Chicken Anemia Virus

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ABSTRACT Chicken anemia virus is commonly found in commercially produced chickens and has a worldwide distribution. It is difficult to inactivate thermally or with common disinfectants, which limits the utility of normal sanitization practices. The virus is important because of the disease it produces following transovarian transmission and because of its potential for inducing immunosuppression alone or in combination with other infectious agents. Control measures are directed at limiting vertical transmission and preventing coinfections with other lymphocidal agents.

(*Key words*: chicken, chicken anemia virus, immunosuppression, anemia, disease)

1998 Poultry Science 77:1190–1192

INTRODUCTION

The chicken anemia virus (CAV) was first described in 1979 by Yuasa *et al.* (1979) in commercially produced chickens. Since that time, the virus has been detected by isolation or serology in most other countries in both laying and broiler chickens (von Bulow and Schat, 1997). The first report of a CAV isolation in the U.S. was by Rosenberger and Cloud (1989a). It is clear, however, based on retrospective serological and clinical evaluations that the virus has existed in the U.S. for at least 25 yr.

The virus, when transmitted by the transovarian route, can result in severe disease in progeny characterized by anemia, subcutaneous hemorrhage, and a decreased resistance to secondary bacterial diseases such as gangrenous dermatitis. Affected birds, if coinfected with infectious bursal disease virus, may develop a profound immunosuppression with enhanced susceptibility to a wide range of viral and bacterial pathogens (Rosenberger and Cloud, 1989b; Cloud *et al.*, 1992).

Infections with CAV are considered to be economically significant because of the clinical disease associated with vertical transmission and because of its potential for inducing immune dysfunction alone on in combination with other pathogens. McNulty *et al.* (1988) reported that apparent subclinical infections of commercially produced broilers may result in increased mortality and condemnations.

CHICKEN ANEMIA VIRUS

The CAV is a small nonenveloped virus resistant to thermal inactivation and treatment with lipid solvents and many of the common disinfectants (von Bulow and Schat, 1997). The viral genome is a single-stranded DNA that is circular and covalently linked and consists of approximately 2,300 base pairs in its replicative form. The intact virion is icosahedral with an average diameter of approximately 25 nm and a buoyant density in cesium chloride of 1.33 to 1.34 g/mL (von Bulow and Schat, 1997). The physical and chemical characteristics of the virus are consistent with the circodna group.

DISEASE

Disease usually occurs during the first 3 wk of life resulting from vertical transmission or contact exposure close to hatch. Chickens at any age are susceptible to infection by the oral or respiratory routes, but do not show apparent signs of disease because of an age-associated resistance that is consistent with the ontogeny or maturation of the immune system. The period of susceptibility to disease may be extended by early exposure to infectious bursal disease virus, Marek's disease virus, or selected avian reoviruses that interfere with normal immune system development (Engstrom *et al.*, 1988; von Bulow and Schat, 1997).

Lesions of uncomplicated CAV infection in young chickens typically consist of bone marrow aplasia with reduction in hematocrit values (≤ 25%). Chickens are anorexic, depressed, and exhibit a marked pallor that may extend to the internal organs. Hemorrhages can be

Received for publication August 3, 1997.
Accepted for publication February 17, 1998.
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Abbreviation Key: CAV = Chicken Anemia Virus.

observed in the musculature and subcutaneously, with the wing tips frequently affected. The bone marrow is pale or yellow in color and may have a fatty consistency.

Thymic atrophy and congestion is common and is considered diagnostic when associated with other typical signs or lesions. Bursal atrophy is generally modest and transitory, typically occurring at 10 to 14 d of age in chickens vertically infected. All of the aforementioned lesions are exacerbated and more persistent in chickens coinfected with infectious bursal disease virus or other lymphocidal agents.

