In ovo*, individual (eye drop, injection-subcutaneous or wing-web) or mass (spray, aerosol) depending on the vector	Stimulates (IgY, IgM, IgA) depending on the vector and route of administration	Stimulates both cellular and humoral immune response against Newcastle disease and the vector virus	Kapczynski et al. (2012); Dai et al. (2015); Dimitrov et al. (2017)
Newcastle disease (ND)	Antigen-antibody complex vaccine (experimental vaccine)	Live	*In ovo* at 18 or 19 d of embryonation	Slows the replication without adversely affecting the hatchability	After the birds hatched, the Newcastle disease virus-specific antibodies were able to separate from the vaccine virus, enabling hatchability that was otherwise impossible with even the least virulent, asymptomatic wild type or recombinant Newcastle disease virus strains given *in ovo*	Kapczynski et al. (2012); Dai et al. (2015); Dimitrov et al. (2017)
Newcastle disease (ND)	Nanoparticle based vaccines	Live	Mass (spray, aerosol, drinking water) or individual (eye drop)	Two chitosan derivatives, O-2'-hydroxypropyltrimethyl ammonium chloride chitosan and N-2-hydroxypropyl trimethyl ammonium chloride chitosan, have been utilized to make nanoparticles as a mucosal delivery vehicle for live attenuated Newcastle disease vaccines	Provides stronger cellular, humoral, and mucosal immune responses	Kapczynski et al. (2012); Dai et al. (2015); Dimitrov et al. (2017)
Infectious bronchitis (IB)	Massachusetts-based M41 serotype, the Dutch H52 and H120 strains	Live attenuated	Mass (spray, aerosol, drinking water) or individual (eye drop)	Live vaccines often are used in broilers and as boosters for breeders	Provides cellular and humoral immune responses	Cavanagh, (2003); Johnson et al. (2003)

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Table 1 (*Continued*)

Respiratory pathogens	Vaccine	Types of vaccine	Route of administration	Precautions	Impact	References
Infectious bronchitis (IB)	Combined vaccines (Live attenuated infectious bronchitis vaccines combined with other virus vaccines such as those against Newcastle disease, Marek's disease virus, and infectious bronchitis virus	Live as (Nobilis IB-Ma5; AviPro IB H120; Nobilis 4−91; Gallivac CR88; POULVAC IB QX)	Mass (spray, aerosol, drinking water) or individual (eye drop)	Used for broilers, layers, and breeders	Provides cellular and humoral immune responses	Cavanagh, (2003); Johnson et al. (2003)
Infectious bronchitis (IB)	Monovalent or combined	Inactivated	Injection	Used for layers and breeders at 13−18 wk of age	Provides maternal derived antibodies to protect progeny	Cavanagh, (2003); Johnson et al. (2003)
Infectious bronchitis (IB)	Recombinant adenovirus vaccine containing infectious bronchitis virus-S1-glycoprotein	Live	Mass (spray, aerosol, drinking water) or individual (eye drop)	Provides a protective antibody titer	Provides 90−100% protection	Cavanagh, (2003); Johnson et al. (2003
Infectious bronchitis (IB)	Recombinant fowl pox virus vaccine expressing infectious bronchitis virus-S1-gene and chicken interferon-γ gene [rFPV-IFNγS1]	Live	*In ovo*, individual (eye drop, injection-subcutaneous or wing-web) or mass (spray, aerosol)	Provides immunity	Enhances humoral and cell-mediated immune responses that protects chickens against homologous and heterologous challenge with LX4, LHLJ04XI, and LHB infectious bronchitis virus strains	Cavanagh, (2003); Johnson et al. (2003
Infectious bronchitis (IB)	Subunit and peptide-based vaccines (multiple epitopes from S1- and N-protein genes)	Live	*In ovo*, individual (eye drop, injection-subcutaneous or wing-web) or mass (spray, aerosol) depending on the vector	Provides immunity	Provides protective humoral and cell-mediated immune responses	Kapczynski et al. (2003); Yang et al. (2009) ; Bande et al. (2015)
Infectious bronchitis (IB)	Plasmid DNA vaccines (A DNA vaccine designated pDKArkS1-DP has been developed, based on the S1-genes of Arkansas infectious bronchitis virus serotypes)	Reverse genetic-based live attenuated infectious bronchitis virus vaccines	*In ovo*	This was followed by immunization with a live attenuated vaccine at 2-wk intervals	Provides 100% protection against clinical disease	Kapczynski et al. (2003); Yang et al. (2009) ; Bande et al. (2015)
Infectious bronchitis (IB)	Reverse genetic vaccines (BeauR- infectious bronchitis virus vaccine, modified H120 (R-H120)	Live	*In ovo*, individual (eye drop, injection-subcutaneous or wing-web) or mass (spray, aerosol) depending on the vector	Provides immune response	More research is needed to determine whether these more recent immunizations will make mutation and viral selection pressure	Kapczynski et al. (2003); Yang et al. (2009) ; Bande et al. (2015)
Mycoplasma	AviPro104 MG BACTERIN	Bacterin, strain R	Injection	For layer and breeder flocks	Provides maternal immunity to protect progeny at first 2 wk of life	Ishfaq et al. (2020); El-Naggar et al. (2022); Marouf et al. (2022b)
Mycoplasma	MG-Bac vaccine	Bacterin	Injection	For layer and breeder flocks	Provides maternal immunity to protect progeny at first 2 wk of life	El-Naggar et al. (2022); Marouf et al. (2022b)
Mycoplasma	Bivalent MG and MS	Bacterin	Injection	For layer and breeder flocks	Provides maternal immunity to protect progeny at first 2 wk of life	Ishfaq et al. (2020); El-Naggar et al. (2022)
Mycoplasma	Pentavalent MG, MS, and other *Salmonella* species	Bacterin	Injection	For layer and breeder flocks	Provides maternal immunity to protect progeny at first 2 wk of life	Ishfaq et al. (2020); Marouf et al. (2022b)
Mycoplasma	Vaxsafe MG (Strain TS-11)	Live-attenuated vaccine, strain ts-11	Mass (spray, aerosol, drinking water) or individual (eye drop)	For layer and breeder flocks	Provides protective antibody titer but, some vaccines showed vaccinal strain shedding hazard	Ishfaq et al. (2020); El-Naggar et al. (2022)
Mycoplasma	*Mycoplasma gallisepticum* vaccine	Live-attenuated vaccine, strain ts-11	Mass (spray, aerosol, drinking water) or individual (eye drop)	For layer and breeder flocks	Provides protective antibody titer but, some vaccines showed vaccinal strain shedding hazard	Ishfaq et al. (2020); El-Naggar et al. (2022); Marouf et al. (2022b)

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Table 1 (*Continued*)

