Review Article

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Global perspectives on infectious coryza: Pathology, immunity, and economic impact in chickens

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ABSTRACT

Coryza is an acute respiratory infection of chickens (*Gallus gallus domesticus*) caused by the Gram-negative bacterium *Avibacterium paragallinarum* of the *Pasteurellaceae* family. This disease is highly contagious and has spread widely throughout the world, with relatively high incidence rates in Southeast Asia, South America, and Africa. Common clinical manifestations include infraorbital sinus swelling, nasal exudate, conjunctivitis, and decreased production performance, both in broiler chicken growth and egg production in laying hens. Infection can be acute, subclinical, or chronic, depending on the virulence of the bacterial strain, the host's immune system, and environmental factors. Pathologically, the disease begins with bacterial colonization of the upper respiratory tract epithelium, followed by activation of a local inflammatory response that leads to neutrophil and lymphocyte infiltration, mucosal edema, and increased mucus secretion. The immunity developed is serotype-specific, so the use of vaccines appropriate for the serotypes circulating in a region is the primary preventative measure. Transmission occurs through direct contact between individuals, aerosols, or through contaminated objects (fomites), with the rate of spread influenced by population density, cage sanitation, and the movement of chickens between farms. Other predisposing factors include stress, the presence of secondary infections, and suboptimal environmental management. Diagnosis is established through a combination of clinical observation, bacterial isolation and culture, serological testing, and molecular techniques such as PCR to differentiate it from other respiratory diseases with similar symptoms. Effective control requires a comprehensive approach, including serotype-based vaccination, strict biosecurity practices, good cage management, and efforts to minimize stress on chickens. Economically, coryza causes significant losses due to reduced productivity and increased costs of treatment and prevention. This review aimed to provide the latest updates on epidemiology, pathogenic mechanisms, clinical presentation, diagnostic methods, therapy, and control strategies for coryza, while also identifying challenges and prospects for further research.

Introduction

Coryza is an acute respiratory disease of chickens (*Gallus gallus domesticus*) caused by the Gram-negative bacterium *Avibacterium paragallinarum* of the *Pasteurellaceae* family (Guo *et al*., 2022a). Clinical cases were first reported in Europe in the early 20th century, and since then, the disease has become a significant problem in the global poultry industry (El-Gazzar *et al*., 2025). Clinically, coryza is characterized by facial swelling, conjunctivitis, nasal discharge, and decreased production performance (Mei *et al*., 2020). Its impact is seen in stunted growth in broiler chickens and reduced egg production in laying hens (Blackall, 1999). The combination of adverse clinical symptoms and its highly contagious nature makes coryza a serious threat to the productivity and sustainability of poultry farming (Han *et al*., 2016).

Epidemiologically, coryza is endemic in various regions, including Southeast Asia, South America, Africa, and several European countries (Blackall, 1999). High population density, inadequate ventilation systems, poor barn sanitation, and the movement of chickens between farms are factors that accelerate disease transmission (Kovács *et al*., 2025). Infection can manifest in acute, subclinical, or chronic forms, influenced by the virulence of the *A. paragallinarum* isolate, the host's immune system, and environmental factors (Guo *et al*., 2022b). The presence of chickens with subclinical or persistent infections further complicates control efforts, as these individuals remain a source of transmission that remains undetected clinically (Wang *et al*., 2026).

A. paragallinarum has a wide variety of serotypes. Its classification is based on the Page system, which divides the species into serotypes A, B, and C, and the Kume system, which provides a more detailed classification (Cao *et al*., 2024). These serotype differences significantly impact the effectiveness of immunization programs, as the immunity developed is generally specific to a particular serotype and does not necessarily provide protection against different (heterologous) isolates (Liu *et al*., 2025a). The development of molecular techniques, such as polymerase chain reaction (PCR) and gene sequencing analysis, has identified genetic variation between isolates (Shelkamy *et al*., 2025a). This approach not only improves diagnostic accuracy, including in subclinical cases, but also supports more comprehensive mapping of the disease's epidemiology.

The pathogenesis of coryza begins with bacterial attachment and colonization of the upper respiratory tract epithelium, particularly in the infraorbital sinus, which then triggers a local inflammatory response (Guo *et al*., 2022b). Stimulation of epithelial cells and macrophages leads to neutrophil and lymphocyte infiltration, accompanied by mucosal edema and increased mucus production. These changes are clinically visible as facial swelling and mucopurulent exudate (Blackall, 1999). The condition can be more severe if secondary infections by opportunistic bacteria such as *Escherichia coli* or *Mycoplasma gallisepticum* occur (Wu *et al*., 2019). These coinfections have the potential to worsen clinical manifestations, prolong recovery, reduce production performance, and increase economic losses in livestock farming (Abd El-Ghany *et al*., 2023).

Coryza diagnosis is carried out through an integrated approach that includes clinical examination, bacterial isolation and culture, serological testing, and molecular detection methods (Han *et al*., 2016). This multi-parameter strategy is necessary to ensure accurate differentiation from other respiratory diseases with similar symptoms, such as fowl chol-

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era, avian influenza, or *M. gallisepticum* infection, each of which requires different management and control policies (Blackall, 1999). Optimal control efforts include vaccination based on circulating serotypes, strict biosecurity implementation, improved environmental management of the cage, and reduction of stress factors to support and maintain chickens' mucosal immunity (Wang *et al*., 2026).

In general, coryza remains a significant problem in the global poultry industry despite being known for over a century. Its impact extends beyond clinical symptoms to substantial economic losses, necessitating a comprehensive approach that integrates an understanding of the pathogen, host immune response, risk factors, and effective diagnostic methods and control strategies. This review aimed to summarize the latest developments related to coryza, including epidemiology, pathogenesis mechanisms, clinical presentations, diagnostic procedures, and control efforts. Furthermore, the article identifies ongoing challenges and potential future research opportunities.