Severely affected birds generally die within 2 to 4 wk of age and survivors are often stunted. Because of the immunosuppression, affected chickens frequently develop secondary infections with *Clostridium perfringens* and *Staphylococcus aureus* in the subcutaneous tissues and musculature that results in losses due to gangrenous dermatitis. There may also be an increased susceptibility to adenovirus-associated inclusion body hepatitis and respiratory disease.

Microscopically, the bone marrow of CAV-infected chickens is depleted of erythrocytes, thrombocytes, and granulocytes and their precursor cells, which are replaced by adipose tissue. Thymic atrophy is the result of lymphocyte depletion in both the cortex and medulla. The bursa of Fabricius and spleen may also be depleted of lymphoid cells, but the involvement and duration is less extensive than seen with the thymus. Survivors of CAV infection usually return to normal by approximately 4 wk of age, which coincides with the onset of measurable antibody responses.

LABORATORY DIAGNOSIS

Affected flocks exhibiting pathognomonic signs and lesions are relatively easy to identify and a diagnosis can usually be made based on clinical presentation without the need for virus isolations or serological assessments. If confirmation is required, the virus can be isolated from buffy coats, liver, or splenic tissue collected during the clinical stage of the disease. The virus can be propagated in MDCC-MSB-1 cell cultures or susceptible immunosuppressed chickens. We believe that the best host system for initial isolations of CAV may be the chicken, as a number of recently characterized CAV isolates cannot be propagated in MDCC-MSB-1 cell cultures.

Antibody to CAV can be assessed by indirect immunofluorescence in infected MDCC-MSB-1 cells or by use of a commercially available ELISA kit. The PCR has also been used to identify CAV, but appropriate primers have not been prepared for all isolates and the test will not differentiate viable and inactivated virus.

PREVENTION AND CONTROL

Chicken Anemia Virus is generally considered to be ubiquitous in both egg and meat-type chickens worldwide. Because the virus is very resistant to inactivation and easily transmitted, it is probably unrealistic to assume that exposure can be limited with conventional housing and production parameters.

Infections of breeder flocks naturally or by the introduction of CAV prior to the onset of egg production will render them immune to subsequent exposure and prevent transovarian transmission. Based on serological evaluations, most flocks produced in the U.S. are infected prior to 12 wk of age. However, it is not uncommon, particularly in new production facilities, to find pullet flocks, that are serologically negative but that become infected with CAV during or just prior to the onset of production and shed virus to progeny. When this occurs, vertical transmission, on a flock basis, generally persists for 4 to 6 wk and ends when a majority of the hens seroconvert. Acquired immunity prevents vertical transmission for the life of the flock. It is well documented that immunosuppressive factors contribute to the enhanced susceptibility to CAV. We have demonstrated in our laboratory that day of age infection with infectious bursal disease virus can increase the susceptibility to CAV by as much as 100-fold. Coinfections as late as 2 wk of age can affect serological responses to common respiratory pathogens (Cloud *et al.*, 1992). Accordingly, it is essential that infectious bursal disease virus infections be controlled with breeder flock vaccination programs designed to provide progeny with maternal antibody derived protection during the first 2 to 3 wk posthatch. This approach is very effective in limiting the interactions between CAV and bursal disease. It is also important to control Marek's disease because CAV infections can exacerbate the effect of virulent strains of Marek's disease virus challenge in poorly immunized birds.

Less clear is the economic importance of horizontal or contact transmission of CAV that occurs in young broilers, which is uncomplicated by infectious bursal disease or other immunosuppressive agents. Based on limited studies done at the University of Delaware, it appears that hyperimmunization of breeders with inactivated CAV vaccines can result in better performance of progeny. Improved performance may be the result of longer and more consistent maternal antibody-mediated protection during the periods of greatest susceptibility, i.e., 1 d to 2 wk of age.

Although it is not feasible to totally prevent exposure to CAV, it is important to minimize its effects by proper sanitization and by optimizing the control of other factors that can contribute to an enhancement of the inherent immunosuppressive nature of CAV.

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