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# Comparative analysis of atrophic rhinitis and bronchitis: Etiology, clinical features and therapeutic approaches

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**Abstract**
Atrophic Rhinitis (AR) and bronchitis are significant respiratory diseases of swine that contribute to substantial economic losses through impaired growth, reduced feed efficiency, and increased mortality. AR is a chronic, degenerative disorder of the nasal mucosa characterized by turbinate atrophy, mucosal desiccation, and excessive crust formation, frequently accompanied by the malodor known as ozaena. Although multiple organisms may be involved, the pathogenesis typically begins with colonization by *Bordetella bronchiseptica*, which promotes mucosal inflammation and ciliary dysfunction, facilitating secondary infection by toxigenic *Pasteurella multocida*. The dermonecrotoxin produced by *P. multocida* disrupts bone remodeling, resulting in progressive turbinate destruction, facial deformities, and compromised growth. Epidemiological studies show variable prevalence influenced by housing, ventilation, season, and management practices. Diagnosis relies on clinical evaluation, microbial detection, PCR assays, and histopathology. Treatment strategies include antimicrobial therapy, anti-inflammatory support, and optimized husbandry, while prevention emphasizes biosecurity, vaccination, sanitation, and selective breeding.
Bronchitis in swine arises from complex interactions among viral and bacterial pathogens, including PRRSV, swine influenza virus, PRCV, *B. bronchiseptica*, and *Mycoplasma hyopneumoniae*. Coinfections amplify inflammatory responses, leading to more severe pulmonary lesions than single-pathogen infections. Macroscopic lesions commonly present as cranioventral lung consolidation, while microscopic findings range from mild interstitial thickening in single-pathogen infection to severe lymphocytic cuffing, epithelial necrosis, and syncytial cell formation in dual infections. Diagnosis integrates clinical signs with bacterial culture, PCR testing, serology, and histopathology. Management includes appropriate antimicrobial therapy for bacterial agents, environmental control, vaccination, and ongoing herd-level health monitoring. Together, AR and bronchitis exemplify the multifactorial nature of swine respiratory disease and highlight the need for integrated diagnostic, preventive, and therapeutic approaches.

**Keywords:** Atrophic rhinitis, bronchitis, *Bordetella bronchiseptica*, *Pasteurella multocida*, porcine respiratory coronavirus, swine respiratory disease, turbinate atrophy, coinfection dynamics

## Introduction

### Atrophic Rhinitis

Atrophic rhinitis (AR) is a chronic degenerative disorder of the nasal mucosa characterized by progressive architectural loss of the turbinates, excessive crust formation, and the development of a distinctive malodor (ozaena). Although the precise etiological trigger remains incompletely understood, the disease manifests through mucosal desiccation, accumulation of thick secretions, and an enlarged nasal cavity with paradoxical airflow disturbances (Zohar *et al*., 1990) [25]. In swine, the progressive form traditionally referred to as turbinate atrophy is primarily associated with toxigenic Pasteurella multocida (capsular types A or D), which produces clinical signs such as facial distortion, sneezing, nasal exudation, and growth impairment. AR has historically been described under several synonymous terms, including *rhinitis atrophicans*, *coryza foetida*, *acute necrotizing rhinitis*, and *rhinitis chronica foetida* (Loch & Reiriz, 1978; Ruskin, 1932) [14, 23].

Severe forms, often labeled “ozaena,” denote cases in which fetid odor is a predominant feature (Menzio, 1950) [16]. The condition may occur as simple, subacute, or ozenous variants, reflecting differences in clinical progression and mucosal pathology.

**Bronchitis**

Respiratory disease in swine reflects a multifactorial interplay of infectious agents viral and bacterial as well as environmental stressors. Among the diverse pathogens capable of initiating bronchial inflammation, *Porcine reproductive and respiratory syndrome virus* (PRRSV), *Swine influenza virus* (SIV), and *Porcine respiratory coronavirus* (PRCV) are considered major contributors, often acting synergistically with bacterial agents such as *Bordetella bronchiseptica* (Brockmeier *et al*., 2008) [2]. This microbial complexity frequently culminates in chronic respiratory compromise and variable clinical severity.

