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The Role of Head Associated Lymphoid Tissues in Infectious Bronchitis Virus

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ABSTRACT

Infectious bronchitis virus (IBV) replicates primarily in the epithelial tissues of the respiratory tract, particularly the trachea. However, little is known about IBV replication and immune responses in relation to head-associated lymphoid tissue (HALT), such as the Harderian gland (HG) and choanal cleft, as well as respiratory (turbinate) tissues. Furthermore, few studies have looked into the role of the HG, choanal cleft, pharyngeal tissues, and turbinate in innate, cellular, and mucosal immune responses of commercial broiler chickens and laying hens infected with IBV, compared to the studies about the effects on the trachea. This review article overviewed the role of avian HALT, conjunctival-associated lymphoid tissue (CALT), concerning the anatomical, physiological, and immune responses to infectious bronchitis disease in chickens. The HG, choanal cleft, and turbinate in innate, mucosal, and cellular immune responses play a significant role in avian protection through virulent or attenuated vaccines of IBVs. The IBV viruses could not reach the trachea in chickens vaccinated with IBV vaccine due to the inhibition of viruses by HALT and respiratory tissues by innate, mucosal, and cellular immunity. It can be concluded that other than the trachea, the HALT and respiratory tissues play an important role in the infectivity and immune induction against IBVs due to their proximity to the upper air passages.

Keywords: Avian immunity, Chicken, Harderian gland, Infectious bronchitis, Turbinate

INTRODUCTION

Infectious bronchitis (IB) is a highly contagious and acute disease that can affect broiler, breeder, and layer chickens, resulting in economic losses for the poultry industry (Cavanagh, 2005). The IB is a respiratory infection, prevalent in chickens aged 2-3 weeks, with a high mortality rate and congested respiratory pathways at post-mortem (Schalk and Hawin, 1931). Later, a virus named infectious bronchitis virus (IBV) was identified as the causative agent of this disease (Jackwood 2012). Since the first report, many serotypes and variants have been isolated and characterized (Jackwood, 2012; Jackwood and de Wit, 2013). Many countries have reported multiple variant IBV strains circulating in their poultry farms so far (Sjaak de Wit et al., 2011; Awad et al., 2014; Alsultan et al., 2019; Sabra et al., 2020; Yehia et al., 2020; Jasim et al., 2022).

The Harderian gland (HG) produces all of the local immunoglobulins (IgM, IgA, and IgY) in the lachrymal fluids, which provide local protection in the upper respiratory tract via these immunoglobulins (Baba et al., 1988). It has also recently been reported that using both the Ma5 and 4/91 vaccine strains at the same time results in high levels of immunoglobulins (IgA and IgY) in the upper respiratory tract (URT) and high levels of CD8+ T cells by HG (Smialek et al., 2016).

Following IBV infection, immunoglobulins, such as IgM, IgA, and IgY will increase the tracheal epithelium (Nakamura et al., 1991). The IBV-specific IgA antibodies have been found in the lamina propria, tracheal washes, and epithelial cells of IBV-infected chickens' trachea (Joiner et al., 2007). IgA and IgY antibodies specific to IBV have also been found in lachrymal fluid (Al-Rasheed et al., 2021; Al-Rasheed et al., 2022). It has also been reported that IBV-specific IgA in tears correlates with resistance to IBV reinfection. IBV-specific IgA was first detected in the lachrymal fluid 10 days after vaccination with an attenuated Ark DPI-type IBV live vaccine (Gelb et al., 1998).

Recently, the role of HG, choanal cleft, and turbinate in terms of IBV M41 has been investigated in comparison with trachea. The viral load, pro-inflammatory cytokines (IL-6), and host gene mRNA expression, including Toll-like receptor 3 (TLR3), Melanoma differentiation-associated protein 5 (MDA5), IFN-α and IFN-β in the HALT and respiratory tissues were examined in 21-day-old chickens. After the virulent IBV M41 challenge, the viral RNA expression detected either by quantitative RT-PCR or immunohistochemistry peaked at 2-3 days post-challenge (dpc) in all tissues. Significant increases of lachrymal fluid anti-IBV -specific IgA and IgY levels were found at 4-5 dpc. The

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results demonstrate innate, cellular, and mucosal immune responses at 1-3 days after M41 challenge in the HALT and respiratory tissues (Al-Rasheed et al., 2022).

