CEVA HANDBOOK
OF POULTRY DISEASES

8 INFECTIOUS CORYZA

INFECTIOUS CORYZA

DEFINITION

Infectious Coryza (IC) is an infectious contagious disease affecting the upper respiratory tract of fowl, manifested mainly with via rhinitis, conjunctivitis and infraorbital sinusitis.

HISTORY

The disease was first described in the 1920s (Beach, 1920). After successful isolation and identification in the Netherlands, the etiological agent was named Bacillus hemoglobinophilus coryzae gallinarum (De Blieck, 1932). After bearing the name Haemophilus paragallinarum for a long period of time, a recent reclassification determined the bacterial agent of IC as Avibacterium paragallinarum (Blackall et al., 2005). Over the years, other known synonyms of disease include: Coryza, diphtheria, contagious or infectious catarrh (Blackall & Soriano, 2008).

CHARACTERISTICS AND CLASSIFICATION OF THE PATHOGEN

The etiological agent behind IC is *Avibacterium paragallinarum*, a gram-negative, non-motile, short, coccus-like, non-spore-forming rod. The isolation of the bacterium from inflammatory exudate in nasal orifice mucosa is possible only in the early stages of the disease. Later, due to development of side microflora, it becomes difficult or impossible (Obreshkov et al., 1978).

Page (1962) reported that all strains isolated from IC cases required only V-factor (nicotinamide adenine dinucleotide – NAD) to grow, but not both "V" and "X" (hemin) factors as had been previously suggested (Shalm & Beach, 1936). The growth medium must therefore be selected with care. *Av. paragallinarum* usually grows in an atmosphere supplemented with 5% carbon dioxide, but may also develop in reduced oxygen or anaerobic environments. The optimal culture temperature is 34–42°C (Blackall & Soriano, 2008). In vitro, most *Av. paragallinarum* isolates require the reduced form of NAD

to grow. Horner et al. (1995) report the occurrence of NAD (V-factor) - independent commercial chicken isolates in South Africa. Although NAD-independent, these isolates share an identical DNA profile with *H. paragallinarum* (Mouahid et al., 1992). Some strains may require supplementing of the culture medium with 1% chicken serum, while in others this supplement only improves growth (Blackall & Reid, 1982). This relatively poor growth should be taken into consideration, as in a number of cases it is a cause for reporting negative culture results (Blackall, 1983).

It is acknowledged that the microbial agent of IC may reduce nitrates to nitrites, ferments glucose without formation of gas, exhibits oxidase activity and is not able to form indole (Blackall et al., 2005). For serological classification of *Av. paragallinarum* strains, the system proposed by Page is the most popular, defining three different serovars (A, B and C) by agglutination testing (Page, 1962). The worldwide distribution of Page's serovars is different. The hypothesis that serovar B was not a true serovar (Kume et al., 1983) was proven to be false (Yamaguchi et al., 1991). On the basis of haemagglutination inhibition (HI) test, a modified scheme consisting of 9 serovars has been proposed (Kume et al., 1983) but it did not find a wide application. The importance of serotyping is that inactivated vaccines protect only against the serovar they contain (Blackall et al., 1997). The three Page's serovars A, B and C are in fact different immunovars. Bivalent vaccines containing Page's A and C serovars protect against the Spross strain of serovar B, but not against the Page serovar B South African isolates (Yamaguchi et al., 1991).

Based on serovar-specific monoclonal antibody tests, and the failure of commercial vaccines, the appearance of variants or unusual serovars was assumed (Terzolo et al., 1993; Blackal et al., 1994). There is evidence that serovar B isolates from Argentina are genetically very different from other strains of this *Av. paragallinarum* serovar (Bowles et al., 1993). This would imply that the unique nature of Argentine an serovar B isolates distinguishes them from vaccinal strains encountered in Europe and North America and, therefore, protection can not be ensured (Terzolo et al., 1997).