Respiratory pathogens	Vaccine	Types of vaccine	Route of administration	Precautions	Impact	References
Mycoplasma	*Mycoplasma gallisepticum* vaccine (TS-11)	Live vaccine strain ts-11	Mass (spray, aerosol, drinking water) or individual (eye drop)	For layer and breeder flocks	Provides protective antibody titer but, some vaccines showed vaccinal strain shedding hazard	El-Naggar et al. (2022); Marouf et al. (2022b)
Mycoplasma	CEVAC MG F	Live-attenuated vaccine, strain F	Mass (spray, aerosol, drinking water) or individual (eye drop)	For layer and breeder flocks	Provides protective antibody titer but, some vaccines showed vaccinal strain shedding hazard	Ishfaq et al. (2020); Marouf et al. (2022b)
Mycoplasma	Nobilis MG 6/85	Live vaccine strain 6/85	Mass (spray, aerosol, drinking water) or individual (eye drop)	For layer and breeder flocks	Provides protective antibody titer but, some vaccines showed vaccinal strain shedding hazard	Ishfaq et al. (2020); Marouf et al. (2022b)
Mycoplasma	*Mycoplasma gallisepticum* vaccines	Live-attenuated vaccine, strain R, strain F-36	Mass (spray, aerosol, drinking water) or individual (eye drop)	For layer and breeder flocks	Provides protective antibody titer but, some vaccines showed vaccinal strain shedding hazard	Ishfaq et al. (2020); El-Naggar et al. (2022)
Mycoplasma	VAXSAFE MG VACCINE	Live-attenuated vaccine, strain ts-11	Mass (spray, aerosol, drinking water) or individual (eye drop)	For layer and breeder flocks	Provides protective antibody titer but, some vaccines showed vaccinal strain shedding hazard	Ishfaq et al. (2020); El-Naggar et al. (2022); Marouf et al. (2022b)
Escherichia coli	Nobilis *E. coli*	Inactivated subunit vaccine consisting of fimbrial antigen F11 and a flagellar toxin	Injection	For breeders	Protect progeny against *E. coli* challenge	Swelum et al. (2021)
Escherichia coli	Poulvac *E. coli*	Live attenuated commercial vaccine	Coarse spray	Apply for 1-day-old chick	Protects birds against homologous and some heterologous *E. coli* serotypes challenge	Swelum et al. (2021)
Escherichia coli	*E. coli* EC34195	Live attenuated *E. coli* EC34195 serotype O78 with deleted aroA gene as the active substance	Coarse spray	Apply for 1-day-old chick	Protects birds against homologous and some heterologous *E. coli* serotypes challenge	Swelum et al. (2021)
Infectious coryza	Tetravalent coryza vaccine	Bacterin including *Haemophilus paragallinarum* serotypes A, B, C and variant type B	Injection	For layer and breeder flocks	Provides a protective antibody titer against field challenge	Jacobs et al. (2003); Jackwood and Saif (2008)
Infectious coryza	Whole-cell bacterin ADJUVAC-ART	Bacterin	Injection	For layer and breeder flocks	Provides protective antibody titer against field challenge	Jacobs et al. (2003); Jackwood and Saif (2008)
Infectious coryza	Live temperature-sensitive (ts) mutant of *Bordetella avium*	Live	Spray, eyedrop		Induces moderate levels of serum antibodies	Jacobs et al. (2003); Jackwood and Saif (2008)
Pasteurella multocida	*P. multocida* vaccine serotypes A:1, A:2 and A:3	Inactivated	Injection (S/C of wattle)	For layer and breeder flocks	Provides protective antibody titer against field challenge	Mostaan et al. (2020)
Pasteurella multocida	*P. multocida*, temperature-sensitive mutant-*P. multocida* cexA mutant (PBA875) and acapsular *P. multocida* strain (AL18)	Live attenuated	Spray or drinking water	For layer and breeder flocks	They may have serious drawbacks such as causing systemic infection, disease outbreaks, no or weak protection against fowl cholera in chickens, and weight gain but induces heterologous protection, cellular immunity induction, and showed longer lasting protection	Mostaan et al. (2020)

2017). This suggests that other factors may influence pathogenicity in addition to the F protein cleavage location (Bergfeld et al., 2017).

There are different classifications of NDVs based on antigenic and genotypic variations of their fusion protein and gene, respectively (Collins et al., 1993; Capua and Alexander, 2004). Of particular interest is the work by Czeglédi et al. (2006), where the authors described 2 classes of NDV (class I and class II) based on genomic data and sequencing of the fusion (F) and RNA-directed RNA polymerase (L) genes. These 2 classes contain all the genotypes and lineages already reported. Aldous et al. (2003) classified all avirulent PPMV-1 strains into lineage one. The clinical course can range from acute to subacute or chronic and is known by symptoms in the respiratory and digestive systems and nervous lesions occurring later in the course of the disease. The mortality rate associated with this lineage can be as high as 100%. The World Health Organization has designated it a notifiable animal illness (OIE, 2021). NDV is spread by wild birds (Alexander, 2000) and thus management strategies include mandatory vaccination and a no-kill policy (Capua and Alexander, 2004).

In Egypt, ND has been a dangerous avian infection over the last decade, particularly in broilers (Orabi et al., 2017; Abd El-Hamid et al., 2020). Respiratory discomfort and neurological problems are the most observed symptoms in diseased farms. These symptoms include eye lacrimation, sneezing, rales, cough, and respiratory difficulties. Nerval inflictions such as tilting and torticollis are also often observed (El-Bagoury et al., 2015). Mortality rates tend to be between 15 and 20% but can reach up to 80% or more, even in vaccinated flocks (Osman et al., 2014; El-Bagoury et al., 2015).

In this regard, it is vital to remember that ND is diagnosed mostly from clinical indications and postmortems. On the other hand, virus detection is limited to a general detection of NDV without differentiation of pathotype. As a result, clinical indications of ND outbreaks in vaccinated farms may be masked or generated by other infections, with NDV being identified as the vaccine-type virus (Zanaty et al., 2019).

ND was first detected in Egypt in 1948 (Daubney and Mansy, 1948). NDV genotypes II and VII have been recorded (Radwan et al., 2013) and are considered prevalent strains (Mohamed et al., 2009). As well as domestic fowl, NDV infection has been observed in pigeons and numerous wild bird species (Moharam et al., 2019). Moreover, coinfections with IB and AI viruses have been reported in Egypt (Naguib et al., 2017; Samy and Naguib, 2018; Moharam et al., 2019). Currently, at least 3 NDV genotypes are circulating in Egypt. As well as vaccine-type genotype 2.II viruses, there is virulent genotype 2.VIIb, 2.VIIg, and 2.VIIb. Genotype 2.VIIb and 2.VIIg has been isolated from infected pigeons and is regarded as pigeon paramyxovirus (**PPMV**) (Rohaim et al., 2016).

Genotype 2.VIIb, in contrast, is relevant to poultry, with outbreaks of ND occurring in all avian sectors. Despite broadscale vaccinations with genotype 2.II vaccinations, substantial mortality has occurred, particularly in broiler farms (Orabi et al., 2017; Abd El-Hamid et al., 2020). Genotype VII is thought to have originated in the Far East and expanded to other parts of the world (Herczeg et al., 1999).

Currently, genotype VII is predominant among velogenic NDV and can be further subdivided (Dimitrov et al., 2019) into sublineage VII.1. This sublineage encompasses viruses that emerged in the 1990s in the Far East, Europe, Asia, and the Middle East, and were responsible for the fourth NDV panzootic and VII.2 viruses which emerged in Indonesia, attacking Asia, the Middle East, Europe, and Africa. These viruses were also accountable for the fifth NDV panzootic (Miller et al., 2015).

In Egypt, however, despite routine ND vaccinations, NDV genotypes II, VI, and VII.1.1 have been recorded continuously over the last 10 years (Orabi et al., 2017), based on the novel classification scheme suggested by Dimitrov et al. (2019). The last sequenced ND virus indicated that all the viruses belong to subgenotype 2. VII.1.1 (formerly 2.VII-b), the dominant NDV genotype in the Middle East and northern Africa (Dimitrov et al., 2019). Because of Egypt's endemic ND condition, which is projected to result in significant economic losses, aggressive immunization efforts are being widely implemented for the control and prevention of ND (Bastami et al., 2018).

Vaccination protocols for broilers comprise at least 2 live vaccine administrations during the 35- to 40-days fattening period, with one inactivated vaccine in between. An aggressive vaccination strategy for layers and breeders is employed throughout the rearing of birds, with continuous use of live and killed vaccines, which further vaccinations may follow during the production phase as deemed appropriate (Bastami et al., 2018).

Nonetheless, ND outbreaks are recorded regularly and result in substantial losses of birds, particularly broilers. The presence of NDV in these flocks is regarded as a sign that vaccination and hygiene efforts have failed (El-Bagoury et al., 2015). This has raised questions about the effectiveness of the country's ND vaccination programs. It has been theorized that accumulating mutations are to blame, similar to the highly virulent AI virus (Moharam et al., 2019). New antigenic variations of the NDV have emerged, enhancing its capacity to spread among vaccinated people. As a result, viruses that outcompete earlier virus populations have acquired Darwinian fitness, as evidenced by mutations at antigenically significant areas (Moharam et al., 2019).