The host's innate, humoral, mucosal, and cellular immune responses in the HALT and respiratory tissues were also recently evaluated in 41-week-old egg-laying hens following administration of Mass (Mass) or 793B live vaccines, either by oculonasal or drinking water methods (Al-Rasheed et al., 2021).

Infectious bronchitis virus

Infectious bronchitis virus structure

Infectious bronchitis virus (IBV) is the causative agent of IB. It belongs to the genus *Gamma corona virus*, the family Corona viridae, and the order Nidovirales (Cavanagh, 2007). The IBV is a single-stranded (ss) RNA virus with a positive sense, a 120-160 nm diameter, and large surface spikes of 20 nm. The virus has a round to pleomorphic shape with heavily glycosylated spike projections. The virus genome is approximately 27.6 kilobases long. It contains structural proteins, spike (S1 and S2), envelope (E), membrane (M), nucleocapsid (N), and non-structural proteins (Nsps) that are important for virus replication or proliferation. The S1 and S2, cleaved forms of the S protein found on the surface of the viral envelope (virion), have molecular weights of 92 kDa and 84 kDa, respectively. The S1 subunit contains epitopes that induce neutralizing antibodies against IBV, whereas the S2 subunit controls virus fusion to host cells (Ignjatovic and Sapats, 2005; Belouzard et al., 2012). The replicase gene (gene 1) comprises two open reading frames, 1a and 1b, located near the 5' untranslated region (UTR) and the leader sequence. These genes encode proteins that participate in RNA replication and transcription. The *S*, *E*, *M*, and *N* genes are located near the 3' UTR and encode proteins found in virus particles. These structural protein genes are interspersed with Nsps, 3b, 5a, and 5b accessory genes that are not required for replication (Armesto et al., 2012).

Replication of infectious bronchitis virus

The virus replicates in the cytoplasm. The IBV replication begins with S1 binding to cell membrane receptors, specifically those of -2,3-sialic acid (Belouzard et al., 2012; Shahwan et al., 2013). Following that, during biosynthesis, host-cell-dependent proteolytic cleavage of the viral S protein is observed, as is viral envelope fusion with the plasma membrane. The virus enters the cell via fusion with the host plasma membrane or receptor-mediated endocytosis (Brian and Baric, 2005). Virus (+) ssRNA is used as a host polymerase template to synthesize viral RNA polymerase directly. The IBV replication begins with S1 binding to cell membrane receptors, specifically those of α2,3-sialic acid (Belouzard et al., 2012; Shahwan et al., 2013). Subsequently, host-cell-dependent proteolytic cleavage of the viral S protein during biosynthesis is observed as a viral envelope fusion with the plasma membrane. The virus enters the cell via fusion with the host plasma membrane or receptor-mediated endocytosis (Li and Cavanagh, 1992). The first step in virus assembly is the binding of N protein to viral RNA, resulting in the formation of the helical nucleocapsid (Weber and Schmidt, 2005), followed by the integration of the M and E proteins into the membrane of the host cell's endoplasmic reticulum (ER; Vennema et al., 1996). The S protein interacts with the M protein at the pre-Golgi complex, forming an S-M complex (Nguyen and Hogue, 1997).

Strains (variant) distributions

The IBVs have poor serotype cross-protection, highlighting the importance of ongoing identification and surveillance (Jackwood, 2012). The IBV exists in various antigenic or genotypic types, referred to as variants (Sjaak de Wit et al., 2011; Cook et al., 2012). It can produce new variant strains through mutation or gene recombination in the *S1* gene, and can occur due to the introduction of a current strain from another region (Jones, 2010). The mechanism underlying the emergence of new virus types and variants is mainly unknown (Jackwood, 2012). Despite being discovered in the United States, the classical M41 serotype and the Dutch H120 serotype, derived from a 1955 Dutch isolate, are the most widely used vaccine viruses (Sjaak de Wit et al., 2011). Variants have been discovered all over the world (Table 1). Arkansas is the most common type of IBV found in the United States. Connecticut and Massachusetts (Mass) viruses are also frequently detected in the United States (Jackwood et al., 2005; Jackwood et al., 2010; Jackwood, 2012).

Strain classification

Serotypes

The IBV strains are classified into different serotypes based on the antigenicity of the S protein using VN and HI testing (de Wit, 2000). Some laboratories also use enzyme-linked immunosorbent assays (ELISA) with monoclonal antibodies (mAbs) directed against specific epitopes of the S1 protein to distinguish different strains of the virus; however, cross-reactions between serotypes can occur, especially when serum is collected from field samples (Jackwood and de Wit, 2013). The ELISA method has some disadvantages, such as the limited availability of mAbs and the need to develop new mAbs for each new variant (Karaca et al., 1992). Furthermore, due to the limited availability of an increasing number of reference sera associated with various serotypes, VN and HI are not commonly used for serotyping studies.