History

Coryza was first clinically reported in chickens in Europe in the early 19th century. In 1920, Theobald Smith and Frederick L. Kilborne described a rhinitis accompanied by facial swelling in laying hens in England, which later became known as "snuffles" or infectious coryza (Deresse *et al*., 2022). These early reports highlighted the highly contagious nature of the disease and its high incidence in intensive livestock systems of the time.

The successful isolation of the etiologic agent of this disease occurred several decades after its initial report. In 1932, Ernst Neter identified a Gram-negative bacterium associated with lesions in the infraorbital sinus of chickens, although at that time, biotype and serotype characterization were still incomplete (Akter *et al*., 2014). Subsequently, in 1943, L. A. Page developed a classification system that divided isolates into serotypes A, B, and C based on serologic responses, which remains the cornerstone of epidemiological studies of this disease (Cui *et al*., 2025).

Developments in molecular biology in the 1990s marked significant advances in the understanding of coryza. PCR techniques and genetic analysis began to be used to study the genetic diversity of *A. paragallinarum*, enabling the identification of subclinical isolates and the determination of serotypes with greater accuracy (Deresse *et al*., 2025). These findings also provided the basis for the development of vaccines tailored to specific serotypes and the formulation of more targeted control strategies based on epidemiological approaches (Muhammad and Sreedevi, 2015).

Entering the early 21st century, coryza remains a challenge for the poultry industry in many countries, despite the expansion of biosecurity measures and vaccination programs (Wang *et al*., 2026). Reports from Southeast Asia from 2010–2015 indicate persistently high levels of endemicity, particularly on small- to medium-scale farms (El-Gazzar *et al*., 2025). This confirms that the disease continues to have a significant economic impact, despite being scientifically known and studied for nearly a century.

Etiology

Poultry coryza is caused by *A. paragallinarum*, a Gram-negative bacterium belonging to the *Pasteurellaceae* family (Li *et al*., 2024). This microorganism is a specific pathogen in chickens and has the ability to colonize the upper respiratory tract, particularly the infraorbital sinus (Xie *et al*., 2023). Morphologically, *A. paragallinarum* isolates are generally pleomorphic coccobacilli and facultative aerobes (Wahyuni *et al*., 2018). For optimal growth in certain culture media, this bacterium usually requires factor X (hemin), although some strains have been reported to be able to grow independently of this factor (Srednik *et al*., 2025).

Based on biochemical and serological tests, *A. paragallinarum* is classified into several commonly recognized serotypes using the Page system (A, B, and C) and the Kume system, which provides a more detailed classification (Mei *et al*., 2023). These serotype variations play a crucial role in epidemiology and the success of vaccination programs, as the immune response is generally specific to a particular serotype (Guo *et al*., 2025a). Furthermore, recent molecular studies have revealed genetic diversity among isolates, potentially influencing the virulence level and the bacterial spread capacity within chicken populations (Deresse *et al*., 2025).

In addition to primary infection by *A. paragallinarum*, chickens with coryza often experience coinfection with opportunistic bacteria such as *E. coli*, *Pasteurella multocida*, or *M. gallisepticum* (Stępień-Pyśniak *et al*., 2024). The presence of these secondary pathogens can exacerbate clinical manifestations, increase morbidity, and complicate accurate diagnosis (Blackall, 1999). The interaction between the primary causative agent and these secondary bacteria underscores the need for a comprehensive control strategy, including appropriate vaccination, improved cage management, and strict biosecurity practices (Leus *et al*., 2026).

Coryza has the ability to spread rapidly between chickens, primarily through direct contact and aerosols from respiratory secretions (Wang *et al*., 2026). Environmental conditions such as high population density, suboptimal ventilation, and stress from transportation or feed changes contribute to the risk of transmission (Deshmukh *et al*., 2015). A comprehensive understanding of the etiology of *A. paragallinarum* and the predisposing factors for infection is essential for designing more targeted and sustainable prevention strategies. Figure 1 illustrates the etiology and main clinical signs of infectious coryza in poultry, highlighting *A. paragallinarum* as the primary causative agent that colonizes the upper respiratory tract and infraorbital sinus. Typical manifestations include facial swelling, nasal discharge, conjunctivitis, and decreased egg production.

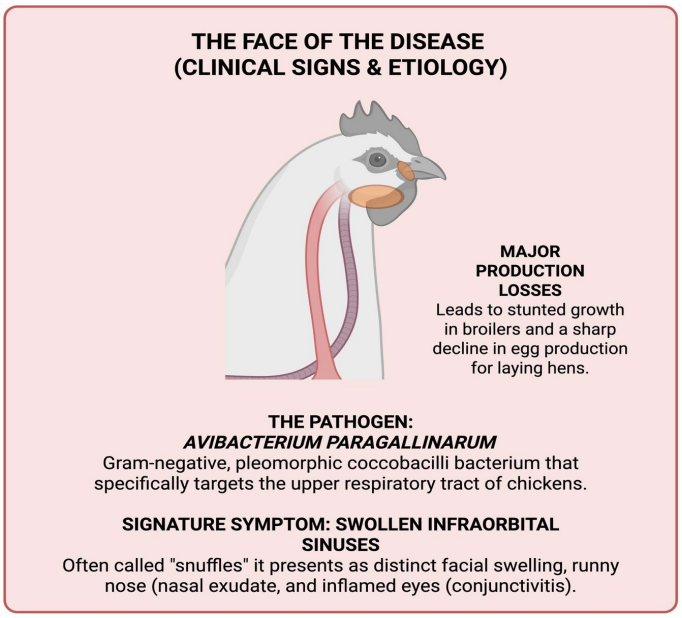

Figure 1. Clinical signs and etiology.