IBV

IBV belongs to coronaviridae, genus gamma coronavirus (Cavanagh, 2003; Klestova et al., 2022). IBV is a positive sense, single-stranded nonsegmented RNA genome, about 27.6 kb in length. This genome encodes 4 structural proteins: nucleocapsid (**N**), membrane (**M**),

envelope (**E**), and spike (**S**) (Jin-Ling et al., 2011). S protein is a major structural protein divided into 2 small protein subunits (S1 and S2) and is the most variable gene. The sequence of the S1 gene can be employed to distinguish IBVs into 6 IBV genotypes (GI−GVI), and 32 lineages (1−32) that have been detected all over the world (Valastro et al., 2016).

In general, different serotypes do not have cross-protection against each other, so the classification of IBV strains about their "protectotype" has been proposed (Dhinakar and Jones, 1996). The various serotypes, subtypes, and variations of IBVs are most likely the consequence of nucleotide point mutations, insertions, deletions, and recombinations and cause outbreaks in vaccinated chicken flocks (Mahmood et al., 2011) or a response in the form of mass vaccinations, which might contribute to immunological stress and the generation of novel variations (Umar et al., 2016). The formation of novel IBV strains results from many virus types being infected simultaneously, and the use of live vaccines makes vaccination only partially effective because of the antigenic variations (Bayry et al., 2005).

IB is a contagious respiratory infection that results in considerable financial loss for the avian sector (Butcher et al., 2011). Chickens are the natural host of IBV. Disease severity varies according to age, being most severe in young chicks. IBV is an air-borne infection transmitted horizontally by direct contact with diseased chickens or indirect contact (by mechanical spread, like contaminated equipment, workers, and trucks) (Cavanagh et al., 2002).

IBV is a respiratory infection because it multiplies in the tracheal epithelium causing respiratory illness (nasal discharges, sneezing, cough, tracheal rales, and gasping) (Khataby et al., 2016). Recently, IBV has been reported with kidney and reproductive lesions, presenting as nephritis and severe atrophy of the oviduct, resulting in lowered or permanent loss of egg production (Balestrin et al., 2014). Mortality rates ranged from 14 to 82% according to the serotype and coinfections (Knoetze et al., 2014). IBV is a worldwide infection, and it has been detected in Africa, Asia, Australia, Europe, and the Americas (Cook et al., 2012; Jackwood, 2012). It was first recognized in North Africa, particularly in Egypt, in the 1950s by Ahmed (1954) in birds with respiratory manifestations.

In Egypt, IB has been accompanied by different illnesses involving respiratory and renal problems and a decrease in egg production, resulting in huge economic losses in the Egyptian poultry sector (Eid, 1998). Coinfections of avian respiratory viruses, including IBV may induce similar illnesses (Nguyen et al., 2013). In Egypt, IBV isolates were closely correlated to Massachusetts (Mass), Dutch D3128, D274, D-08880, and 4/91 variants (Bastami et al., 2018).

Subsequently, in 1998, the Egyptian variant "Egypt/BeniSeuf/01" was recovered from broiler farms vaccinated with H120. This IBV strain was defined as the Egyptian variant-1 closely related to the nephron-pathogenic Israeli strains (IS/720/99 and IS/885/00) (Abd El-Moneim et al., 2002). One year later, serological characterization and genotyping using S1 gene sequence analysis revealed that the Egyptian strain of IB "Egypt\F/03" is closely related to the Massachusetts serotype Beaudette-US, H120, and M41, with high nucleotide sequence identity (Abd El-Moneim et al., 2006). El-Mahdy et al. (2010) also showed that the Egyptian IB nephron-pathogenic strain was correlated to the IS/1494/06 variant strain. Recent studies have revealed that IBV circulating in Egypt during 2012 was classified into 2 variant groups. The first group comprised variants from IS/885, and the second group was related to variant vaccine strain viruses like 4/91 and CR/88121 (Selim et al., 2013). In addition, 2 emerging variants, IBV-CU-2-sp1 and Eg/12120s/2012 were isolated from different Egyptian poultry farms by Selim et al. (2013). AlBeltagi et al. (2014) showed that the Egyptian isolates recovered from broiler chickens with severe renal and respiratory illness could be considered as a variant 2. Thus, there were 2 Egyptian variant subgroups (Egyptian variant-1 and Egyptian variant-2).

Sultan et al. (2015) demonstrated the extensive circulation of multiple genotypes of IBV in Egyptian chickens, often combined with other respiratory viruses. IBV strains were categorized by cluster analysis based on phylogeny into 4 groups: variant-2, variant-1, Mass, and the 793B groups, where the Mass group related to vaccines H120, and Ma5 and 793B related to vaccines CR88 and 4/91 were used in Egyptian chickens as commercial live vaccines. Some studies showed that IBV strains documented in 2017−2018 should be classified into 2 groups where the IBV-Ch-KafrAshaykh-2017 isolate clusters with a group (Egy/variant 1) of the GI-23 lineage of IS/1494/06 genotype origin. The IBV-Ch-Sharkia-2018 isolate was more closely related to IB vaccine strains like the Mass type, which points to independent IBV evolution and persistence of divergent IBV strains occurring in Egypt (Esmaiel et al., 2019).

Hammouda and Arafa (2019) also stated that IBV isolates circulating in Egypt are related to the variant-2 group, and closely related to both the IBVEg/1265B/2012 strain and the Israeli strain IS/1494/06. Later, Setta et al. (2018) reported that IBV genotypes closely correlated to variant II strains Eg/12120S/2012, IS/885, and IS/1494, with 4 isolates clustered in a new group. Recently, based on the diversity of the full S1 sequence, IBV strains have been classified into 7 genotypes (GI−GVII) with dozens of genetic lineages. Genotype GI has the largest number of genetic lineages (n: 29). The Mass type belongs to the GI-1 lineage, while the Egyptian variants 1 and 2 belong to the GI-23 lineage, which has also spread to many countries in Africa, Asia, and Europe (Ma et al., 2019; Moharam et al., 2020).

IBV vaccines are live attenuated or inactivated from classical or variant serotypes. Mass-type and variant vaccine strains have provided comprehensive protection in chickens. The lack of cross-protection between imported vaccinal and field strains may explain the

failure to establish a full vaccination program (Toro et al., 2015). Establishing a universal vaccination program against IBV is restricted due to the circulating evolution of several serotypes and new genotypes of IBV and the resultant limited cross-protection (Fathy et al., 2019).

Respiratory issues and nephropathy accompany many IBV infections, and high mortality rates are mainly attributed to the nephron-pathogenic IBV variant-2 strains (Hassan et al., 2016; Abozeid et al., 2017). IBV vaccines are live-attenuated or killed vaccines derived from classical or variant strains. In Egypt, inactivated vaccines are of one type, Mass (M41) (Jansen et al., 2006). Recent studies have shown that the efficacy of attenuated vaccines varies from 90 to 100% protection when compared to a homologous challenge, based on the cryostasis score and percentage protection (Ali et al., 2018).

The efficacy of the inactivated IBV strains (classical H120 and variant-2 IS/1494/06) as vaccines is high, with 96% homologous protection (Fathy et al., 2019). Another protection and control attempt aimed to extend the protective effect of IBV vaccines by vaccination with imported variant IBV. However, this level of efficacy still does not meet the target level of protection (Hassan et al., 2016). As a result of the continuous spread of the IBV variant-2 serotypes in Egypt (El-Mahdy et al., 2010), a live-attenuated VAR2 vaccine was produced from the classic IBV strains (IBM41 and IB2) and a nephron-pathogenic strain (IBvar2).

The IB syndrome clinical signs and pathognomonic postmortem lesions of different forms (respiratory, renal, and reproductive) are presented in Figure 2.