**Etiology**

**Atrophic Rhinitis**

The earliest pathogenic event in AR typically involves colonization of the nasal mucosa by *Bordetella bronchiseptica*, which induces acute mucosal inflammation and mild turbinate remodeling. This initial insult facilitates subsequent colonization by toxigenic strains of *Pasteurella multocida*, whose dermonecrotoxin exerts direct cytotoxic effects on osteoclasts and osteoblasts, accelerating turbinate atrophy and septal deviation (De Jong, 1999; Rimler & Brogden, 1986; Pedersen & Barfod, 1982) [5, 20, 19]. Experimental observations confirm that coinfections rather than *P. multocida* alone produce more severe and persistent lesions and significantly impair growth performance (Cowart *et al*., 1989) [3].

**Bronchitis**

The etiology of bronchitis in swine involves a combination of primary and secondary pathogens, including *P. multocida*, *A. pleuropneumoniae*, *B. bronchiseptica*, and *Mycoplasma hyopneumoniae*. Although each microorganism is capable of inducing respiratory pathology independently, concurrent infections tend to amplify tissue damage, prolong disease, and increase economic losses. Despite general recognition of the multifactorial nature of porcine respiratory disease, the specific immunological and microbiological interactions that underpin disease exacerbation remain only partially defined (Brockmeier *et al*., 2008) [2].

**Epidemiology**

**Atrophic Rhinitis**

Atrophic rhinitis is widely recognized as a prevalent condition in swine-producing regions, particularly in the Midwestern United States. Abattoir-based investigations have consistently reported a high burden of turbinate lesions, with some surveys indicating that up to half of market-weight pigs and sows demonstrate varying degrees of conchal atrophy at slaughter (Gardner *et al*., 1994) [7]. The clinical incidence, typically ranging between 5-30%, varies considerably depending on detection method, herd management, and implementation of control programs. Evaluation of snout sections from slaughtered pigs frequently yields lesion rates between 14-50%, although values may be underestimated or overrepresented based on the sourcing of the animals and prior disease-control interventions.

Comparative analyses across farms and production systems have demonstrated substantial variability in lesion scoring, partly due to the historical absence of standardized grading protocols. Recent adoption of structured scoring methodologies has improved diagnostic consistency. Seasonal influences and facility type also contribute to epidemiological differences: a two-season assessment of 21 herds revealed that AR lesions were generally more severe in pigs slaughtered during summer months, while pneumonic lesions predominated in winter (Cowart *et al*., 1992) [4].

Environmental and housing factors play an important role. Pigs reared in enclosed, mechanically ventilated buildings especially when farrowed in centralized farrowing units tend to develop more pronounced turbinate atrophy than pigs raised in outdoor systems such as sow huts or dirt lots. These findings suggest that the severity of observed lesions may reflect not only the conditions present at slaughter but also the historical rearing environment during early developmental stages.

**Pathogenesis**

**Atrophic Rhinitis**

Initial colonization of the nasal epithelium by *B. bronchiseptica* results in close association with ciliated epithelial cells. The organism secretes a heat-labile cytotoxin that induces ciliary dysfunction, epithelial hyperplasia and metaplasia, mucociliary impairment, and early-stage remodeling of the conchal bone within 2-4 weeks of infection. Although this non-progressive turbinate atrophy may partially resolve, toxigenic strains of *P. multocida* can subsequently colonize the altered mucosa more effectively. Once established, *P. multocida* elaborates a dermonecrotoxin that profoundly disrupts bone homeostasis by stimulating osteoclastic activity and suppressing osteoblastic repair mechanisms (Rozengurt *et al*., 1990; Mullan *et al*., 1996) [22, 17].

Experimental studies have demonstrated that intranasal exposure of gnotobiotic pigs to dermonecrotoxin-producing type D *P. multocida* results in rapid and severe bilateral turbinate destruction, with irreversible changes occurring within days and extensive conchal loss evident within 10-14 days (Jordan *et al*., 2003) [9]. The toxin also exerts systemic immunomodulatory effects: pigs harboring toxigenic *P. multocida* exhibit suppressed antigen-specific IgG responses, suggesting altered immune regulation and increased susceptibility to co-infecting pathogens (Jordan *et al*., 2003) [9].

The degenerative process is characteristically non-inflammatory, a unique feature supported by findings that parenteral administration of the toxin alone can reproduce turbinate atrophy in the absence of local infection. Severe cases are associated with facial deformities, including dorsal displacement of the nasal bones, twisting of the snout, and varying degrees of maxillofacial malalignment. These structural changes can impair prehension and mastication, contributing to compromised growth performance. Controlled studies further indicate that AR reduces feed intake and activity levels, particularly under challenging environmental conditions (Van Diemen *et al*., 1995) [24].