Epidemiology

Infectious coryza is a respiratory disease of chickens that has a widespread global distribution, particularly in areas with intensive poultry production systems (Wang *et al*., 2026). Cases have been reported in various regions, including Asia, Africa, South America, and parts of Europe, with significant economic impacts due to decreased egg production, stunted chicken growth, and increased treatment and control costs (Blackall, 1999). Disease prevalence rates vary across countries, influenced by management practices, poultry population density, and the effectiveness of vaccination programs against *A. paragallinarum*. Table 1 presents an overview of the global distribution and epidemiological patterns of infectious coryza in chickens across the world.

Table 1. Global distribution and epidemiology of infectious coryza in chickens.

Region	Country	Epidemiological pattern	Dominant serotype / Characteristics	Risk factors and contributors	Special notes	References
Southeast Asia	Indonesia, Thailand, and Vietnam	Endemic in small- to medium-scale farms; frequently reported in areas with dense layer or broiler populations	Serotypes A and C are more frequently detected; local isolates with genetic variation identified by molecular studies	Poor ventilation, inadequate sanitation, high stocking density, and suboptimal farm management	Continuous emergence of new field isolates	(Charoenvisal *et al*., 2017; Van *et al*., 2020; Wahyuni *et al*., 2022; Guo *et al*., 2024; Wigle, 2000)
South America	Brazil, Argentina, and Colombia	Seasonal outbreaks mainly affect commercial layer flocks	Serotypes vary; local strains predominate in outbreaks	Movement of chickens, live bird trade, and inter-farm transmission	Highlights the importance of strict biosecurity in disease control	(Conde *et al*., 2011; Sandoval *et al*., 1994; Tangkonda *et al*., 2025; Thomas *et al*., 2005; Guo *et al*., 2025b)
Africa	Nigeria, Ethiopia, and Egypt	Endemic disease in small-scale poultry farms	Limited epidemiological data on circulating serotypes	Limited vaccine availability and restricted access to veterinary services	High infection incidence in rural poultry production systems	(Adenkola *et al*., 2016; Assad *et al*., 2021; Deresse *et al*., 2025; El-Gazzar *et al*., 2025)
Europe	Countries with intensive poultry production	Sporadic cases with relatively low prevalence	Serotypes vary depending on outbreaks	Strict biosecurity measures and routine vaccination programs	Cases still occasionally reported despite control measures	(Heuvelink *et al*., 2018; Wang *et al*., 2026)

In Southeast Asia, including Indonesia, Thailand, and Vietnam, coryza cases are frequently found in small- to medium-scale farms, particularly in areas with dense populations of layer or broiler chickens (Charoenvisal *et al*., 2017; Van *et al*., 2020; Wahyuni *et al*., 2022). Environmental conditions such as inadequate ventilation and poor barn sanitation contribute to increased opportunities for disease transmission (Wigle, 2000). Reports on serotypes in the region indicate that serotypes A and C of *A. paragallinarum* are more frequently detected, although local isolates with new genetic variations continue to be identified through molecular studies (Guo *et al*., 2024).

In South America, coryza cases have been reported in Brazil, Argentina, and Colombia, with a seasonal pattern and a predominance of cases affecting commercial laying hens (Conde *et al*., 2011; Sandoval *et al*., 1994; Tangkonda *et al*., 2025). Epidemiological studies indicate that inter-farm transmission frequently occurs through the movement of chickens and the live bird trade (Thomas *et al*., 2005). These findings underscore the importance of strict biosecurity as a key component in controlling the disease caused by *A. paragallinarum* (Guo *et al*., 2025b).

In Africa, coryza remains a major constraint on small-scale poultry farms in Nigeria, Ethiopia, and Egypt (Adenkola *et al*., 2016; Assad *et al*., 2021; Deresse *et al*., 2025). Limited vaccine availability and access to adequate veterinary services contribute to the high incidence of infections caused by *A. paragallinarum* (El-Gazzar *et al*., 2025). In contrast, in Europe, the incidence of this disease is relatively low due to strict biosecurity practices and routine vaccination (Heuvelink *et al*., 2018). However, sporadic cases are still reported in several countries with intensive chicken production systems (Wang *et al*., 2026).

Pathogenesis

The pathogenesis of coryza in chickens begins with infection by *A. paragallinarum*, which has a tropism for the upper respiratory tract, particularly the infraorbital sinus, conjunctiva, and nasal mucosa (Ali *et al*., 2013). The bacteria enter the body through aerosol inhalation or direct contact with contaminated secretions, then initially adhere to the respiratory epithelium, a crucial step in the infection process (Soliman *et al*., 2023). This adhesion process is mediated by bacterial surface components, such as fimbriae and adhesins, which play a role in facilitating colonization and helping the bacteria evade mucociliary clearance mechanisms in the respiratory tract (Liu *et al*., 2025a).

After binding to the epithelial surface, *A. paragallinarum* penetrates the mucosal layer and triggers a local inflammatory response (Balouria *et al*., 2019). Stimulation of epithelial cells and macrophages leads to the release of proinflammatory cytokines, which then recruit neutrophils and lymphocytes to the infected tissue area (Faisal *et al*., 2026). This inflammatory process causes mucosal edema, increased mucus secretion, and hyperemia. Clinically, these changes manifest as facial swelling, conjunctivitis, and nasal discharge (Xu *et al*., 2019).

Coryza infection is generally acute, but under certain conditions it can develop into subclinical or chronic forms, depending on the virulence of the *A. paragallinarum* isolate, the immune status of the chickens, and the presence of secondary infections (Yehia *et al*., 2023). Coinfection with opportunistic bacteria such as *E. coli* or *M. gallisepticum* often exacerbates tissue damage, triggers more extensive complications, and prolongs the recovery period (Paudel *et al*., 2017a). In general, the disease can last from a few days to several weeks. Although the mortality rate is relatively low, the impact on productivity remains significant and causes significant economic losses (Mei *et al*., 2020).