MOST COMMON AVIAN RESPIRATORY INFECTIONS CAUSED BY BACTERIA

Mycoplasma (Mycoplasmosis)

Mycoplasma is very small size bacterium, represented by more than 25 types. They have no cell wall and are only surrounded by plasma membrane. The most common economically serious types are *Mycoplasma gallisepticum* (**MG**) and *Mycoplasma synoviae* (**MS**) (Raviv and Ley, 2013; Marouf et al., 2022a). They have a hemagglutinating effect against chicken and turkey RBCs (Bradbury, 1984; Samy and Naguib, 2018). MG is causing a subclinical upper respiratory disease that is typically nearly asymptomatic however, symptoms can appear and be severe if the infection is accompanied by other respiratory diseases, including AI, ND, IBV, *E. coli* infection, and infectious synovitis when it is systemic (Lockaby et al., 1999). *Mycoplasma* reduce the activity and power of the immune system against other diseases, leading to high morbidity and mortality in chickens with chronic respiratory disease (**CRD**), reducing bird performance and increasing condemnation rates at the time of sale (Papazisi et al., 2003; Osman et al., 2009).

MG destructs respiratory system cilia in turkeys producing hyperplasia of epithelial cells, a pathology that is severely exaggerated in the presence of other live respiratory vaccines (Sid et al., 2016), revealing rales, coughing, sneezing, and eye lesions, and infraorbital sinusitis, in addition to air saculitis which increases condemnation rate of carcasses, deterioration of egg production curves, growth retardation, and economic losses due to medication costs (Raviv and Ley, 2013; Felice et al., 2020).

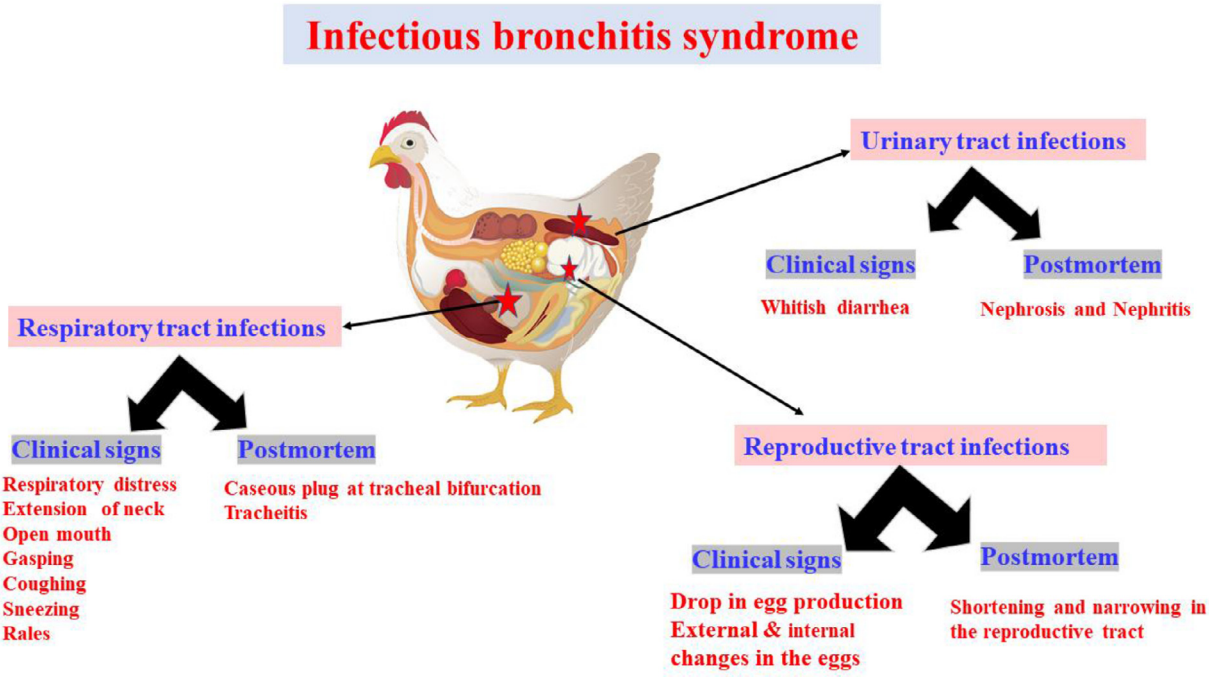


Figure 2. Infectious bronchitis syndrome and its clinical signs and pathognomonic postmortem lesions of different forms (respiratory, renal, and reproductive).

There are many reports on the prevalence of *Mycoplasma* in Egypt from broilers. Applying duplex PCR for recognition of MG and MS, Emam et al. (2020) mentioned that the greatest prevalence of *Mycoplasma* was in winter and autumn (9.85% for MG and 1.6% for MS). Osman et al. (2009) reported infection with MG in layer flocks (33.3%), broiler breeders (30.5%), and broilers (4.9%). Ten years later, Abd El-Hamid et al. (2019) reported that the total isolation rates of *Mycoplasma* species from tested chicken and turkey flocks were 29.3 and 30%, respectively, especially from tracheal samples by 29.4%. Mahmoud et al. (2016) surveyed MG in Sharkia, Kaliobia, Gharbia, Monufia, Giza, Faiyum, and Minya governorates, finding that incidence was from 70.9% according to PCR on 48-h PPLO broth, 65.45% based on direct PCR of infected tissue, however only 17.66% according to traditional methods.

Slide plate agglutination (**SPA**) examination is the most used test for detecting *Mycoplasma* in field cases. Using this approach, the incidence of digitonin-sensitive *Mycoplasma* was 25.4%. Digitonin-sensitive isolates were serotyped into MG (56.2%) and MS (31.2%), and using PCR mgc2 and 16S ribosomal RNA genes, they were positive for MG (53.7%) and MS (24.3%) (Marouf et al., 2020; Abd El-Hack et al., 2022b). Recently, El-Ashram et al. (2021) used SPA, which resulted in MG and MS of 10.9 and 13.2%, respectively, in Giza, and using the hemagglutination inhibition (HI) test, it showed the same percentage. Through the minimal inhibitory concentration (**MIC**) test, MG showed sensitivity to tilmicosin, tiamulin, and spiramycin (Emam et al., 2020).

MG control strains, typically in the form of inactivated vaccines (killed organisms with oil-emulsion adjuvant), are used to prevent *Mycoplasma* infection (El-Naggar et al., 2022). Live attenuated vaccine (live F strain MG vaccine), which is a mild strain prepared from the Connecticut F strain, and the ts-11 MG vaccine prepared from the Australian MG field isolate (strain 80083), are also effective vaccines used for the prevention of *Mycoplasma* infection (Raviv and Ley, 2013).

The transmission of avian mycoplasma, by both vertical route (from infected hens to its progeny) and horizontal route (from infected chickens to susceptible ones) is presented in Figure 3.

Staphylococcus (Staphylococcosis)

Staphylococcus is a gram-positive bacterium that infects most poultry species and is a normal flora on mucous membranes and skin (Szafraniec et al., 2022). *Staphylococcus* has a clumping factor that forms blood clots by converting the soluble blood protein fibrinogen into insoluble fibrin molecule. The bacterium is hidden inside fibrin clots from phagocytic cells, in addition to protein A, which binds to a portion (**FC**) of immunoglobulin and consequently inhibits the phagocytosis of the bacteria. Even though *Staphylococcus aureus* is the most common cause of avian staphylococcosis, other *Staphylococcus* species have been recovered from skeletal lesions, including *S. epidermidis, S. agnetis, S. cohnii, S. simulans*, and *S. hyicus* (Szafraniec et al., 2022). Lebdah et al. (2015) isolated *S. aureus* and *S. lentus* form broiler chickens suffering from leg infections.

Eid et al. (2019) isolated *S. aureus* from diseased ducks (12.2%), while Ammar et al. (2016) recorded *S. aureus* in poultry farms with a prevalence of 3.77%. However, Lebdah et al. (2015) found that *S. aureus* isolates were sensitive to cefotaxim, ciprofloxacin, enrofloxacin, and sulpha trimethoprim. Antibiotic sensitivity tests should be applied, however, before any treatment strategies against *S. aureus* infections to avoid the development of antibiotic drug resistance strains (Lebdah et al., 2015).