Pathogenicity varies among the different *Av. paragallinarum* isolates depending on a number of factors. It is believed that the capsule plays a significant role and that it is associated with colonization and IC lesions (Sawata et al., 1985). The clinical manifestations of the disease

were suggested to be caused by a toxin, released by pathogens during their in vivo multiplication (Kume et al., 1983). It was also demonstrated that the capsule of *Av. paragallinarum* protects it from the bactericidal activity of normal chicken serum (Sawata et al., 1984). The resistance of the organism is not high. In excreted inflammatory exudate outside the host, *Av. Paragallinarum* perishes rapidly. Temperatures of 45–55°C kill it after 2–10 min (Blackall & Soriano, 2008). It may survive in a refrigerator at positive temperatures for up to 2 weeks, and in a lyophilized state For up to 2 years (Obreshkov et al., 1978).

EPIDEMIOLOGY

The main sources of infection are sick and remitted birds which contaminate the environment with nasal, conjunctival and oral discharges. Birds which recover become reservoir of infection for a long time (a year or more) (Obreshkov et al., 1978). Inactivated vaccines providing inefficient immunity, especially some vaccinal strains from serovars with unsatisfactory cross protection, could also play a role in IC spread (Rimler et al., 1977). Epidemiological research has shown that *Av. paragallinarum* may be introduced to isolated farms by the aerogenic route (Yamamoto & Clarc, 1966).

IC is encountered in all poultry production regions in the world. Backyard, village and hobby poultry all contribute to the persistence of infections. Chickens are susceptible to *Av. paragallinarum*, where as other avian species such as turkeys, ducks, pigeons, crows and sparrows are not susceptible to experimental infection (Yamamoto, 1972; 1978). Birds of all ages are affected but the disease is more pronounced during the active egg laying period. In juvenile birds, the course of IC is milder (Blackall & Soriano, 2008). The economic impact of IC mainly affects the egg production industry although cases in poultry raised for meat are reported (Droual et al., 1990). The penetration of infection into disease-free farms occurs by carrier birds (having remitted from infection). Such birds present varying levels of immunity, and after re-exposure could become ill

again. Via direct or indirect contact (nasal discharge, contaminated watering and feeding troughs) or aerogenically at short distances, lateral transmission may occur. In both cases, contact with nasal or pharyngeal mucous coats is direct. Recently, an outbreak of IC was reported in two mixed-age hobby flocks in England, presumably transmitted by asymptomatic carriers (Welchman et al., 2010). In mixed-age flocks, the transmission of infection to susceptible birds usually occurs 1–6 weeks after moving such birds near to groups of infected older birds. The infection is not transmitted through breeder eggs nor via one-day-old chicks. IC is highly contagious and spreads very quickly among flocks (Obreshkov et al., 1978).

Predisposing causes may include poor hygienic conditions: overcrowding, extreme ambient temperature variations, increased humidity, inadequate ventilation, vitamin A dietary imbalance, stress etc. The infection is more common during fall and winter (Blackall & Soriano, 2008).

CLINICAL SIGNS AND PATHOLOGY

The incubation period is short (1–3 days), and disease course is noticeably longer in birds of productive age. Without a concurrent infection, the usual course of IC is 2 to 3 weeks. Clinicomorphologically, IC is manifested in the form of catarrhal rhinitis, sinusitis, and conjunctivitis. More rarely, the inflammation might affect the lower respiratory tract and result in aerosacculitis and pneumonia if secondary microflora are involved. During the disease course, feed and water consumption are reduced and a variable drop in egg production ensues (10–40%). The morbidity rate is high, but mortality is not significant.

Fig.1

One of the first clinical signs of the disease is catarrhal discharge from nasal orifices.

Fig.2

At a later stage, the inflammatory exudate becomes catarrhal, mucinous and sticks around the nasal orifices and the beck.

Fig.3

Frequently, the discharge is mixed with feed particles resulting in crustlike deposits.

Fig.4
A typical clinical sign is facial swelling the region of infraorbital sinuses.