In addition to causing local lesions in the respiratory tract, *A. paragallinarum* can also affect the systemic immune response (El-Gazzar *et al*., 2025). Antibody formation is generally specific to a particular serotype, so the resulting immunity does not provide optimal cross-protection against other serotypes or re-exposure (Sakamoto *et al*., 2012). This explains why outbreaks can recur in the same population, particularly on farms with inadequate biosecurity practices and vaccination programs that are not tailored to circulating serotypes.

Immune response

Infection by *A. paragallinarum* induces a complex host immune response, involving both the innate and adaptive immune systems (Diptesh *et al*., 2020). The initial phase of defense is mediated by respiratory epithelial cells and macrophages, which detect bacterial components through pattern recognition receptors (PRRs) (Guo *et al*., 2022b). Activation of these PRRs triggers the production and release of proinflammatory cytokines, including interleukin-1 (IL-1), IL-6, and tumor necrosis factor-alpha (TNF-α) (Mogensen, 2009). These mediators play a role in recruiting immune cells to infected mucosal tissues, thereby strengthening the local defense response against bacterial colonization (Carty *et al*., 2021).

In addition to innate immune mechanisms, the adaptive immune response also plays a crucial role in controlling infections caused by *A. paragallinarum* (Chen *et al*., 2024). B lymphocytes produce specific antibodies against bacterial surface antigens, primarily immunoglobulin A (IgA) in mucosal secretions and immunoglobulin G (IgG) in the systemic circulation (Zhang *et al*., 2025). Mucosal IgA functions to inhibit bacterial

adhesion to the epithelium and neutralize it, while IgG contributes to the opsonization process and increases the effectiveness of phagocytosis by macrophages and neutrophils (Gao *et al*., 2024). On the other hand, T cell activation also plays a role in coordinating the immune response (van den Biggelaar *et al*., 2020). Helper T cells (CD4+) support antibody production and regulate the inflammatory response, while cytotoxic T cells (CD8+) have the potential to assist in the elimination of infected cells (Swain *et al*., 2012).

Although an immune response is activated after infection or vaccination, protection against *A. paragallinarum* is generally serotype-specific (Saiful *et al*., 2025). The immunity developed tends to be effective only against the same serotype, leaving chickens at risk of reinfection if exposed to a different serotype (Liu *et al*., 2025a). This presents a significant challenge in field coryza control efforts, particularly on farms with multiple serotypes circulating simultaneously (Pérez *et al*., 2025).

Coinfection with opportunistic bacteria can affect the dynamics of the immune response, increase the production of inflammatory mediators, and prolong the disease course (Paudel *et al*., 2017a). In the case of coryza caused by *A. paragallinarum*, this condition can exacerbate clinical outcomes and delay recovery (Mei *et al*., 2020). Therefore, optimal control relies not only on vaccination programs but also requires the implementation of good livestock health management, strict biosecurity, and efforts to minimize stress to optimize the host's immune response capacity.

Pathology

Infection by *A. paragallinarum* in chickens causes characteristic pathological changes in the upper respiratory tract and surrounding tissues (Diptesh *et al*., 2020). Macroscopically, common lesions include edema of the infraorbital sinus, conjunctival hyperemia, and mucopurulent exudate in the nostrils (Helliwell, 2010). Facial swelling and nasal discharge reflect the accumulation of inflammatory fluid in the submucosal tissue. In chronic cases, the infraorbital sinus can undergo fibrosis and mucosal thickening, potentially leading to prolonged respiratory distress (Wahyuni *et al*., 2018). Table 2 summarizes the pathological changes found in chickens infected with *A. paragallinarum*.

Histopathologically, coryza infection shows a fairly massive infiltration of inflammatory cells, primarily neutrophils, lymphocytes, and macrophages, in the epithelium and submucosal tissue of the sinuses and nasal mucosa (Blackall, 1999). The respiratory epithelium often shows changes ranging from degeneration to cell necrosis, while the mucosal glands undergo hyperplasia in response to inflammatory stimuli and invasion by the bacterium *A. paragallinarum* (Akter *et al*., 2014). This inflammatory reaction is generally accompanied by increased mucus production, the formation of mucopurulent exudate, and thickening of blood vessel walls due to vasodilation and increased vascular permeability (Paudel *et al*., 2017a).

Systemic lesions are relatively rare, as *A. paragallinarum* typically acts as a local pathogen in the upper respiratory tract (Xie *et al*., 2023). However, coinfection with opportunistic bacteria such as *E. coli* or *Mycoplasma* spp. can extend damage to the lungs and other organs, increasing morbidity and reducing production performance (Crispo *et al*., 2019). These pathological changes underlie the typical clinical signs, such as sneezing, conjunctivitis, and decreased productivity. Despite this, mortality rates generally remain low in chicken populations with good underlying health (Xu *et al*., 2019).

Understanding the pathological changes in coryza is essential for establishing a laboratory diagnosis and formulating effective control strategies. Characteristic lesions in the infraorbital sinus and the presence of inflammatory cell infiltration can be used as indicators of infection by *A. paragallinarum* (Paudel *et al*., 2017b). Furthermore, histopathological evaluation plays a role in assessing the response to therapy and the effectiveness of vaccination programs (Guo *et al*., 2025b). Combining pathological findings with clinical data and immunological parameters will improve diagnostic accuracy and facilitate more informed decision-making in disease management in the field.