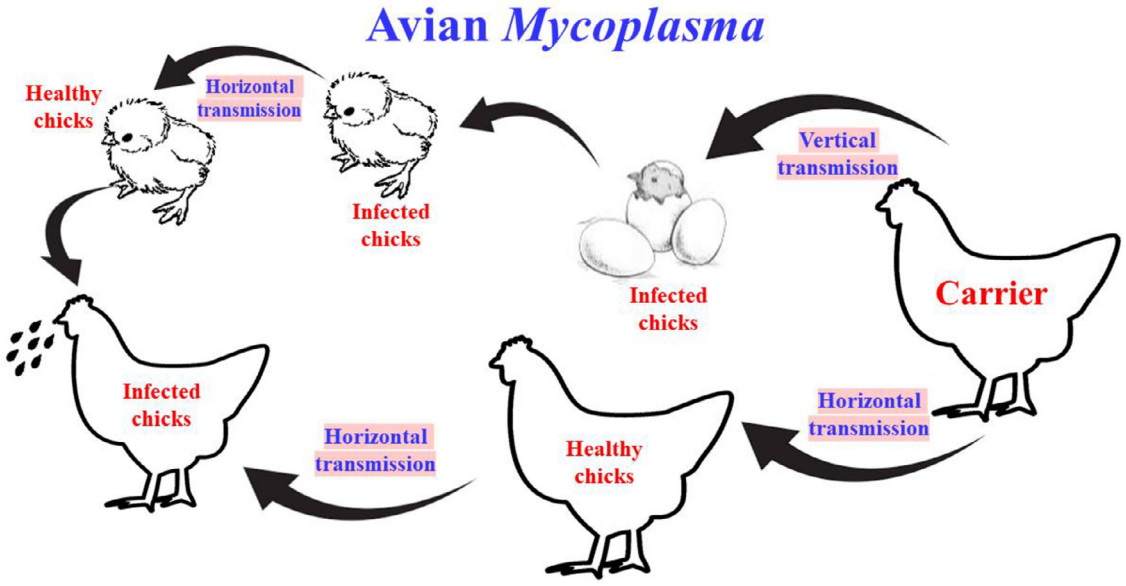


Figure 3. The transmission of avian *Mycoplasma*, by both vertical route from infected chickens to its progeny, and horizontal route from infected chickens to susceptible chickens.

Regarding confection with AIVs, *Staphylococcus* species produce soluble proteases that can activate the HA of AIVs (Scheiblauer et al., 1992).

Bordetella (Turkey Coryza or Bordetellosis)

Turkey coryza is a serious respiratory infection caused by *Bordetella avium*, known as bordetellosis. It infects all ages of turkeys and is associated with high morbidity, reaching 100% but with low mortalities (Jackwood and Saif, 2008). *B. avium* causes a marked reduction in weight gain and, consequently, high losses in the poultry sector (Jackwood and Saif, 2008). The symptoms of turkey coryza are restricted to the respiratory systems involving nasal and eye discharge, sneezing, coughing, and submaxillary edema; the disease lasts about 2 to 4 wk (Jackwood and Saif, 2008). Postmortem findings involve air-saculitis, pneumonia, and tracheitis bronchitis, with exudates in the nasal cavity and trachea without lesions in the internal organs, except for complicated cases with secondary infections (Jackwood and Saif, 2008). *B. avium* was reported in Egyptian turkey flocks. Two strains were isolated from 21 turkey farms in 5 Egyptian localities (Erfan et al., 2018). Saad Eldin et al. (2020) recorded *B. avium* infection in turkeys in Egypt of various ages using PCR. The overall PCR-confirmed prevalence of *B. avium* was 22.95%. The identified *B. avium* strains harbored virulence-associated genes accountable for colonization in the respiratory tract of turkeys involving *B. avium* virulence gene (100%), fimbriae (71.14%), and filamentous hemagglutinin (85.68%).

Recently, Gütgemann et al. (2022) tested the sensitivity of *B. avium* against some antibiotics and the tested strains were sensitive to streptomycin, florfenicol, tetracyclines, and trimethoprim/sulfamethoxazole. Prevention and control, biosecurity and antimicrobial sensitivity testing, and vaccination are all ways to control this disease. A commercial vaccine is available, but not yet in Egypt (Boulianne et al., 2013).

Chlamydophila psittaci (Chlamydiosis)

Avian chlamydiosis (**AC**) is a disease of avian species caused by species of the genus *Chlamydia* (OIE, 2021). *C. psittaci* is an obligate intracellular bacterium that attacks the respiratory system of poultry, although it is known both as psittacosis and parrot fever. It has zoonotic importance because it causes human ornithosis (Chau et al., 2015; Dumke et al., 2015; Knittler and Sachse, 2015). In addition, the respiratory system infection symptomatic of avian chlamydiosis in poultry varies from acute to chronic forms, resulting in severe economic losses for the poultry farming industry (Taylor-Brown et al., 2015). It threatens public health and causes outbreaks due to pneumonia (Bommana and Polkinghorne, 2019).

The prevalence of *C. psittaci* was 29.91% according to a complement fixation test. PCR was then conducted on the samples for confirmation. This was all undertaken on serologically positive, apparently healthy chickens, but they had been in contact with small ruminants exposed to abortion and were positive for *C. abortus* (Osman et al., 2007).

Hegazy et al. (2014) demonstrated that the highest incidence of avian chlamydiosis was detected in chickens (92%) followed by ducks (88%), turkeys (76%), and pigeons (72%). The highest percentage of *Chlamydia* infection was in chickens and turkeys (91.6%) based on PCR reactions and 83.3 and 75% based on direct immunofluorescence tests, followed by ducks and pigeons. A complement fixation test was carried out on 48 fecal swabs; the proportion of *Chlamydia* shedding in chickens, pigeons, ducks, and turkeys was 91.6, 83.3, 75, and 66.6%, respectively. Sorour et al. (2014) recorded the prevalence of *Chlamydia* infections in different breeds and ages of pigeons from various localities in Egypt. They found that the prevalence was 15 to 40% in tested pigeons, with a higher incidence of infection in diseased pigeons (72.72%), while no *C. psittaci* was isolated from apparently healthy pigeons (0%).

El-Jakee et al. (2017) recorded incidence of *C. psittaci* of 74.5, 79.2, 5.6, and 17.5% in turkeys, pigeons, ducks, and chickens, respectively, at a local commercial market in Egypt. Using PCR techniques, Amal et al. (2021) detected *C. psittaci* in different avian species. The incidence of infection was 91.7%, pigeon (86.7%), quail (85.7%), and duck (80%) in fecal samples. All isolates were positive for the 16S ribosomal RNA gene of *C. psittaci*. Ruiz-Laiton et al. (2022) estimated the prevalence of *C. psittaci* in parrots to be 144 of 177 tested individuals (81.3%). While vaccines are not available in Egypt (Sorour et al., 2014), for the control and prevention of infection, quarantine and testing of imported birds for the elimination of disease with the application of biosecurity measures are essential procedures (Matsui et al., 2008).

Avian chlamydiosis host range (pigeons, ducks, turkey, chickens, and psittacine birds) with its zoonotic importance is presented in Figure 4.

Ornithobacterium rhinotracheale

O. rhinotracheale (**ORT**) is a gram-negative, nonmotile, nonsporulating, rod-shaped, pleomorphic bacteria of the rRNA superfamily V, belonging to the genera of *Cytophaga, Riemerella*, and *Flavobacterium* (Vandamme et al., 1994). ORT has been linked to poultry respiratory sickness worldwide (Hashish et al., 2022). It can infect all avian species, especially chickens and turkeys (Chin et al., 2008). It is transmitted horizontally through air or water and is robust to low temperatures; it has a higher incidence in winter and is commonly found with other respiratory pathogens (Chin et al., 2008).