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Genotype

In recent years, traditional serotyping methods used to demonstrate field strain identity have been replaced by DNA sequencing and genotyping based on the S1 region of the spike gene (Jackwood et al., 1992; Cavanagh et al., 1999; Lee et al., 2000; Jackwood and de Wit 2013). Strains are classified using this method based on the genetic characterization of the S1 subunit, particularly the S1 hypervariable region (Cavanagh 2005).

Protectotype

The most important system from a practical standpoint is protectotype or immunotype classification, playing an important role in the efficacy of vaccine programs in the field. Strains that induce cross-protection against each other, such as M41 (de Wit, 2000) and QX-like, belong to the same protectotype (Bru et al., 2017). However, vaccine strains that are not serologically linked (belong to a different serotype) may still provide cross-protection. For instance, the live H120 vaccine was shown to induce protection against an Australian T strain challenge or variant isolates in commercial farms (Darbyshire, 1985; Awad et al., 2015). A cross-immunization challenge study is required to determine a strain's protectotype. However, this type of research is time-consuming, costly, and necessitates a large number of birds and isolation facilities (de Wit, 2000; Sjaak de Wit et al., 2011). Alternatively, tracheal organ cultures or oviduct organ cultures from vaccinated hens were used in a cross-immunity test; these cultures were challenged using in vitro heterologous or homologous inoculation strains to assess cross-immunity (Raj and Jones, 1996).

Table 1. Infectious bronchitis virus strains types reported worldwide in broiler chickens

Country	Strains/Types	Reference
United State	Beaudette M41 Arkansas Connecticut Mass types California variant Delaware GA08 GA98	(Beaudette and Hudson, 1937) (Bracewell, 1975) (Jackwood, 2012; Jackwood et al., 2010; Jackwood et al., 2005)
Brazil	BR I BR II Mass 793B	(Villarreal et al., 2010)
Mexico	Connecticut Maxx Arkansas	(Jackwood, 2012)
United Kingdome	793B (CR88, 4/91)	(Parsons et al., 1992; Adzhar et al., 1997; Worthington et al., 2008).
Italy	Italy O2 D274 D1466 793B	(Worthington et al., 2008; Sjaak de Wit et al., 2011)
Australia	"T'' strain 793B	(Cumming 1963)
China	QX Q1	(Yudong et al., 1998) (Yu et al., 2001)
Moroccan	G strain 4/91	(El-Houadfi et al., 1986) (Jones et al., 2004)
Libya	IS/885/00 IS/1494/06 (Variant 2)	(Awad et al., 2014a)
Iran, Iraq, and Saudi Arabia (Middle East)	Variant 2 Mass 793B QX Q1	(Alsultan et al. 2019; Amin et al. 2012; Boroomand et al. 2011; Ganapathy et al., 2015)

Anatomy, physiology, and immune responses of head-associated lymphoid and respiratory tissues

The respiratory and lymphoid tissues in the head are the most likely lymphoid tissues in poultry to be initially exposed to vaccines or pathogenic respiratory agents. Since most respiratory vaccines are delivered via spray or eye/nose drops, understanding the immune responses in the head-associated lymphoid tissue (HALT) and upper respiratory tissue in domestic poultry is important. The IBV targets the mucosal surface and periocular lymphoid tissue (van Ginkel et al., 2008). The HG, conjunctiva-associated lymphoid tissue, and lymphoid follicles distributed throughout the mucosal

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surfaces are the primary inductive sites of mucosal immunity in the HALT (Maslak and Reynolds, 1995). There has been little research on the role of HALT and respiratory tissues, including the turbinate, in immune responses against vaccine or virulent strains of IBV in chickens (Al-Rasheed et al., 2021).