Clinical manifestations

Coryza in chickens exhibits typical respiratory manifestations resulting from the invasion of the upper respiratory tract by *A. paragallinarum* (Han *et al*., 2016). Initial symptoms generally appear 1–3 days after exposure, characterized by sneezing, nasal discharge, and conjunctivitis accompanied by eyelid edema (Blackall, 1999). In the acute phase, facial swelling—particularly in the infraorbital sinus area—is often evident and serves as a strong clinical clue in establishing the initial diagnosis (Guo *et al*., 2022b).

In addition to local manifestations in the respiratory tract, chickens infected with *A. paragallinarum* may also exhibit decreased appetite, lethargy, and reduced egg production and weight gain (Liu *et al*., 2025a). In the acute phase, the formation of a thick, mucopurulent exudate can block the nasal passages, triggering respiratory distress and increasing discomfort in the chickens (Welchman *et al*., 2010). Meanwhile, subclinical or chronic forms generally occur in populations with partial immunity or due to exposure to a different serotype (Marit, 2024). In these conditions, clinical signs tend to be mild or even invisible, but infected individuals still have the potential to become a source of infection within a flock (Han *et al*., 2016).

Coinfection with opportunistic bacteria or other respiratory viruses can exacerbate the clinical picture, triggering mild to moderate fever, exacerbating the decline in egg production, and prolonging the recovery period (Paudel *et al*., 2017a). This situation explains why coryza continues to have a significant economic impact despite its relatively low mortality rate (Damasio *et al*., 2015). Variations in symptom severity are also influ-

Table 2. Pathological lesions in chickens infected with A. paragallinarum.

Lesion type	Location / tissue	Macroscopic / microscopic features	Clinical / physiological impact	Additional notes
Macroscopic lesion	Infraorbital sinus, conjunctiva, and nostrils	Edema of the infraorbital sinus, conjunctival hyperemia, mucopurulent exudate in the nostrils, facial swelling, and nasal discharge	Produces typical clinical signs such as sneezing, conjunctivitis, nasal discharge, and respiratory distress	In chronic cases, fibrosis and mucosal thickening of the infraorbital sinus may occur, leading to prolonged respiratory disturbance
Microscopic lesion	Sinus epithelium, nasal mucosa, and submucosal tissue including mucosal glands	Massive infiltration of inflammatory cells (neutrophils, lymphocytes, macrophages); epithelial degeneration and necrosis; mucosal gland hyperplasia; excessive mucus production; mucopurulent exudate; thickening of blood vessel walls due to vasodilation and increased vascular permeability	Reflects a strong local inflammatory response that disrupts normal respiratory function and mucociliary clearance	Histopathological examination of sinus and nasal tissues is important for confirming infection and assessing tissue damage
Systemic lesion (rare)	Lungs and occasionally other organs during secondary infection	Lesions associated with secondary bacterial infection, particularly by *E. coli* or *Mycoplasma* spp., which may extend inflammation to the lower respiratory tract	Increased morbidity, respiratory complications, and decreased production performance	*A. paragallinarum* primarily acts as a localized pathogen; mortality generally remains low in otherwise healthy flocks

enced by host factors, such as age, immune status, and environmental stress, as well as the virulence of the infecting *A. paragallinarum* isolate (Byarugaba *et al*., 2007).

Diagnosis

Diagnosing coryza in chickens requires a comprehensive approach that includes clinical examination, histopathological evaluation, and laboratory testing to confirm the presence of *A. paragallinarum* (Liebhart *et al*., 2023). Clinically, the most common signs include facial swelling, mucopurulent exudate in the infraorbital sinus, conjunctivitis, and nasal discharge (Pérez *et al*., 2025). While these presentations are quite typical, similar manifestations can also be seen in other respiratory diseases, making laboratory confirmation crucial for a definitive diagnosis (Calderaro *et al*., 2022).

Conventional laboratory approaches involve isolating and identifying bacteria from nasal or infraorbital sinus secretions using selective media enriched with factor X (hemin) (Wahyuni *et al*., 2018). Through this culture, *A. paragallinarum* can be characterized based on morphology, biochemical properties, and serotyping (Feberwee *et al*., 2019). Serological methods such as the hemagglutination-inhibition (HI) test and enzyme-linked immunosorbent assay (ELISA) are used to detect specific antibodies against certain serotypes (Sun *et al*., 2007). These tests provide insight into the immune status of the population and the distribution patterns of circulating serotypes in the field.

Molecular-based approaches, particularly polymerase chain reaction (PCR), have significantly improved the sensitivity and specificity of *A. paragallinarum* detection (Anjaneya *et al*., 2014). This method allows for rapid bacterial identification, including in chickens with subclinical infections and in populations with high maternal antibody levels (Krylova *et al*., 2024). Furthermore, several real-time PCR developments are capable of serotype differentiation, providing crucial support for developing more targeted and effective vaccination strategies (Patil *et al*., 2017).

In addition to laboratory examinations, combining clinical information, pathological findings, and immune response parameters can improve diagnostic accuracy (Calderaro *et al*., 2022). Histopathological findings in the infraorbital sinus, including neutrophil and lymphocyte infiltration and respiratory epithelial damage, can provide supporting evidence in confirming infection with *A. paragallinarum* (Paudel *et al*., 2017b). This integrated approach is crucial for differentiating coryza from other respiratory diseases such as avian influenza, *M. gallisepticum* infection, and P. multocida infection, allowing appropriate and effective control and therapeutic measures to be implemented.

Differential diagnosis

Clinical signs of coryza, including swelling of the infraorbital sinuses, nasal discharge, conjunctivitis, and mucopurulent exudate, can mimic the symptoms of various other respiratory diseases in chickens (Wahyuni *et al*., 2019). Therefore, differential diagnosis is crucial to distinguish coryza from infections caused by other respiratory pathogens that can have similar effects on poultry health and productivity (Glisson, 1998).