Robust experimental models using sequential exposure to *B. bronchiseptica* and toxigenic *P. multocida* in gnotobiotic piglets have proven valuable for elucidating disease mechanisms and evaluating vaccine effectiveness (Ackermann *et al*., 1991) [1].

**Bronchitis**

The innate immune response of the lung orchestrates both the initial defense against pathogens and the subsequent activation of adaptive immunity. Recognition of pathogen-associated molecular patterns triggers rapid secretion of proinflammatory mediators such as TNF-α, IL-1, IL-6, IL-8, and MCP-1, which facilitate leukocyte recruitment and containment of infection (Brockmeier *et al*., 2008) [2]. Coinfection with multiple respiratory pathogens alters these cytokine expression patterns, resulting in more pronounced inflammatory cascades than those observed in single-agent infections. In co-infection models involving *B. bronchiseptica* and PRCV, the combined pathogen burden produces a distinct cytokine signature characterized by amplified proinflammatory signaling and enhanced tissue injury. This synergistic response likely contributes to the more severe histopathological lesions observed during dual infection, including extensive interstitial thickening, epithelial necrosis, and heightened cellular infiltration (Brockmeier *et al*., 2008) [2].

**Clinical Findings**

**Atrophic Rhinitis**

In controlled experimental settings, pigs infected with *P. multocida* or *B. bronchiseptica* generally maintain normal appetite and exhibit unaltered behavior, although sporadic sneezing may occur. The clinical spectrum depends largely on the stage and severity of turbinate degeneration. During the acute phase commonly affecting piglets between 3 and 9 weeks of age irritation of the nasal mucosa leads to frequent sneezing, intermittent coughing, and mild serous to mucopurulent nasal discharge. Transient unilateral or bilateral epistaxis may develop as mucosal fragility increases.

Sneezing frequency has been proposed as an indirect indicator of disease severity and is reduced markedly in piglets derived from sows vaccinated with combined *B. bronchiseptica* and *P. multocida* bacterins compared with piglets receiving *B. bronchiseptica* vaccine alone. Mild ocular epiphora may accompany nasal irritation, sometimes resulting in streaking of dried debris beneath the medial canthus. Growth rates may be negatively affected during this stage.

In infections restricted to *B. bronchiseptica*, clinical signs often resolve spontaneously over several weeks. In more advanced or progressive cases driven by toxigenic *P. multocida*, respiratory compromise intensifies, with thickened nasal secretions, repeated episodes of bleeding, and difficulty nursing in young piglets due to obstruction. Chronic cases exhibit accumulation of inspissated material within the nasal passage that may be forcibly expelled during paroxysms of sneezing.

As maxillofacial deformities evolve, dorsal displacement of the nasal bones, distortion of the premaxilla, and misalignment of the incisors become apparent. Brachygnathia superior, protrusion of lower incisors, and impaired mastication frequently contribute to poor body condition. Severe cases produce characteristic facial "dishing" with overlying skin wrinkling (Hamilton *et al*., 1996) [8].

**Macroscopic & Microscopic Lesions**

**Atrophic Rhinitis**

Gross examination of affected nasal cavities reveals turbinate structures with pale, mottled surfaces and varying degrees of atrophy. Rostral sections typically show epithelial metaplasia with limited neutrophilic infiltration in both the epithelium and submucosa. Occasional epithelial necrosis may be present, though microabscesses are uncommon. Mucous glands in the submucosa may appear compressed or reduced in volume. Histologically, osteoclast numbers are elevated and frequently observed within resorption lacunae along the osseous core of the conchae, reflecting active bone remodelling. As atrophy progresses, conchal architecture becomes severely reduced or absent, often without a significant inflammatory reaction (Hamilton *et al*., 1996) [8].

**Bronchitis**

Clinical signs of bronchitis caused by bacterial or viral agents tend to be mild, presenting mainly as sporadic coughing, sneezing, transient lethargy, and exercise intolerance. Despite the modest clinical appearance, pathological lesions may be more extensive.