Harderian gland

The HG is a major periocular gland of domestic birds that is an immune-endocrine organ located in orbit behind the eye. It is located in the ventral orbit, posteromedial to the eyeball (Wight et al., 1971; Mobini, 2012; Kaiser and Balic, 2015). Since it is loosely connected to the periorbital fascia, it may remain in orbit after eye removal (Dyce et al., 2010; Olah et al., 2014, Figure 1). The HG was first introduced in 1964 (Dyce et al., 2010). Many anatomical studies on these glands, known as HGs, have been published (Davelaar and Kouwenhoven, 1976; Olcese and Wesche, 1989; Scott et al., 1993; Spalevic et al., 2012). The HG is the main orbit gland in birds (Walls 1942) and plays an important role in local eye and upper respiratory immunity, as well as lubricating and cleaning the nictitating membrane via an excretory duct (Burns 1992; Kaiser and Balic, 2015).

The gland is structurally divided into head and body sections based on differences in the surface epithelium and underlying lymphoid organization (Olah et al., 1996). The HG head resembles a typical secondary lymphoid organ, with B cell-dependent germinal centers, the follicle-associated epithelium, and T cell-dependent interfollicular regions containing isolated T cells and macrophages. Several B-lymphocytes and plasma cells are found in the HG body. The B and T lymphocytes are classified according to their developmental stage (Davison et al., 2008). Since HG contains a large population of plasma cells, it significantly affects the immunity of the eye and upper respiratory tract. Furthermore, it is thought to be a significant contributor to antibody production and protect the oculonasal mucosa from airborne viruses, such as IBV, infectious laryngotracheitis, and avian metapneumovirus (Bang and Bang, 1968; Toro et al., 1996; van Ginkel et al., 2008; Spalevic et al., 2012). Bang and Bang (1968) reported chickens raised in a germ-free environment developed lymphocytic infiltrates in the HG, showing that lymphoid tissue within the HG can be induced without microbial stimulation although the possibility of inflammatory agents in the environment cannot be completely ruled out. Van Ginkel et al. (2009) used HG mRNA analysis to confirm the expression of the polymeric immunoglobulin receptor. These findings highlighted the importance of the HG in producing mucosal and systemic immunity against a pathogen, such as avian influenza in chickens following the ocular administration of adenovirus vaccine (Ad5-H5).

From day 5 of age, the HG in young chicks contains single HIS-C7-positive leukocytes and small groups of positive leukocytes in the connective tissue glandular lobes. In addition, B cells, macrophages, and heterophils are present (Mobini, 2012). The number of plasma cells dramatically rises with age, and these leukocytes are located close to tubular ducts and inter-alveolar connective tissue (Savage et al., 1992). Furthermore, the HG is distinguished by many plasma cells capable of proliferating in the area (Scott et al., 1993). Olah et al. (1996) found many IgM and IgA-producing plasma cells in the HG, but only a few IgY plasma cells. In contrast, Jeurissen et al. (1989) found IgY+ plasma cells with IgY in the overlaying epithelium, but only in birds older than 6 weeks old. In 10-week-old chickens, Jalkanen et al., (1984) discovered more cytoplasmic plasma cells c-IgY+ than c-IgM+ and only a few c-IgA+ cells. Lymphocytes from the bursa Fabricius are seeded into the HG prior to hatching and do not appear to be involved in systemic immunity (Baba et al., 1988). Van Ginkel et al. (2008) discovered that 70-90% of HG lymphocytes come from the Fabricius bursa and 10% from the thymus. They also found that HG has many B cells with surface immunoglobulins. Although HG is thought to be the primary source of IgA in lachrymal fluid and not derived from serum IgA migration, IgY+ plasma cells have been observed in the lachrymal fluid contribution. IgA was found in the lachrymal fluid of healthy 2-week-old chicks, and levels increased with age, reaching 0.2mg/ml at 15 weeks. IgY is initially derived from maternal antibodies, decreasing over the first 3 weeks of life before gradually increasing to approximately 2-3 mg/ml at the age of 15 weeks (van Ginkel et al., 2008).

It is still unclear how environmental antigens are absorbed, processed, and presented in the lachrymal fluid, resulting in humoral antibodies. According to Survashe et al. (1979), the HG immune response starts in the lymphoid tissue near the gland duct's access point to the nictitating membrane. According to some researchers, at least in the case of the turkey, antigen uptake takes place in the lower eyelid and is processed in the lymphoid tissue connected to the conjunctiva, producing plasma cells in the HG (Fix and Arp 1989, 1991).

Using the neutralization test, the immunization of 1-day-old chicks with high neutralization index values using the H120 vaccine virus through conjunctival and intranasal routes could result in immunity after 4 weeks. This result is as significant as the induced immunity four weeks later, comparable to that attained by immunizing 20 and 15-day-old chicks with lower levels of maternal antibodies. Successful vaccination is associated with significant stimulation of the HG in the 15-day-old age groups and a rise in lymphocytes and plasma cells, implying that the HG is important in the immune response against IBV (Al-Rasheed et al., 2021).