One disease that should be considered in the differential diagnosis is infection by *M. gallisepticum*, which can also cause chronic conjunctivitis and rhinitis (Xu *et al*., 2025). The difference is that *M. gallisepticum* infections usually develop chronically with a low mortality rate, but are often accompanied by slow growth and decreased egg production (Bottinelli *et al*., 2022). Furthermore, this infection does not cause the acute swelling of the infraorbital sinuses seen in coryza (Blackall, 1999).

Avian influenza and Newcastle disease can also cause nasal discharge and conjunctivitis, but are usually accompanied by more severe systemic symptoms, such as high fever, lethargy, and a higher mortality rate (Dey

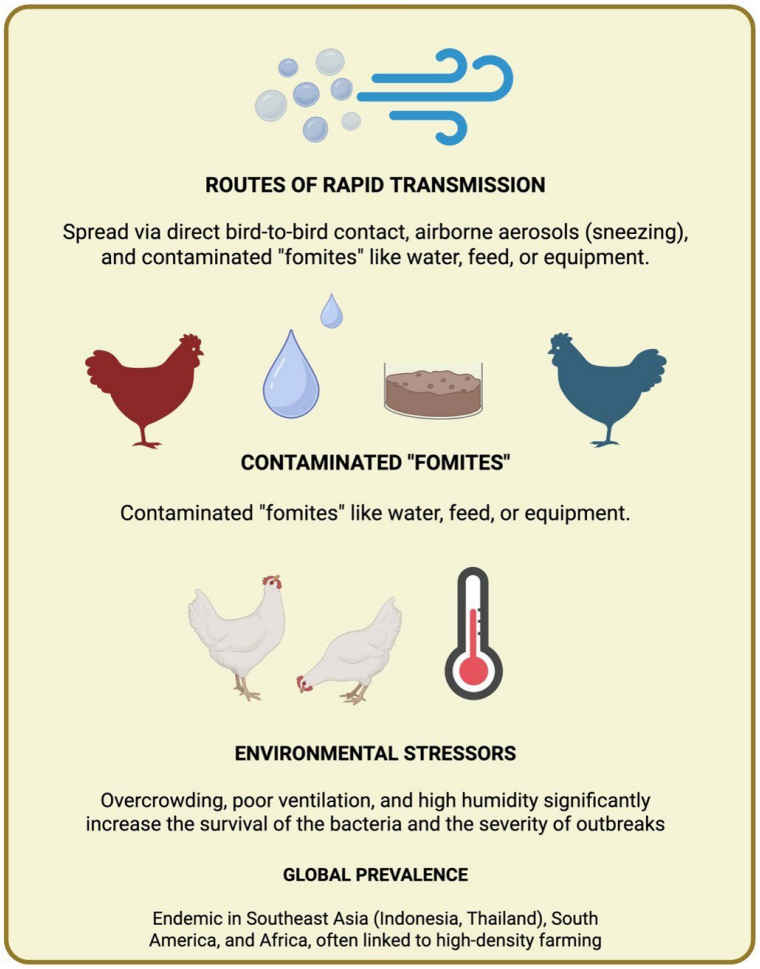

Figure 2. Transmission and risk hotspots.

Table 3. Treatment strategies for coryza in chickens.

Treatment type	Example / Drug / Intervention	Mechanism and effect	Clinical notes / Implementation
Antibiotic therapy	Sulfonamides, tetracyclines, and fluoroquinolones	Inhibit or kill *Avibacterium paragallinarum* to reduce bacterial load and clinical symptoms	Drug selection should be based on antimicrobial sensitivity testing to minimize resistance; mainly applied in acute clinical cases
Supportive therapy	Electrolytes, vitamins, and nutritional supplements	Maintain physiological balance, support immune response, and accelerate recovery	Particularly important during the recovery phase or in weakened and stressed chickens
Environmental management	Isolation of infected chickens, improved coop sanitation, and stress control	Reduces horizontal transmission and improves treatment effectiveness	Essential in both clinical and subclinical infections; apparently healthy birds may act as infection sources
Integrated control measures	Serotype-specific vaccination, enhanced biosecurity, and flock monitoring	Reduce bacterial reservoirs and prevent reinfection within the flock	Treatment alone cannot eradicate the disease; integrated prevention is required for long-term control
Alternative approaches	Immunomodulators and phytobiotics	Enhance respiratory mucosal immunity and reduce bacterial colonization	Currently under investigation; aims to reduce antibiotic dependency and support sustainable disease control

et al., 2023; Regmi *et al*., 2024). Damage to internal tissues, including the lungs and other organs, is more pronounced in these diseases than in coryza, which tends to be limited to the upper respiratory tract (Liu *et al*., 2025b).

Infection with P. multocida, the causative agent of fowl cholera, can also cause facial swelling and mucopurulent exudate (de Cecco *et al*., 2021). However, fowl cholera usually develops acutely and systemically with high mortality and causes multiple lesions in the liver, spleen, and other internal organs, unlike the localized lesions typical of coryza (Christensen and Bisgaard, 2000).

Laboratory examination plays a crucial role in the differential diagnosis process. Through isolation of specific bacteria, serological tests, and PCR, the causative agent can be identified with high accuracy, while histopathological analysis of the infraorbital sinus and respiratory epithelium provides additional information to differentiate coryza from other respiratory infections (Miller *et al*., 2018). This integrated approach ensures that control and therapeutic measures are tailored to the pathogen involved.

Transmission

Chicken coryza is caused by *A. paragallinarum* and is generally transmitted through direct contact between individuals and exposure to respiratory secretions (Diptesh *et al*., 2020). Primary transmission routes include aerosols from coughing or sneezing, nasal secretions, and contamination of equipment, feed, or drinking water by infected chickens (Yehia *et al*., 2023). Subclinically infected or persistently infected chickens can serve as important reservoirs, continuously spreading the bacteria to susceptible populations (Mei *et al*., 2020).