**Macroscopic Pulmonary Lesions**

Across infectious groups, lungs typically exhibit multifocal to coalescing areas of dark red consolidation, predominantly affecting the cranioventral lobes. Coinfected pigs those experimentally inoculated with both *B. bronchiseptica* and PRCV show a greater proportion of lung involvement compared with pigs infected with either pathogen alone (Brockmeier *et al*., 2008) [2].

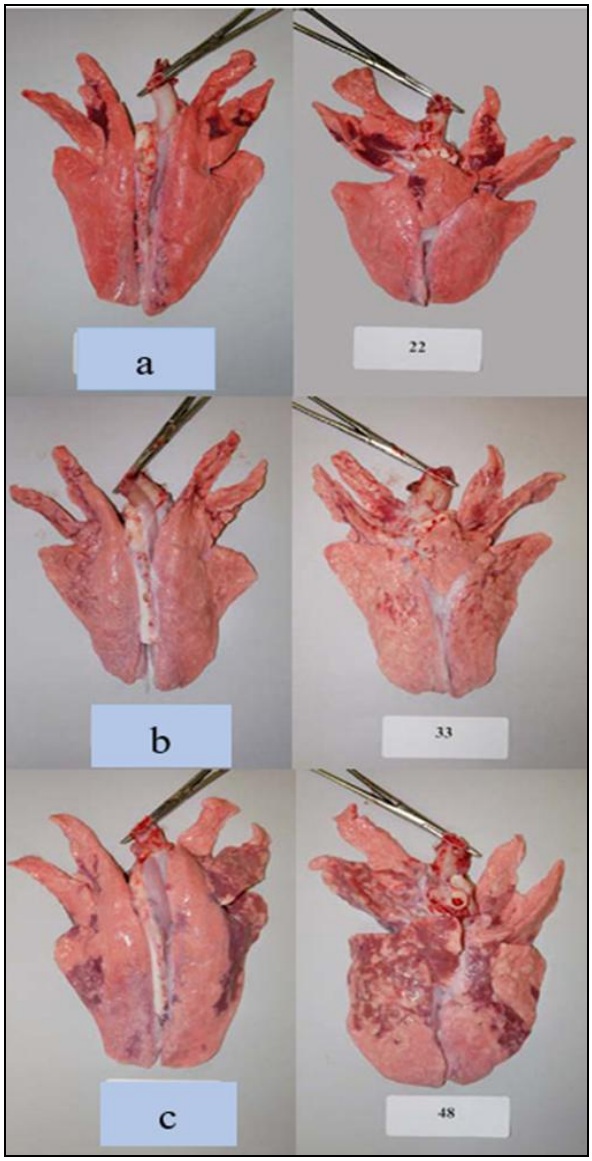


**Fig 1:** Gross lungs showing cranioventral red consolidation, most severe in coinfected pigs.

Regardless of the pathogen involved, the predominant gross lung changes consisted of multiple areas of red consolidation affecting both lungs, most commonly located in the cranial and ventral regions (Figure 1). In the first experiment, pigs infected simultaneously with *B. bronchiseptica* and PRCV showed a greater average proportion of lung affected compared with those infected with either organism alone (Brockmeier *et al*., 2008) [2]. Figure 1 presents representative lungs from the most severely affected pigs in each group: (a) infected only with *B. bronchiseptica*, (b) infected only with PRCV, and (c) coinfected with both pathogens.

These lesions correlate with more severe functional impairment in coinfected pigs and demonstrate the synergistic pathogenic effects of viral-bacterial interaction.

**Microscopic Lesions:**

Histopathological analysis reveals variable patterns depending on the infectious agent:

- ***B. bronchiseptica* alone:** Mild interstitial thickening with sparse mononuclear cell infiltration.
- **PRCV alone:** Moderately thickened alveolar septa with more pronounced mononuclear infiltrates.
- **Dual infection:** Severe interstitial thickening, extensive lymphocytic perivascular and peribronchiolar cuffing, intra-alveolar necrosis, type II pneumocyte hyperplasia, and accumulations of neutrophils, macrophages, and cellular debris. Syncytial cell formation is frequently observed and reflects viral-induced epithelial injury (Brockmeier *et al*., 2008) [2].