Choanal cleft

A literature search has revealed that few publications on chicken currently focus on choanal cleft. The shape of the avian choanal cleft varies by species. The cleft in fowl and pigeons is very long, whereas it is very short in ducks and geese (Nickel et al., 1977). There are six transverse rows of caudally directed filiform papillae on either side of the choanal cleft, as in many avian species, papillae are behind the median palate ridge (Figure 2). The palates of fowl and pigeons have caudally pointing papillae arranged in several transverse rows. In contrast, the palate of the goose has a median row of papillae and 2-3 rows of blunt papillae, which are confined to the optical region (Dyce et al., 2010). Recently, some studies have focused on immune responses against IBV in this tissue, the role of HG and choanal cleft in terms of IBV M41 viral

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load were compared to those of trachea, and immune (innate, cellular and mucosal) responses were studied in 21-day-old commercial broiler chickens. Findings from these studies showed active IBV replication in the HALT (HG and choanal cleft) and turbinate tissues, and the limited subset of immunity-related genes provided further understanding of the immunobiology of IBV in naïve 21-day-old commercial broiler chickens. Such effects were dependent on the tissue type, with significant changes in TLR3, MDA5, IFN-α, and IL-6 mRNA expression in the turbinate and trachea being most notable. Meaningfully, the data highlighted the significant presence of both IgA and IgY in lachrymal fluid following the IBV M41 challenge, suggesting that early detection of both IBV-specific IgA and IgY in lachrymal fluid can be used as important indicators of mucosal immune responses of IBV M41 infection in commercial broiler chickens (Al-Rasheed, 2020; Al-Rasheed et al., 2022).

Turbinate

Conchae or turbinate projections are covered projections within the nasal cavity of extant reptiles, mammals, and birds (Geist, 2000). Birds and mammals typically have an additional nasal cavity elaboration (Hillenius 1992; Witmer, 1995). In contrast to the straightforward conchae structure of reptiles, the avian respiratory turbinate is a highly convoluted, frequently scrolled structure lined with moist mucociliary epithelium (Dyce et al., 2010). The spiral structure of the turbinate increases the surface area of the nasal mucosa, which may prevent dust and foreign matter from entering (Kang et al., 2013). The anterior and middle conchae of the respiratory turbinate of birds are similar to mammalian maxilloturbinates. They are located directly in the path of the nasal epithelial mucosa and lessen the efficiency of respiratory air from the mucosal surface (Geist, 2000). In contrast to the bony respiratory turbinate found in mammals, the avian turbinate is usually cartilaginous. The turbinates are paired and serve as intermittent counter current heat exchangers in the nasal cavity (Schmidt-Nielsen et al., 1969, Figure 3). The innate, cellular, and mucosal responses of the immune system were examined in 21-day-old commercial broiler chickens to see how the turbinate differs from the trachea in terms of IBV M41 virus load. The recent work has demonstrated increased viral loads in the turbinate, HG, and choanal cleft tissues in 21-day-old chickens at 2-3 dpc, which indicated localised infection and persistence of IBV at these tissues compared to tracheal tissues. The early innate, cellular, mucosal and humoral immune responses were also tested on a daily basis in M41-challenged chicks. Gene transcription showed a significant up-regulation of TLR3, MDA5, IL-6, IFN-α and IFN-β, where patterns and magnitude fold-change of mRNA transcription were dependent on the gene and tissue type (Al-Rasheed et al., 2022). Viral load and immune responses in the HG, turbinate and choanal cleft showed that tissues other than the trachea should be considered in IBV immunopathogenesis studies.

Trachea

The presence of lymphoid tissue in the avian trachea has not been described yet. Nonetheless, the tracheal mucosa responds vigorously to infection in Mycoplasma gallisepticum infection models, as evidenced by extensive lymphocyte infiltration following lymphoproliferation (Gaunson et al., 2000, 2006). In contrast to CD4+ cells, dispersed throughout the tracheal mucosa, CD8+ cells are found in groups or structures resembling lymphoid follicles. B cells actively proliferating are a characteristic of mycoplasma-induced tracheal lesions (Gaunson et al., 2006). Similar responses were seen in the tracheal mucosa following IBV infection. The IBV-induced lesions are associated with high heterophil and lymphocyte infiltration in the tracheal lamina propria. The formation of many lymphoid follicles and the infiltration of plasma cells heals tracheal lesions 2 weeks after IBV infection (Kotani et al., 2000a; Kotani et al., 2000b).