Vertical transmission occurs little from reproductive organs, hatching eggs, infertile eggs, or dead embryos (Shahata et al., 2006; Chin et al., 2008). ORT produces

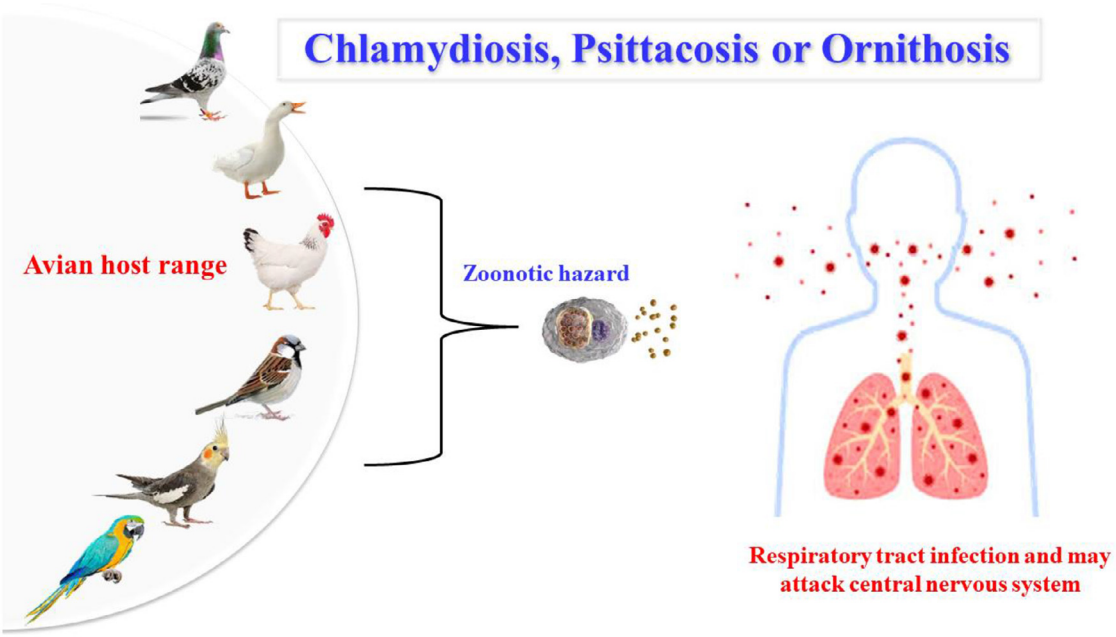


Figure 4. Avian chlamydiosis host range (pigeons, ducks, turkey, chickens, and psittacine birds) with its zoonotic importance.

respiratory signs such as purulent nasal discharge, head swelling, weakness, respiratory distress, air sacculitis, and pneumonia. On postmortem, ORT infections are associated with congestion and consolidation of the lungs (unilateral or bilateral), liver congestion, and spleen enlargement (Chin et al., 2008; Masoud et al., 2015).

ORT can induce severe signs, particularly in cases of vaccination, with the living Newcastle attenuated Lasota vaccine (Hassan et al., 2020). When ORT was experimentally challenged with live attenuated variant IBV 4/91, this results in a reduction in body weight and enhanced feed conversion ratio (Ellakany et al., 2018). In Assiut governorate in Egypt, ORT was found in 32 of 180 samples (17.77%). Lung samples showed the highest incidence (28.3%), then tracheal samples (10%), and the lowest incidence was from air sac samples (15%). PCR confirmed only 6 positive samples (3.33%), and all samples were shown to be serotype A by serological testing (Hassan et al., 2020).

Masoud et al. (2015) confirmed 5 ORT isolates of 16S ribosomal RNA. Moreover, Jamali et al. (2015) examined ORT broiler farms and found 20.9 and 20% prevalence in layer flocks. Masoud et al. (2015) found that ORT isolates were sensitive to lincospectin (lincomycin + spectinomycin) and doxycycline. They also found that many isolates revealed resistance to kanamycin, norfloxacin, enrofloxacin, tetracycline, and chloramphenicol.

Awad et al. (2022) found that in infected broiler chickens, ORT isolates were sensitive to aivlosin with zinc oxide nanoparticles (**ZnO-NPs**). For vaccination, commercial infectious coryza (**IC**) bacterins usually require at least 10^8 colony forming units/mL to be potent. Moreover, there is a mix of bacterins between inactivated viruses and *Avibacterium paragallinarum* (Blackall and Soriano-Vargas, 2013).

Pasteurlla multocida (Fowl Cholera)

Pasteurella multocida causes fowl cholera, a contagious pathogen that infects all birds, causing high mortality and financial losses for commercial and backyard poultry producers (Blackall et al., 2005). *P. multocida* has 4 capsular serogroups in poultry species: A, B, D, and F (Glisson et al., 2008). Strains of serogroup A represent the main serogroup of fowl cholera. There are 16 somatic serotypes (1−16), most of which are found in serogroup A (Glisson et al., 2008).

P. multocida serotypes A:1, A:3, and A:3, 4 are commonly responsible for most cases of fowl cholera in poultry farms (Glisson et al., 2008). In 2017, *P. haemolytica* infections were reported in commercial layer chickens in Egypt (Setta et al., 2017). In the acute form, the signs of infection are fever, loss of appetite, ruffled feathers, unthriftiness, mucous discharge from the mouth, respiratory signs, and watery and whitish diarrhea which turns into a mucoid green color while in chronic form, swelling of wattles, sinuses, leg or foot pads, wing joints, and sternal burse, conjunctival exudate, torticollis, dyspnea, and also tracheal rales were commonly observed (Glisson et al., 2013).

In Egypt, 21 isolates of *P. multocida* were obtained from 275 chickens (7.6%) and assessed by phenotypic characteristics. Somatic serotyping of these 21 isolates revealed 12 isolates being classed as serotype A:1 (57.14%), 4 as serotype A:3 (19.05%), and 5 as not typed (23.8%) (Mohamed et al., 2012). Eid et al. (2019) also reported that the most predominant strains recovered from apparently healthy and infected ducks were *P. multocida* (10.4 and 25.2%, respectively).

Elalamy et al. (2020) surveyed *P. multocida* in apparently healthy and in diseased layer chickens; recovering *P. multocida* in 10 and 4% of birds, respectively. All isolates belonged to capsular type A, and they were multidrug

resistant. Genotypic characterization for antibiotic resistance genes tetH, aphA-1, and BlaROB1 was found to be 100, 70.83, and 8.3%, respectively (Elalamy et al., 2020).

The control of *P. multocida* could be adopted by applying good hygienic measures. Control of rodents is very important even in cases of vaccination or live vaccines formed from serotypes A1, 3, and 4 emulsified in an oil adjuvant (Glisson et al., 2013). Omaleki et al. (2022) recommend that it should be ensured that a successful vaccine strain with a matching anticipated lipopolysaccharides (**LPS**) structure is employed and ensured that a metagenomics culture-independent approach is used, as well as a genetic typing scheme for LPS (Omaleki et al., 2022).

Riemerella anatipestifer

R. anatipestifer is a gram-negative, nonmotile, rod-shaped, nonsporulating bacterium, belonging to the family flavobacteriaceae. It was first described by Riemer (1904). *R. anatipestifer* can infect all domestic poultry, including ducks, turkeys, chickens, pheasants, and waterfowl. It causes high mortality rates, septicemia, and polyserositis, which causes decreases in weight gain, and increases in the frequency of downgraded carcasses thus causes severe financial losses (Abd El-Hamid et al., 2019).

Chen et al. (2022) confirmed that SspA is a T9SS effector protein that plays a role in *R. anatipestifer* pathogenicity and gelatin, fibrinogen, and bacitracin LL-37 proteolysis. Ducklings are more susceptible than older aged animals. Some predisposing factors enhance infection and outbreaks, such as poor environmental conditions and concomitant diseases (Soman et al., 2014).

In Egypt, in 2014, 120 samples were taken from ducks and ducklings from different Egyptian farms in Giza, Qalyubia, Beni Sueif, and Elbehera governorates. The samples were from the blood, liver, heart, and spleen, and the results showed 20 *R. anatipestifer* isolates (16.7%), 14 (11.7%) from ducks, and 6 (5%) from ducklings (Deif et al., 2015). Samples from 50 ducks (5 ducks per farm) were collected from 4 governorates. Of these, only 20% (10 out of 50) were *R. anatipestifer*, while only 4 out of 10 samples were PCR positive (40%). Subsequent gene sequencing of the *OmpA* gene for the 4 isolates confirmed 100% homology and identity (Abd El-Hamid et al., 2019).