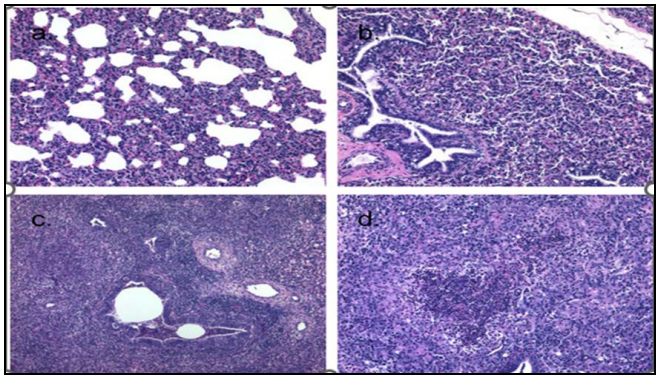


**Fig 2:** Microscopic evaluation of lung sections from the infected pigs showed the following patterns

(a) pigs exposed only to *B. bronchiseptica* exhibited mild interstitial widening of the alveolar walls due to mononuclear cell infiltration;

(b) Lungs from pigs infected exclusively with PRCV displayed moderate interstitial expansion with similar mononuclear infiltrates;

(c and d) pigs coinfected with both *B. bronchiseptica* and PRCV demonstrated pronounced interstitial thickening, prominent lymphocytic cuffs surrounding vessels and bronchioles, and neutrophil accumulation within bronchiolar lumens (c). Additional findings included intra-alveolar aggregates of neutrophils, macrophages, epithelial cells, and necrotic material (d). These tissues correspond to the same animals shown in Figure 1.

Overall, pigs simultaneously infected with PRCV and *B. bronchiseptica* typically developed multifocal areas of moderate to severe interstitial alveolar thickening accompanied by mononuclear cells, lymphocytic perivascular and peribronchiolar cuffing, epithelial necrosis with type II pneumocyte hyperplasia, and the presence of syncytial cells as defining microscopic lesions (Brockmeier *et al*., 2008) [2].

**Diagnosis**

**Atrophic Rhinitis**

**Microbiological Assessment**

Diagnosis often begins with microbiological evaluation of nasal swabs or tissue samples to identify the presence of pathogenic bacteria. Isolation of *Pasteurella multocida* and *Bordetella bronchiseptica* the principal organisms associated with AR provides important etiological evidence (KB & Brockmeier, 2019) [2].

**Serological Analysis**

Serological assays, including ELISA-based detection of antibodies against *P. multocida* and *B. bronchiseptica*, can help determine prior exposure or ongoing infection. Although serology does not distinguish acute from past infections, it contributes valuable epidemiological information (KB & Brockmeier, 2019) [2].

**Molecular Diagnostics**

PCR assays targeting bacterial DNA offer rapid, highly sensitive detection of toxigenic strains, particularly those producing dermonecrotoxin toxin. PCR testing from nasal or tonsillar swabs is effective for large-scale herd screening (Kamp *et al*., 1996) [11].

**Histopathology**

Microscopic evaluation of nasal tissues can reveal epithelial metaplasia, conchal atrophy, glandular compression, and osteoclastic activity, providing definitive pathological confirmation of AR (KB & Brockmeier, 2019) [2].

**Bacterial Isolation and Culture**

Culturing *P. multocida* or *B. bronchiseptica* from nasal samples allows for strain characterization and may support antimicrobial susceptibility testing (KB & Brockmeier, 2019) [2].

**Clinical and Epidemiological Evaluation**

Understanding herd history, introduction of new stock, housing conditions, and the age distribution of affected animals enhances diagnostic accuracy. Epidemiological patterns, such as the presence of carrier animals, also contribute to interpretation (Cowart *et al*., 1991) [4].

Given the economic implications of AR, a comprehensive diagnostic approach incorporating clinical, microbiological, and molecular data is essential to guide prevention and control strategies (Gardner *et al*., 1994) [7].

**Bronchitis**

**Diagnosis of *Bordetella bronchiseptica***

- **Clinical Assessment:** Signs such as coughing, nasal discharge, and respiratory distress suggest possible infection but are non-specific (Brockmeier *et al*., 2008) [2].
- **Bacterial Culture:** Isolation from nasal or tracheal swabs provides definitive identification but requires specialized laboratory techniques.
- **PCR:** Molecular detection of *B. bronchiseptica* DNA offers high specificity and sensitivity (Brockmeier *et al*., 2008) [2].
- **Serology:** Detection of antibodies can confirm exposure but cannot reliably indicate active infection.

