Chicken Infectious Anaemia and Co-Infections

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Submission: August 06, 2018; Published: September 20, 2018

Mini Review

Chicken anaemia virus (CAV) was first isolated by Yuasa and colleagues in Japan in 1979 during the investigation of Marek’s disease outbreaks. The virus experimentally produced severe anaemia and death in chickens [1]. It belongs to Gyrovirus genus of Circoviridae family and is resistant to thermal inactivation and treatment with lipid solvents and many of the common desinfectants [2,3]. The virion of CAV is a small (19-26, 5nm in diameter) nonenvelopedicosahedron [4] and contains a dose circular negative single stranded DNA (2,3kb) [5,6]. The genome codes for three viral proteins: VP1, the 51,6kDa structural capsid protein; VP2, a 24kDa non-structural protein with dual-specificity phosphatase activity; and VP3, the smallest protein (13,6kDa) known as apoptin which induces apoptosis in erythrocyte precursors and thymocytes, resulting in immunodeficiency [7]. For the production of neutralizing antibodies, co-synthesis of VP1 and VP2 are needed [8].

Chicken infectious anaemia (CIA) caused by CAV is a disease which is characterized by increased mortality, reduced weight gain, severe anaemia, intramuscular and subcutaneous haemorrhages, aplasia of bone marrow and atrophy of the thymus [1,9,10]. CIA occurs among all production types - layer chickens, layer hens, broilers, parent flocks and backyard birds [11-13]. The virus spreads both horizontally and vertically in chickens as the transmission of CAV to the progeny can result in increased mortality in young chicks, therefore infections with CAV are considered to be economically significant [2,3,11]. The virus causes clinical and subclinical disease in chickens and is recognized as an important avian pathogen worldwide [2,13]. The clinical disease is mainly noticed in young chickens of 10-14 days of age [14] as CAV causes anaemia due to the destruction of erythroblastoid cells and depletion of thymocytes in the thymus cortex, resulting in immunodeficiency [1,15]. Older chickens are susceptible to virus replication, but do not develop clinical signs [16]. Studies of CAV outbreak show that the vertical transmission of the virus keeps for 3-6 weeks after the initial infection of the breeder flocks, and during this period the majority of the breeders can become infected and spread the virus in the flock [2].

CAV has the potential to induce immunosuppression alone or in combination with other infectious agents [11]. Immunosuppression leads to enhanced susceptibility to other avian pathogens as well as to reduction in response after vaccination [17]. Multiple studies conducted in the field or experimentally indicate that CAV, as an immunosuppressive agent, causes co-infections with other viral, bacterial and fungal agents as the pathogens mutually act and enhance their effects. Such co-infections with exacerbated clinical picture and increased mortality are described in the end of 20th century with reoviruses (blue wing disease, haemorrhagic anaemia syndrome), adenoviruses (inclusion body hepatitis/hyperproteicardium syndrome), IBDV-Infectious Bursal Disease Virus, MDV-Marek’s disease virus (early mortality syndrome), IBV-Infectious Bronchitis Virus, Clostridium perfringens (gangrenous dermatitis), Staphylococcus aureus [18], in 2003 - with Salmonella enterica [19], and in 2010 with Avian Leucosis Virus-J (ALV-J) [20]. Hornok and colleagues [21] described interaction of chicken anaemia virus and Cryptosporidium baileyi in experimentally infected chickens, and Silveira and colleagues [22] found out interaction of Plasmodium juxtanucleare and chicken anaemia virus.

CAV infections are diagnosed based on the clinical signs, macro- and microscopical findings, PCR, serology and CAV isolation. PCR for in vitro amplification of specific DNA fragment has been emerging at the current stage as a main reference high sensitive and specific rapid molecular assay for detection of CAV DNA [23]. CAV isolation is carried out using MDCC-MSB1 cell line [24] and,
although it is laborious and slow, this technique is indicative for the presence of infectious virus in tested sample [25] and enables its further antigenic and molecular characterisation.

References