Fig.5
In some instances, facial swelling descends towards the mandibular space and may affect the wattles, especially in male birds.

Fig.6

After the attenuation of the inflammation, the affected wattles may remain wrinkled as a result of organization of the deposited exudate.

Fig.7

In case of concurrent conjunctivitis and secondary contamination (*E. coli*, *St. aureus* etc.) the inflammation affects the eyeball with signs of keratitis and panophthalmitis.

DIAGNOSIS

The disease history, along with clinical and epidemiological Traits, allow for a tentative diagnosis, but this must be confirmed by a laboratory test.
Sterile swabs should be collected from several birds (at least 3-4) deep within the sinuses, where the agent is most likely to be found in pure form, during the first days after the clinical symptoms appear.
The traditional culture method requires extensive biochemical characterization after isolation of the organism (Blackall et al., 1997). *Av. paragallinarum* is a slow-growing, fastidious microorganism.
A specific PCR test for detection of *Av. paragallinarum*–HP-2 PCR (Chen et al., 1996) has been developed. The advantage of HP-2 PCR is that the results are ready after just 6 hours, where as conventional methods may take several days. Both colonies grown on agar and swab samples from the sinuses of live birds are suitable for testing (Chen et al., 1996). Other advantages are the possibility to run the test using samples which have been stored for a long time, or in NAD-independent *Av. paragallinarum* (Chen et al., 1998).
A number of serological tests have been proposed for the detection of antibodies to *Av. paragallinarum* present in chickens (Blackall et al., 1997). HI tests are most widely used. The technique of using monoclonal antibody-based ELISA for serological detection of IC might be a way of overcoming the problems encountered with the detection of Page's serovar B (Blackall, 1999).

DIFFERENTIAL DIAGNOSIS

IC should be distinguished from other avian diseases with respiratory symptoms and lesions: infectious bronchitis, laryngotracheitis, swollen head syndrome, fowl pox (wet pox), chronic respiratory disease, chronic form of fowl cholera.

TREATMENT

Prior to treatment, it should be remembered that antimicrobial resistance has been observed in *Av. paragallinarum*

(Blackal, 1988). In general, the microorganism is sensitive to tetracyclines, erythromycin and sulfonamides.

PREVENTION AND CONTROL OF IC

The prevention and control of IC requires a combination of specific (vaccination) and non-specific (biosecurity) measures.

Vaccines

Available inactivated vaccines are based on bacterins obtained from broth culture. A number of adjuvants efficient for use with bacterins have been identified, while formalin or thimerosal are the most commonly used inactivating agents, despite conflicting opinions about their use (Davis et al., 1976; Blackall & Ried, 1987).

Most of the commercially-available vaccines are based on Page serovars A and C. Before the pathogenicity of serovar B was confirmed, it was thought that bivalent vaccines (serovars A and C) provided cross-protection. The evidence that serovar B was pathogenic and a true serovar meant it was included in inactivated vaccines to be used in regions where it was spread (Jacobs et al., 1992). Since it became clear that the different serovar B strains provided only partial cross protection (Yamaguchi et al., 1991), the importance of using autogenous bacterins or commercial bacterins containing multiple serovar B strains in endemic regions has been clearly demonstrated (Terzolo et al., 1997; Jacobs et al., 2003). Inactivated vaccines are applied in pullets between 8 and 16 weeks of age. The second injection is made 3 to 4 weeks after the first but not later than 2 weeks before laying begins. The dose and the route of administration (intramuscular or subcutaneous) are recommended by the manufacturer.

Biosecurity

The most important biosecurity measures include introduction of IC-free birds to farms, and isolated rearing during the growing and productive periods. If the infection is to be successfully eradicated from the farm, infected or recovered flocks must be depopulated as they remain reservoir hosts of the infection. After a break of 2-3 weeks, preceded by cleansing, washing and disinfection of premises, the farm can be repopulated with birds.