**Diagnosis of Porcine Respiratory Coronavirus (PRCV):**

- **PCR:** The primary diagnostic tool for detecting PRCV RNA in nasal or lung tissues (Brockmeier *et al*., 2008) [2].
- **Serological Tests:** ELISAs can detect anti-PRCV antibodies but do not differentiate active from prior infection.
- **Histopathology:** Viral pneumonia typically shows epithelial necrosis, syncytia formation, and interstitial inflammation (Brockmeier *et al*., 2008) [2].

**Treatment**

**Atrophic Rhinitis**

**Antibiotic Therapy**

Antimicrobials, such as tetracyclines and macrolides, are commonly administered to manage secondary bacterial infections and reduce the pathogen load. Appropriate drug selection and dosing must be tailored to herd-level sensitivity patterns. Tilmicosin, when used as a feed additive at 363 g/ton for three weeks, has been reported to reduce lesion severity and improve growth performance in pigs challenged with multiple respiratory pathogens (Olson & Bä, 2000) [18].

**Anti-inflammatory Intervention:**

Nonsteroidal anti-inflammatory drugs (NSAIDs) may be used to moderate inflammation and improve animal comfort (Olson & Bä, 2000) [18].

**Supportive Management**

Nutritional support, clean water access, proper ventilation, and a low-stress environment are essential adjuncts to pharmacological treatment (Olson & Bä, 2000) [18].

**Bronchitis**

**Antibiotics**

For bacterial bronchitis caused by *B. bronchiseptica*, antimicrobials such as tetracyclines, macrolides, and fluoroquinolones may be employed. These treatments primarily target secondary bacterial components of the disease rather than viral etiologies (Brockmeier *et al*., 2008) [2].

**Prevention and Control**

**Atrophic Rhinitis**

**Biosecurity Strategies**

Strict implementation of biosecurity including controlled access to facilities, sanitation protocols, and quarantine of new arrivals is essential to prevent the introduction and spread of AR pathogens (Kobisch, 1989) [12]. Movement of personnel, equipment, and vehicles must be managed to minimize cross-contamination.

**Vaccination**

Vaccination against *B. bronchiseptica* and toxigenic *P. multocida* is a primary preventive measure. Vaccine formulations derived from herd-specific isolates and inactivated with glutaraldehyde, adjuvanted with aluminum hydroxide, have proven effective.

**Standard protocols include:-**

- Sows vaccinated twice late in gestation
- Piglets vaccinated at 7 and 28 days
- Combined sow and piglet vaccination programs (Ross, 2006) [21].
- Herd Management Practices:
- Stress reduction, optimal stocking densities, and maintaining appropriate climatic conditions (temperature, humidity, ventilation) are critical for reducing respiratory disease risk (Kobisch, 1989) [12].

**Genetic Selection**

Long-term mitigation may be achieved through selective breeding for AR-resistant lines (Kobisch, 1989) [12].

**Sanitation**

Routine cleaning, disinfection, and proper waste management help reduce environmental pathogen load.

**Environmental Control**

Adequate ventilation systems reduce ammonia accumulation and airborne microbial exposure, limiting respiratory tract irritation and disease susceptibility (Kobisch, 1989) [12].

**Monitoring and Surveillance**

Routine health assessments and periodic diagnostic testing allow early detection of AR, facilitating timely interventions (Kobisch, 1989) [12].

**Training and Education**

Farm personnel must be well-trained in biosecurity, vaccination procedures, and early recognition of respiratory disease.

**Bronchitis**

**Biosecurity**

Limiting farm access, segregating age groups, and controlling movement of animals and equipment reduce the introduction of respiratory pathogens (Brockmeier *et al*., 2008) [2].

**Vaccination**

Use of vaccines targeting specific bacterial or viral respiratory pathogens should be aligned with regional disease prevalence and veterinary recommendations (Brockmeier *et al*., 2008) [2].

**Environmental and Husbandry Management**

Adequate ventilation, reduced stocking densities, and effective hygiene practices are vital for preventing respiratory stress and cross-infection (Brockmeier *et al*., 2008) [2].

**Diagnostic Monitoring**

Routine respiratory evaluation at the herd level permits early detection of subclinical infection and guides evidence-based interventions (Brockmeier *et al*., 2008) [2].

**Nutritional Support**

Balanced diets that promote immune function enhance herd resilience to respiratory disease challenges.

**Conflict of Interest**

Not available

**Financial Support**

Not available

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