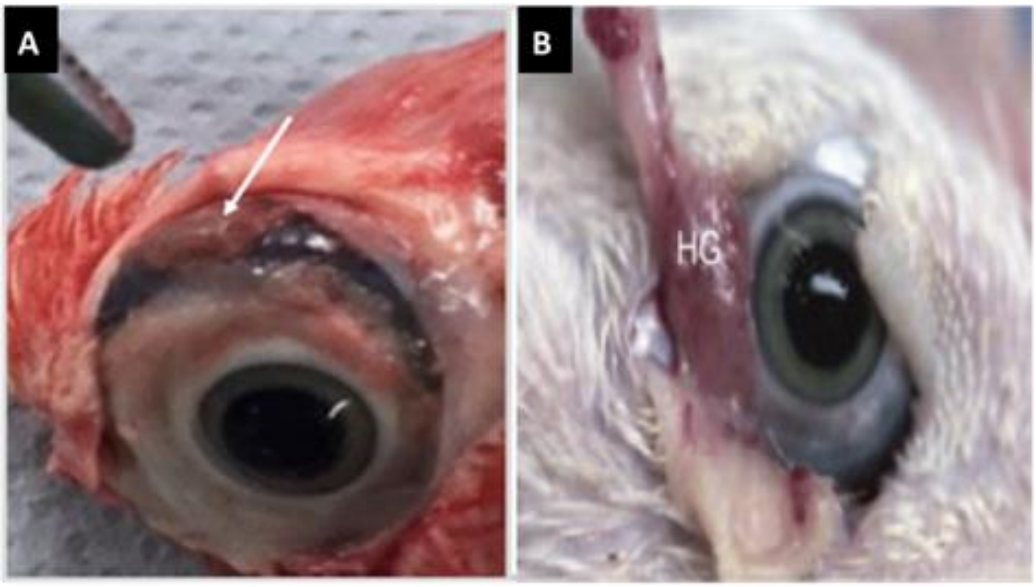

Figure 1. Anatomy of the Harderian gland (HG) in the chicken. The dorsoventral view of the chicken's skull at necropsy with removed skin and the head's rostral aspect to the right. (A) White arrow indicates the left side of HG. By pulling the nictitating membrane with forceps, the attached HG can be withdrawn from the medial surface of the orbit (B; Olah et al., 2014)

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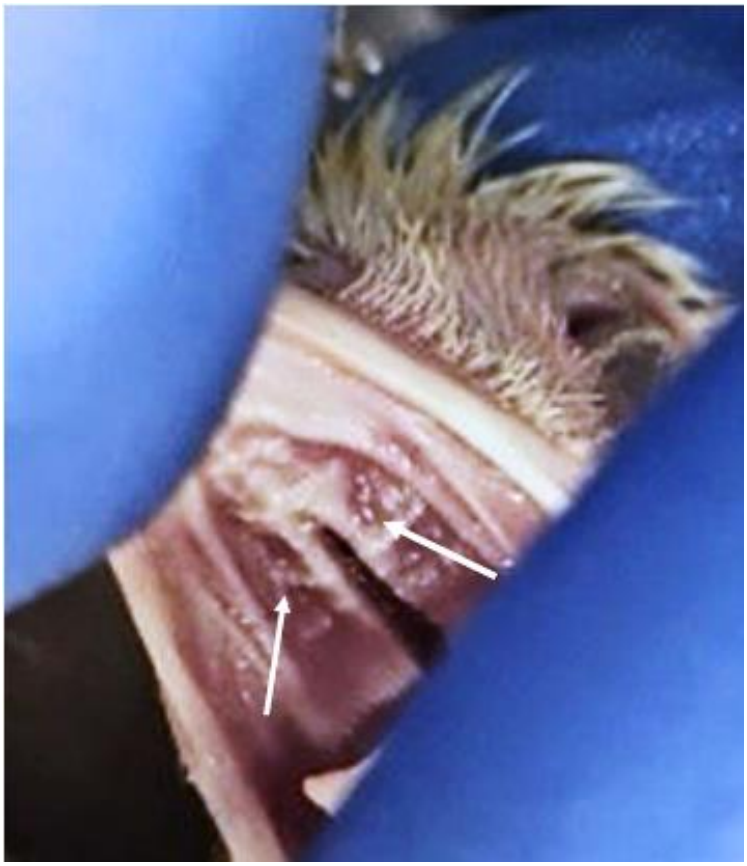

Figure 2. Interior of the upper mandible showing choanal cleft and choanal papillae inside the mouth of a healthy chicken (Al-Rasheed, 2020).