The control and prevention of such infection could be obtained from vaccination with bacterins that form the main serotypes to protect against circulating strains. Also, live vaccines formed against serotypes 1, 2, and 5, and administered via aerosol or in drinking water demonstrated efficient protection (Ruiz and Sandhu, 2013). Gamal et al. (2020) prepared a polyvalent *R. anatipestifer* and AI (H5N1) vaccine, which had higher levels of protection against both of them. Furthermore, this vaccine had greater protection in a booster dose than in the primary dose.

E. coli (Avian Colibacillosis)

The causative agent of avian colibacillosis is *E. coli*, which is present in the normal intestinal flora. It is a gram-negative, non−spore-forming bacilli and spreads within avian populations. Most *E. coli* are motile because *E. coli* has peritrichous flagella (Nolan et al., 2013). Increased severity of pathogenic *E. coli* may be caused by a respiratory viral agent, overcrowding, poor handling of birds, or poor sanitation (Eid et al., 2016). Avian colibacillosis is an infection that is caused by avian pathogenic *E. coli* strains (**APEC**), which affect the poultry industry and cause severe financial losses because of bird growth retardation, elevated morbidity and mortality rates, increased carcass condemnation after slaughtering, and the high monetary costs of medication (Kobayashi et al., 2011).

Avian colibacillosis is a systemic infection that induces different forms such as complicated chronic respiratory disease (**CCRD**), acute septicemia, enteritis, omphalitis, coli granuloma, arthritis, salpingitis, swollen-head syndrome, and oophoritis. Postmortem lesions are fibrinous (air sacculitis, perihepatitis, and pericarditis) and associated with fatal septicemia (Rodriguez-Siek et al., 2005; Mellata, 2013). In Egypt, APEC was the primary etiology of respiratory manifestations in broiler chicken flocks. Hussein et al. (2013) recorded avian colibacillosis within poultry farms in Egypt. The most common clinical signs were coli-septicemia between examined birds. A total of 82 broiler chicken flocks, from which 219 *E. coli* strains were isolated and 12 APEC strains were recorded, had colibacillosis lesions such as air sacculitis. In contrast, swollen head syndrome was recorded among 4 cases from broiler flocks (Hussein et al., 2013).

Abd El-Tawab et al. (2014) observed that the incidences of *E. coli* infections were avian collibacillosis (38%) in infected chicken flocks in different localities in Behna governorate. These incidences were divided into 11, 10.1, 8.7, and 8.2% from Benha, KaferShoker, Toukh, and Shebin El-Kanater. A fatal respiratory disease characterized by the 24.7% infection were isolated from diseased chicken carcasses, and 75.7% from freshly dead chickens. Amer et al. (2018) demonstrated lesions of colibacillosis in diseased and freshly dead broiler chickens in some Egyptian governorates, namely Zagazig, Sharkia, Ismailaia, Sinai, Giza, and Qaliobia, and *E. coli* was present about 61%.

Abd El-Tawab et al. (2016) in Sharkia and Dakalia governorates, found that *E. coli* was isolated from the lung (64%) and trachea (52%), respectively, and the most predominant serogroup was O_{78} (20%). Abd El-Samie (2014) reported that in a bacteriological investigation of 70 chickens in 10 farms in Minea Elkameh in the Sharkia governorate, 34 out of 420 samples were classified as APEC and serotyped as O_{55} (9), O_{78} (13), O_{119} (4), and O_{125} (8) in chickens that showed mild respiratory manifestations and air saculitis at postmortem.

El-Shazly et al. (2017) reported avian colibacillosis in 5 breeds of diseased birds from 20 commercial flocks in 4 locations in Egypt. Fifty strains of *E. coli* were recovered from May to July 2012. Ramadan et al. (2018) showed that the prevalence of pathogenic *E. coli* was 48% (48/

100) of human samples and 31.3% (50/160) of chicken samples. Amer et al. (2018) recorded colibacillosis in 56 samples (35%). Enany et al. (2019) reported *E. coli* with an incidence of 26.76% in the farm environment and 50.44% in diseased broilers in tandem. The most common isolated *E. coli* strains serotypes were O_{78}, O_1:H_7, O_{91}:H_{21}, and O_{126} (Enany et al., 2019).

El-Tahawy et al. (2022) found that cefquinome was a suitable drug for managing colibacillosis in broilers, and its withdrawal duration is advised after therapy. The breast muscles were sore for 3 d, and the liver and kidneys were sore for 7 d. The control and prevention of such a condition could be achieved by applying good biosecurity and selecting a good drug therapy based on antimicrobial sensitivity tests. Abdel El-Mawgoud et al. (2020) stated that the live attenuated *E. coli* vaccine was more powerful in decreasing mortality rate and clinical signs in groups challenged only by one coli serotype, either O_{78} or O_{125}, than groups challenged by both serotypes. Moreover, lectin-containing products can minimize the adverse impacts of *E. coli* infection.

Galal et al. (2021) mentioned that the Poulvac *E. coli* vaccine, which is the O_{78} *E. coli* vaccine after deletion of the AroA gene, in a combination of Linco-Spectin or enrofloxacin antibiotics has the efficacy to prevent *E. coli* infection.

The clinical and pathological forms of *E. coli* infections in poultry are presented in Figure 5.

Swollen Head Syndrome

Swollen head syndrome (**SHS**) is a multifactorial condition caused by bacterial and viral infections and environmental factors (Al-Hasan et al., 2022). SHS is a facial edema disease that affects the upper respiratory tract of broilers and broiler breeders. The causative agent of the disease is a mixed infection of a mainly viral infection such as avian metapneumovirus (**aMPV**) and secondary bacterial infection such as *E. coli* (Shawki et al., 2017).

Environmental conditions affect the severity of the infection, such as overcrowding, ammonia, and poor ventilation (Nakamura et al., 1998). SHS is an infection of the upper respiratory tract, mainly in 4- to 6-wk-old chickens, which leads to depression, not eating, coughing, sneezing, sinusitis, submandibular edema, and swelling of the wattles. Economic losses in the poultry industry due to morbidity may reach 10% and mortalities about 2 to 23% (Nakamura et al., 1998).

The elevation of mortality was up to 25% with secondary infections (Pattison et al., 1989). In Egypt, Moustafa (2005) found that *E. coli* was the most prevalent cause of SHS (50.7%). In addition, due to other bacteria, MG (16%), *Haemophilus paragallinarum* (13.3%), *Pseudomonas* (9%), *Klebsiella* (5.3%), (4%), and *Staphylococcus* (1.3%). Nasif et al. (2019) made the first record of aMPV subtype B in broiler chicken farms in Egypt as the cause of SHS in complications with other bacterial infections: *E. coli, Pseudomonas mirabilis*, and *Pseudomonas aeruginosa*. Ahmed et al. (2021) found that SHS was represented in 20% of chicken farms in Egypt due to bacterial pathogens.

Al-Hasan et al. (2022) found that the main etiological factor causing SHS in poultry could be aMPV. Furthermore, this was the first-time that subtype BaMPV strains were found in broiler farms in Iraq. For control and prevention of the infection, biosecurity and good management practices are needed to prevent secondary bacterial infection and drug of choice after performing sensitivity tests (Silke, 2020).