In addition to horizontal transmission between chickens, environmental factors also play a significant role in disease spread (Cao *et al*., 2024). High population densities in chicken pens, inadequate ventilation, and poor sanitation increase the risk of transmission by facilitating contact between chickens and allowing bacteria to persist longer in the environment (Fauziah *et al*., 2021). Furthermore, the movement of chickens, the trade in live poultry, and the movement of farm equipment and tools contribute to the spread of disease between locations (Xu *et al*., 2019).

Although vertical transmission through eggs has not been shown to play a significant role, the presence of secondary infections by opportunistic bacteria or respiratory viruses may increase the susceptibility of chickens to infection (Yue *et al*., 2026). Stress factors, such as changes in feed, vaccination, or transportation, have the potential to reduce host resistance, facilitating bacterial colonization and shedding to other individuals in the population (Chen *et al*., 2026). Figure 2 illustrates the transmission routes and major risk points for coryza in poultry, emphasizing horizontal spread through direct contact, respiratory secretions, aerosols, and contaminated equipment, feed, or water.

From an epidemiological perspective, coryza spreads quickly and effectively, especially in high-density layers or broiler chicken populations (Guo *et al*., 2025b). Understanding this transmission mechanism is an important basis for designing control strategies, such as isolating sick chickens, improving biosecurity measures, and establishing infection-free zones within the farm (Fauziah *et al*., 2021). Thus, controlling this disease does not only depend on vaccination, but also requires good environmental management and careful monitoring of chicken populations.

Risk factors

Chicken susceptibility to coryza is influenced by a complex interaction between the pathogen, the host, and environmental factors (Lema and Tuli, 2023). One major risk factor is population density; chickens kept in crowded cages increase the likelihood of direct contact between chickens and the spread of aerosols of respiratory secretions containing *A. paragallinarum* (Xu *et al*., 2019). This high level of rearing intensity is directly correlated with the frequency of outbreaks, particularly in commercial layer and broiler farms (Shelkamy *et al*., 2025b).

Environmental factors also play a significant role in increasing the risk of infection (Zhu *et al*., 2022). Inadequate ventilation, high humidity, and poor sanitation can prolong bacterial survival in the environment, facilitate horizontal transmission, and weaken the mucosal defenses of the respiratory tract (Liu *et al*., 2025b). Furthermore, exposure to stress—such as changes in diet, transportation, vaccination, or management disruptions—can decrease the host's immune response, increasing susceptibility to both primary and secondary infections (Balouria *et al*., 2019).

The immune status of both individuals and populations is a key biological factor influencing infection risk (Mei *et al*., 2020). Young chickens that have not yet acquired maternal antibodies, or those from non-immune mothers, are more susceptible to acute infection (Guo *et al*., 2022a). Furthermore, the presence of *A. paragallinarum* serotype variations in the field increases the likelihood of reinfection, as developed immunity is specific to a particular serotype and does not provide protection against different isolates (Wahyuni *et al*., 2018).

Coinfection with opportunistic bacteria, such as *E. coli* or *M. gallisepticum*, can exacerbate clinical symptoms and prolong recovery, making infected chickens a more effective source of infection (Paudel *et al*., 2017a). Furthermore, management factors, including animal rotation, feed and water management, and movement of chickens between farms, also play a significant role in the epidemiology of coryza (Bhebhe *et al*., 2025).

By understanding these risk factors, more effective control strategies can be designed. An integrated approach includes regulating stocking density, enhancing biosecurity measures, managing stress and nutrition, and implementing vaccinations tailored to local serotypes (Van Steenwinkel *et al*., 2011). This combination of efforts aims to minimize coryza transmission while maintaining poultry productivity.

Economic impact

Coryza has a significant economic impact on the poultry industry due

to reduced productivity and increased disease control costs. Infection, whether acute or chronic, can inhibit the growth of broiler chickens, slow feed conversion efficiency, and reduce egg production in layers (Wang *et al*., 2026). This reduced performance directly impacts farmer income, particularly in medium- to large-scale farms, where cumulative losses can account for a significant portion of total production (Caballero-Garcia *et al*., 2022).

In addition to impacting productivity, coryza control also imposes additional economic burdens (Hafez and Attia, 2020). The costs of antibiotics, vaccination, pen management, and enhanced biosecurity add to financial pressure, especially in areas with high disease prevalence (Gray *et al*., 2021). It's important to remember that inappropriate or delayed treatment can prolong the duration of an outbreak, increase the risk of reinfection, and increase economic losses (Fauziah *et al*., 2021).

Subclinical infections also have an often overlooked economic impact. Infected chickens that do not show obvious symptoms still act as a source of infection, causing long-term productivity losses and complicating control efforts (Xie *et al*., 2023). Furthermore, the trade in live chickens and the transfer of equipment between farms can facilitate the spread of disease between regions, increasing the risk of losses at the regional and national levels (Rimi *et al*., 2017).

The economic impact of coryza extends beyond direct financial losses. Decreased egg quality, slowed chicken weight gain, and the need for additional management to prevent disease spread contribute to reduced operational efficiency (Blackall, 1999). A comprehensive evaluation of these economic impacts provides an essential basis for designing integrated control strategies, including the implementation of serotype-appropriate vaccination, improved cage management, and enhanced biosecurity, to minimize losses and maintain optimal poultry productivity (Wang *et al*., 2026).