REFERENCES

- Beach, J.R., 1920. The diagnosis, therapeutic and prophylaxis of chicken pox (contagious epithelioma) of fowls. JAVMA, 58, 301-312.
- Blackall, P.J. & E. V. Soriano, 2008. Infectious coryza and Related Bacterial Infections. In: Saif, Y.M., Fadly, A.M., Glisson, J.R., McDougald, L.R., Nolan, L.K., Swayne, D.E. (Eds), Diseases of poultry, 12th ed, Blackwell publishing, Ames, Iowa, USA, 78-803.
- Blackall, P.J. & G.G. Reid, 1982. Further characterization of Haemophilus paragallinarum and Haemophilus avium, Vet. Microbiol., 7, 359-367.
- Blackall, P.J., 1983. An evaluation of methods for the detection of carbohydrate fermentationpatterns in avian Haemophilus species, J Microbiol Methods, 1, 275-281.
- Blackall, P.J. & G.G. Reid, 1987. Further efficacy studies on inactivated, aluminum-hydroxide-adsorbed vaccines against infectious coryza. Avian Diseases, 31, 527 -532.
- Blackall, P.J., 1988. Antimicrobial drug resistance and the occurrence of plasmids in Haemophilus paragallinarum. Avian Diseases, 32, 742-747.
- Blackall, P.J., 1999. Infectious coryza: Overview of the Disease and New Diagnostic Options. Clinical Microbiology Reviews, 4, 627-632.
- Blackall, P.J., H. Christensen, T. Beckenham, L.L. Blackall, & M. Bisgaard, 2005. Reclassification of Pasteurella gallinarum , [Haemophilus] paragallinarum, Pasteurella avium and Pasteurella volantium as Avibacterium gallinarum gen. nov., comb. nov., Avibacterium paragallinarum comb. nov., Avibacterium avium comb. nov., and Avibacterium volantium comb. nov. Int. J. Syst. Evol. Microbiol., 55, 353-362.
- Blackall, P.J., M. Matsumoto, & R. Yamamoto, 1997. Infectious coryza. in: Calnek, B.W., Barnes, H.J., Beard, C.W., McDougald, L.R. & Saif, Y.M., eds.) Diseases of Poultry. Iowa State University Press: Ames, 179-190.
- Blackall, P.J., E.N. Silva, T. Yamaguchi, & Y. Iritani, 1994. Characterization of isolates of avian haemophili from Brazil. Avian Diseases, 38, 269-274.
- Bowles, R., P.J. Blackall, H.R. Terzolo, & V.E. Sandoval, 1993. An assessment of the genetic diversity of Australian and overseas isolates of Haemophilus paragallinarum by multilocus enzyme electrophoresis, p. 146. In Proceedings of the Xth Wolrd Veterinary Poultry Association Congress.
- Chen, X., J.K. Miflin, & P.J. Blackall, 1996. Devolopment and application of DNA probes and PCR tests for. Avian Dis., 40, 398-407.
- Chen, X., Y. Song, Y. Gong, & P.J. Blackall, 1998. Further studies on the use of a polymerase chain reaction test for the diagnosis of infectious coryza. Avian Pathol., 27, 618-24.
- Davis, R.B., R.B. Rimler, &, E.B. Shotts Jr, 1976. Efficacy studies on Haemophilus gallinarum bacterin preparations. Am. J. Vet. Res., 37, 21-222.
- De Blieck, L., 1932. A haemoglobinophilic bacterium as the cause of contagious catarrh of the fowl (Coryza infectiosa gallinarum). Vet. J., 88, 9-13.
- Droual, R., A.A. Bickford, B.R. Charlton, G.L. Cooper, & S.E. Channing, 1990. Infectious coryza in Meat Chickens in the San Joaquin Valley of California. Avian Diseases, 34, 1009-1016.
- Horner, R.F., G.C. Bishop, C.J. Jarvis, & T.H.T. Coetzer, 1995. NAD (V-factor) - independent and typical Haemophilus paragallinarum infection in commercial chickens: a five year field study. Avian Pathol., 24, 453-463.
- Jacobs, A.A.C., W. Guenen, & P.K. Storm, 1992. Efficacy of a trivalent Haemophilus paragallinarum vaccine comprated to bivalent vaccines. Vet. Microbiol., 32, 43-49.
- Jacobs, A.A.C., K. Van Den Berg, & A. Malo, 2003. Efficacy of a new tetravalent coryza vaccine against emerging variants type B strains. Avian Pathol., 32, 265-269.