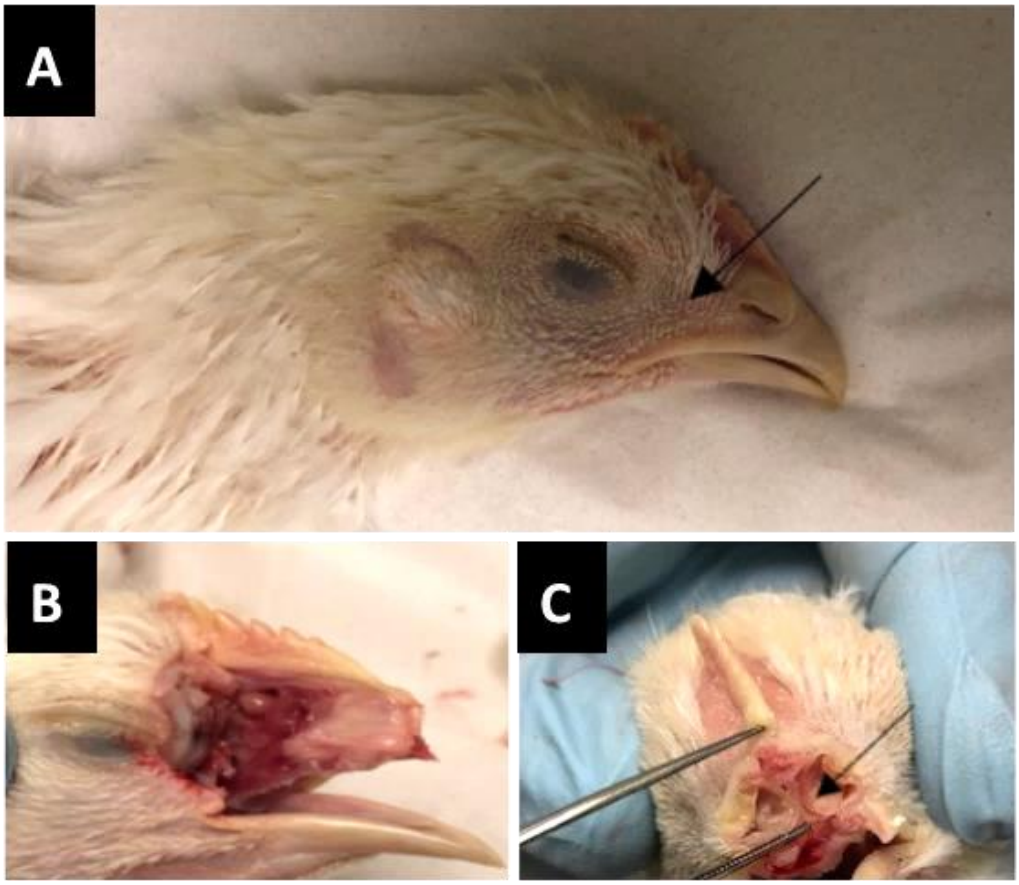

Figure 3. Lateral view of a chicken's head. Right lateral of the head showing the nasal cavity and nostril (A, Arrow). Nasal cavity through a longitudinal sectional view of a chicken's skull (B). Transverse view of the cross-sections from a chicken's nasal cavity showing the spiral structure of a chicken's turbinate, and a pair of turbinate's was located on the wall of the nasal cavity, which is termed concha nasalis media (C, Middle turbinate, arrow; Al-Rasheed, 2020).

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Pathogenesis of infectious bronchitis virus

Infectious bronchitis virus is primarily epitheliotropic, causing lesions in the kidneys, reproductive organs (testes, oviduct), lungs, and respiratory tract (nasal turbinate, trachea, HG) after replicating. The virus can also reproduce in different gastrointestinal tract cells, frequently resulting in mild lesions (Raj and Jones, 1997). It can alter the small intestine macroscopically and microscopically, which could be related to mutations that alter the S1 structure (Hauck et al., 2016). Although all IBV strains infect the respiratory system of avians, it later spreads to target tissues for replication and persistence within 18-36 hours (Jackwood and de Wit, 2013).