A. paragallinarum (Infectious Coryza)

Avian infectious coryza (**AIC**), is an upper respiratory disease affecting chickens, and it is caused by *H.*

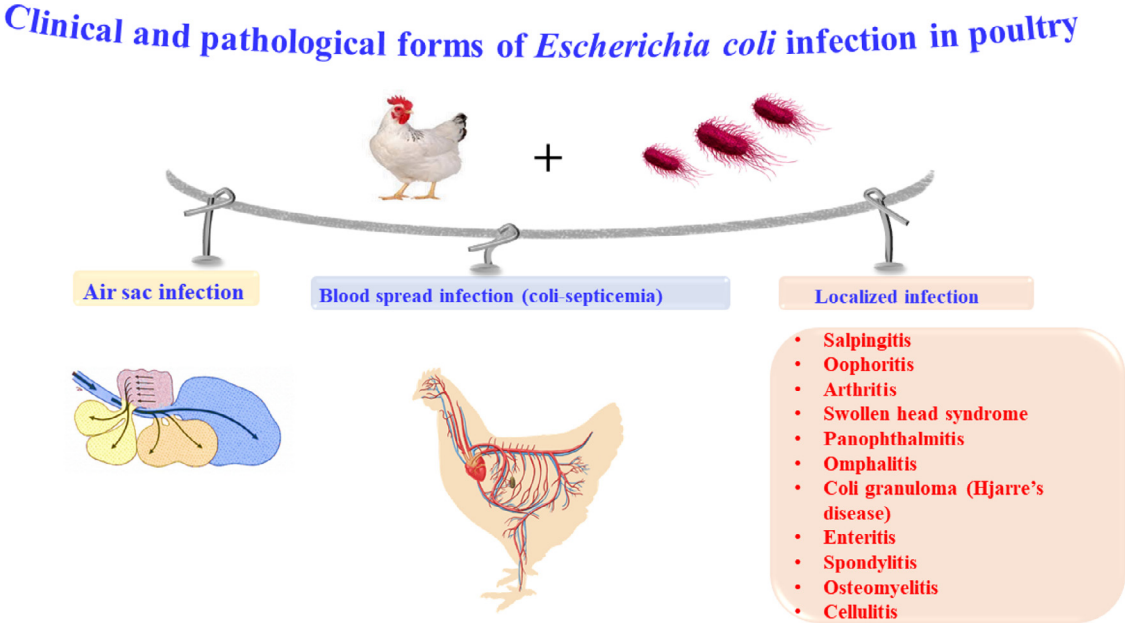


Figure 5. Clinical and pathological forms of *Escherichia coli* infection in poultry including air sac infection, septicemic and localized infections.

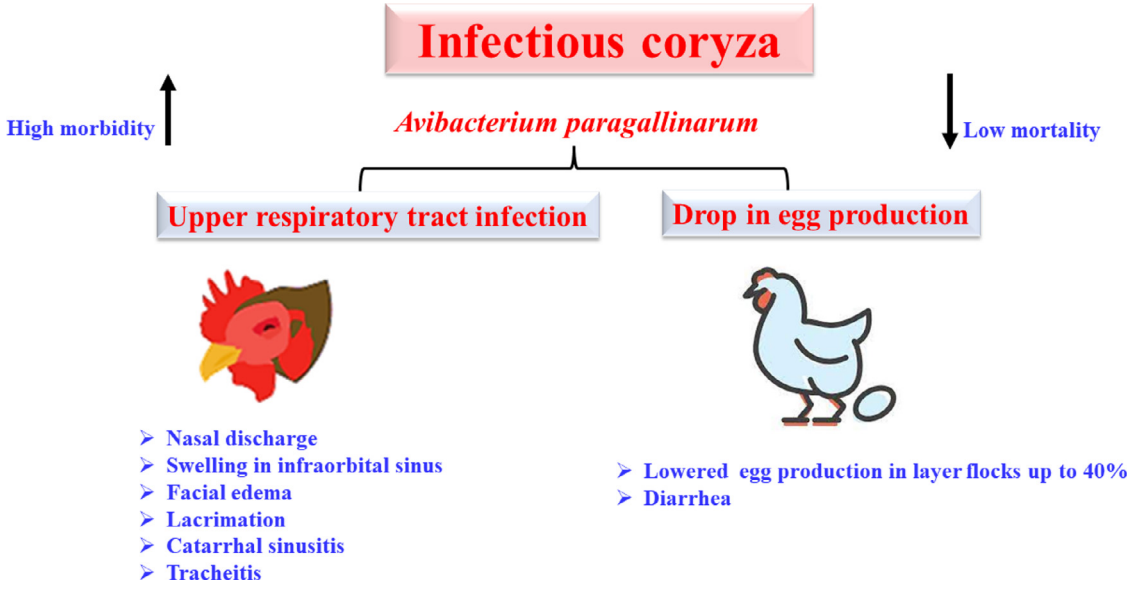


Figure 6. Clinicopathological forms of infectious coryza. This disease is characterized by high morbidity but low mortalities with upper respiratory tract signs including (nasal discharge, swelling in infraorbital sinus, catarrhal sinusitis, facial edema, lacrimation, tracheitis) with an adverse impact on egg production including lowering the egg production up to 40% and in some cases, diarrhea was recorded.

paragallinarum (**HPG**) which is recently called *A. paragallinarum* (Blackall, 1989; Blackall et al., 2005). HPG is gram-negative, capsulated, nonmotile, has coccobacilli or short rods 1 to 3 μm in length and 0.4 to 0.5 μm in width, and is categorized as bipolar bacteria (Garcia et al., 2004). AIC caused a problem in the poultry industry due to chicken outbreaks, lowering egg production in layer flocks by up to 40% (Mouahid et al., 1989). The symptoms of the infection are marked by nasal discharge, fecal edema, lacrimation, diarrhea, and high morbidity between 20 and 50%, with mortality up to 20%. The postmortem findings are catarrhal sinusitis and tracheitis (Byarugaba et al., 2007; Blackall and Soriano, 2013).

In Egypt, Awad Alla et al. (2009) reported infectious coryza in Dakahlia governorate, where 12 isolates of HPG were isolated from 180 samples taken from commercial layers, broilers, breeders, and native breed flocks. Nsengimana et al. (2022) recorded *A. paragallinarum* and *Klebsiella pneumoniae* coinfections in a gray crowned crane. These isolates were differentiated according to morphological, biochemical, and serological features into 8 isolates classified into serotype A and 4 isolates into serotype C. Awad Alla et al. (2009) found HPG in chickens at 18- to 180-days old suffering from sneezing, nasal discharge, lacrimation, facial swelling, and anorexia. These birds were from various chicken farms in Dakahlia governorate. The prevalence of HPG in broiler breeders and native breed chickens was 4.23%, while it was 11.3% in layer chickens. Serotype A was the predominant strain and serotype C was isolated.

Fedawy et al. (2016) collected 120 infraorbital sinus swabs from chickens during 2013−2015 and registered *A. paragallinarum* (with 3 serovars: A, B, and C) as the cause of infectious coryza in layer flocks in Egypt. Guo et al. (2022) tested the sensitivity of *A. paragallinarum* against certain antibiotics and found that they were sensitive to oxytetracycline hydrochloride, amoxicillin, ampicillin, and ceftiofur. In terms of prevention and control, sound management practices and vaccinations can help prevent infections. Still, treatment with certain antibiotics should be conducted after applying sensitivity tests of *A. paragallinarum* toward antibiotics (Tabbu, 2000).

The clinical and pathological forms of infectious coryza infections in poultry are presented in Figure 6.

CONCLUSIONS

The performance of poultry is heavily influenced by respiratory infections. The microbial composition of avian species has provided information that will help us better understand the poultry respiratory microbiome. This review summarized the negative effects of secondary viral and bacterial infections on the respiratory tract of poultry, as well as the synergy among pathogens. It has also been observed that respiratory tract-related bacterial and viral infections can account for a staggeringly large percentage of recorded cases in different nations under field conditions. This percentage is substantially greater in developing nations, where biosafety precautions are less prevalent, resulting in significant monetary losses in the poultry sector. This could also make syndromic surveillance more difficult. During the inspection of an infected poultry flock, it is recommended to diagnose other pathogens that may vary depending on coinfections or preinfection history. The implementation of field vaccination would be more challenging as a result. Uncontrolled vaccination, such as poor administration and distribution of a vaccine and the use of subpar vaccines, increases the risk of subsequent outbreaks. As a result, periodic molecular monitoring of circulating field respiratory pathogens, strict

hygienic measures, proper management, balanced food rations, biosecurity measures, and proper vaccination programs are critical to overcome respiratory tract infections in various poultry farms.

DISCLOSURES

Authors declare no conflict of interests.

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