Treatment

Treatment for coryza aims to alleviate clinical symptoms, accelerate recovery, and prevent further spread of the disease within the chicken population (Medelley, 2022). The primary therapeutic approach involves the use of antibiotics effective against *A. paragallinarum*, with drug selection based on sensitivity testing to prevent resistance (Rajurkar *et al*., 2010). Antibiotics such as sulfonamides, tetracyclines, or fluoroquinolones are typically used selectively in acute cases, while supportive therapy including electrolyte and vitamin supplementation helps maintain the birds' physiological condition during recovery (Tangkonda *et al*., 2025). Table 3 summarizes the treatment strategies and interventions used in infected chickens.

In addition to antibiotic use, environmental management plays a crucial role in treatment effectiveness (El-Naenaeey *et al*., 2021). Isolating infected chickens, improving cage sanitation, and controlling environmental stress can enhance the response to therapy while reducing the risk of horizontal transmission (Wang *et al*., 2026). These efforts are especially crucial in subclinical cases, where chickens appear healthy but still have the potential to spread the infection (Peighambari *et al*., 2022).

It's important to remember that treatment alone isn't sufficient to eliminate the disease long-term (Blackall, 1999). Infection with a different serotype or an antibiotic-insensitive isolate can lead to reinfection (Chukiatsiri *et al*., 2012). Therefore, treatment should be combined with preventative strategies, such as serotype-appropriate vaccination, increased biosecurity, and monitoring the chicken population to reduce the bacterial reservoir on the farm (El-Gazzar *et al*., 2025).

In addition to pharmacological treatments, alternative approaches such as the use of immunomodulators or phytobiotics are being investigated to strengthen the respiratory mucosal defense against bacterial colonization (Fauziah *et al*., 2021). This strategy aims to reduce reliance on antibiotics, lower the risk of resistance, and increase the effectiveness of sustainable disease control.

Control

Controlling coryza in chickens requires an integrated approach encompassing immunization, environmental management, and enhanced biosecurity to limit the spread of *A. paragallinarum* and minimize economic losses (Cui *et al*., 2025). Vaccination is the primary control method, particularly the use of inactivated or live vaccines tailored to local serotypes (Ibrahim *et al*., 2024). The immunity developed is specific to a particular serotype, so serotype mapping in the field is crucial to ensure vaccine effectiveness and prevent reinfection (Han *et al*., 2016).

In addition to vaccination, good poultry management plays a crucial role in limiting disease transmission (Islam and Rahman, 2023). Controlling population density, adequate ventilation, routine sanitation, and hygienic management of feed and drinking water can reduce direct contact between chickens and lower bacterial concentrations in the environment (Dereja, 2017). Isolating sick or subclinical chickens is also an important preventive measure to prevent horizontal spread within the flock (Derksen *et al*., 2018). Figure 3 illustrates an integrated poultry coryza control strategy, which combines serotype-specific vaccination, improved environmental management, strict biosecurity practices, and increased host resistance.

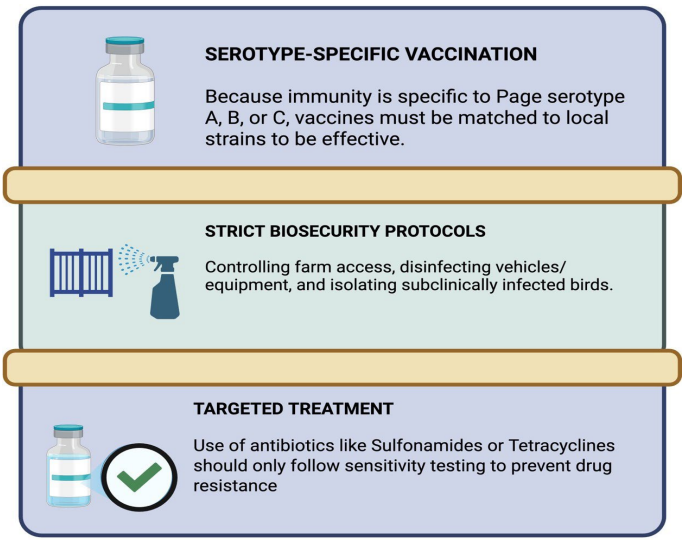

Figure 3. The integrated control strategy.

Biosecurity at the farm level is a crucial aspect of control. Restricting access to pens, disinfecting equipment, clothing, and vehicles, and monitoring the movement of chickens and equipment between farms help prevent the spread of disease across locations (Pierdon *et al*., 2025). Consistent implementation of biosecurity protocols has been shown to be effective in reducing the frequency of outbreaks and limiting the presence of bacterial reservoirs in the production environment (Youssef *et al*., 2021).

Coryza control can also be enhanced through stress management and increased chicken immunity (Diptesh *et al*., 2020). Providing nutritional supplements, immunomodulators, and implementing stress-reducing husbandry practices can enhance the mucosal immune response to bacterial colonization, reduce the severity of clinical symptoms, and accelerate the recovery process (Cui *et al*., 2025).

Conclusion

Coryza is an acute respiratory disease of chickens caused by *A. paragallinarum*, which has a significant impact on poultry health and productivity. The disease is highly contagious, primarily transmitted through direct contact and aerosols of respiratory secretions. It is influenced by factors such as population density, barn sanitation, and stress. The pathogenesis of coryza involves colonization of the upper respiratory tract epi-

thelium and activation of a local inflammatory response. Immunity is specific to certain serotypes, making appropriate vaccination crucial.

Diagnosis of coryza requires a combined approach that includes clinical examination, serology, bacterial culture, and molecular detection methods to differentiate it from other respiratory diseases. Effective control strategies include serotype-specific vaccination, environmental management, strict biosecurity practices, and stress reduction for chickens. Risk factors such as inadequate isolation, movement of chickens between farms, and secondary infections also need to be considered in control planning.

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Conflict of interest

The authors declare that there is no conflict of interest.

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