Harderian gland

Following experimental infection with live attenuated H120, IBV was isolated from the HG (Toro et al., 1996). This study has concentrated on the role of this lymphoid tissue in mucosal immunity. Recently, researchers investigated the role of HG, choanal cleft, and turbinate in terms of IBV M41 viral load in 21-day-old commercial broiler chickens. The antigen concentration peaked at 2-3 dpc in all head-associated lymphoid and respiratory tissues. At 4-5 dpc, there was a significant increase in lachrymal IBV-specific IgA and IgY levels, and gene transcription showed a significant up-regulation of TLR3, MDA5, IL-6, IFN-α, and IFN-β, where patterns and magnitude fold-change of mRNA transcription, demonstrating active IBV M41 replication in the HG, CC, and turbinate, compared to levels of replication found in the trachea (Al-Rasheed et al., 2021; Al-Rasheed et al., 2022).

Turbinate

In a study, nasal turbinate organ cultures inoculated with six different IBV strains (H52, H120, M41, Connecticut, Australian T strain, and British field strain HV-10) yielded maximum viral titers 48-72 hours after infection (Darbyshire et al., 1978). Following vaccination with live IBV H120, the antigen was detected in the turbinate organ culture (Darbyshire et al., 1976). Dolz et al. (2012) used in-situ hybridization to isolate and identify the presence of viral RNA of the IBV Italy 02 serotype in the nasal turbinate prior to detection in the trachea. Recently, IBV-attenuated vaccines were found in Mass and 793B-vaccinated hens after M41 challenge. In airborne infectious, the turbinate is likely the first defensive barrier (Al-Rasheed et al., 2021; Al-Rasheed et al., 2022).

Trachea

The tracheal epithelium was examined for lectin reactivity, and it was discovered that the susceptible cells have high levels of 2,3-linked sialic acid expression. According to these findings, 2,3-linked sialic acid is essential in IBV infection on the respiratory epithelium (Winter et al., 2008). The IBV replication has previously been observed in tracheal ciliated and mucus-secreting epithelial cells (Yagyu and Ohta, 1990; Nakamura et al., 1991; Owen et al., 1991; Benyeda et al., 2010), and IBV is frequently isolated from the trachea (Otsuki et al., 1990; Janse et al., 1994; Lee et al., 2000). The surveillance of the virus in the trachea depends on the virus strain. By contrast, 793B was isolated from the trachea of infected specific pathogen-free (SPF) chicks up to 7 days post-infection. In contrast, infection by strain G results in the highest viral titers at 3 days post-infection, with viral isolation observed up to 14 days post-infection (Ambali and Jones, 1990; Raj and Jones, 1996). Tracheal dysfunction has been linked to IBV strain virulence that could measure by ciliary activity (Otsuki et al., 1990; Dhinakar Raj and Jones, 1997). According to the cilia-stopping test, the QX-like and 793B strains were the least virulent, while the M41 infection was more severe (Benyeda et al., 2009).

CONCLUSION

There is a dearth of information about the role of head-associated lymphoid tissues (HALT) in chickens. The research presented in this review has provided evidence that other than the trachea, the HALT and respiratory tissues play an important role in the infectivity and immune induction against the IBVs due to their proximity to the upper air passages. Based on IBV qRT-PCR and immunohistochemistry, it is demonstrated that IBV actively replicates in HALT (HG and choanal cleft) and turbinate tissues other than the trachea, leading to infection and immunobiology. An essential finding was the role of anti-IBV IgA and IgY in the lachrymal fluid as a quantitative IBV vaccine efficacy biomarker. The findings highlighted the role of gene signatures, type 1 interferons, and cytokines following virulent or vaccine inoculation of young and adult chickens. As a result, a series of studies must be conducted to indicate the mucosal, humoral, and cellular immunity mechanisms of the HALT in chickens given either vaccine or virulent IBV viruses.

DECLARATIONS

Authors' contribution

The idea was created by Mohammed Al Rasheed, who also carried out the laboratory work. Mohamed Al Rasheed and Mohamed Shawky, who are both authors, contributed to the writing, editing, and creation of the final draft. The final submission was approved and confirmed by all authors.

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Ethical consideration

All authors check plagiarism, consent to publish, misconduct, data fabrication and/or falsification, double publication and/or submission, and redundancy already.

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Competing interests

The authors claim to have no conflicts of interest.

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