- Kume, K., A. Savata, T. Nakai, & M. Matsumoto, 1983. Serological classification of Haemophilus paragallinarum with a hemagglutinin system. J. Clin. Microbiol., 17, 958-964.
- Mouahid, M., M. Bisgaard, A.J. Morley, R. Mutters, & W. Mannheim, 1992. Occurrence of V-factor (NAD) independent strains of Haemophilus paragallinarum. Vet. Microbiol., 31, 363-368.
- Obreshkov, K., I. Vasilev, B. Natchev, et al., 1978. Diseases of poultry, Zemizdat, Sofia, pp. 109-112.
- Page, I.A., 1962. Haemophilus infections in chickens. 1. Characteristics of 12 Haemophilus isolates recovered from diseased chickens. Am. J. Vet. Res., 23, 85-95.
- Rimler, R.B., R.B Davis,. & R.K. Page, 1977. Infectious coryza: cross protection studies, using seven strains of Haemophilus paragallinarum. American Journal Veterinary Research, 38, 1587-1589.
- Savata, A., K. Kume, & K. Nakai, 1984. Susceptibility of Haemophilus paragallinarum to bactericidal activity of normal and immune ckicken serum. Jpn. J. Vet. Sci., 46, 805-813.
- Savata, A., K. Nakai, K. Kume, H. Yoshikawa, & T. Yoshikawa, 1985. Intranasal inoculation of chickens with encapsulated or nonencapsulated variants of Haemophilus paragallinarum: electron microscopic evaluation of the nasal mucosa. Am. J. Vet. Res., 46, 2346-2353.
- Schalm, O.W. & J.R. Beach, 1936. Studies on infectious coryza of chickens with special reference to its aetiology. Poult. Sci., 15, 473-482.
- Terzolo, H.R., F.A. Paolicchi, V.E. Sandoval, P.J. Blackall, T. Yamaguchi, & Y. Iritani, 1993. Characterization of isolates of Haemophilus paragallinarum from Argentina. Avian Diseases, 37, 310-314.
- Terzolo, H.R., V.E. Sandoval, & F. Gonzales Pondal, 1997. Evaluation of inactivated infectious coryza vaccines in chickens challenged by serovar B strains of Haemophilus paragallinarum. Avian Pathol., 26, 365-376.
- Welchman, D.B., S.A. King, P. Wragg, et al., 2010. Infectious coryza in chickens in Great Britain. Veterinary Record, 167, 912 -913.
- Yamaguchi, Y., P.J. Blackall, S. Takigami, Y. Iritani, & Y. Hayashi, 1991. Immunogenicity of Haemophilus paragallinarum serovar B strains. Avian Dis., 35, 965-968.
- Yamamoto, R. & G.T. Clark, 1966. Intra- and interflock transmission of Haemophilus gallinarum. Am. J. Vet. Res., 27, 1419-1425.
- Yamamoto, R., 1972. Infectious coryza. in: Hofstad, M.S., Calnek, B.W., Helmboldt, C.F., Reid, W.M. & Yoder Jr, H.W. (eds.). Diseases of Poltry, 6th ed. Iowa State University Press: Ames, 272-281.
- Yamamoto, R., 1978. Infectious coryza. in: Hofstad, M.S., Calnek, B.W., Helmboldt, C.F., Reid, W.M. & Yoder Jr, H.W. (eds.). Diseases of Poltry, 7th ed. Iowa State University Press: Ames, 225-232.