Chicken Anemia Agent (CAA)[1]

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In recent years, there has been much discussion on the significance of a newly identified disease of poultry, chicken anemia agent (CAA). Concerns have centered on this disease organism's effects on chickens' immune systems and on its relationship to other poultry diseases. This paper will describe the disease and its significance to poultry producers.

The CAA was first described in Japan in 1979. This agent has since been shown to be present in poultry flocks (layer and broiler breeds) worldwide. The CAA, a virus, is very small and rather resistant to chemical and physical treatment. For example, it can resist a pH of 3.0 and heat at 176°F for 30 minutes. These factors suggest the virus is difficult to eliminate on a poultry farm once the premises become contaminated. The virus has been classified as a parvo-like virus; however, it may instead represent a new group of poultry viruses.

The chicken appears to be the only known host for the CAA. That is, the virus does not infect humans or other animals. It can be transmitted both horizontally, from infected to susceptible birds, and vertically, through the eggs of seronegative, infected breeders to chicks. The CAA is shed through the egg until the breeder develops antibodies. The virus also can be transmitted mechanically from farm to farm by people, equipment and vehicles. The presence of the infection in some specific-pathogen-free flocks, used for vaccine production, may partially explain the worldwide incidence of the disease.

Chickens of all ages are susceptible to infection with the virus; however, only young chickens without maternal antibody protection develop the disease. Chickens younger than 2 weeks of age are particularly susceptible. Although chickens become increasingly resistant to the disease with age, they may still become infected and shed the virus at any age. Following infection, vertical transmission has been observed to occur for 14 days and horizontal transmission for 6 weeks.

CAA infection in young, susceptible chickens results in increased mortality of chickens 12 to 28 days of age. Affected birds generally have a depressed, pale (anemic) and anorexic appearance. In uncomplicated cases, surviving chicks recover from the depression and anemia; however, their body weights remain low. Lesions can lead to atrophy of the bone marrow and thymus. The bone marrow becomes fatty and yellowish. Atrophy and aplasia of the blood-forming tissues can be seen microscopically, as well as replacement by adipose tissue and nonfunctional supportive tissue. The thymus may completely atrophy and become reddish brown. Microscopically, severe lymphoid depletion is visible throughout the gland. Gross lesions in the bursa of Fabricius are minimal and may be difficult to detect. In many cases, the liver may be swollen and mottled, and massive hemorrhages may be observed in the proventricular mucosa and in subcutaneous tissue and muscle, particularly in the wing.

The extent of loss that will occur in an infected flock depends on the age at which the chickens are infected,

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the level of their maternal antibody protection against the CAA and whether they are concurrently infected with other immunosuppressive viruses, such as infectious bursal disease (IBD or Gumboro) virus or Marek's disease virus. Co-infection with the CAA and other immunosuppressive viruses increases the severity of disease due to the CAA, reduces the protective effect of maternal antibodies against the CAA and decreases age-related resistance to CAA. High levels of mortality associated with CAA infection are also commonly due to secondary bacterial infection. The term *blue wing* refers to a CAA infection in which initial skin lesions progress and become gangrenous because of secondary bacterial infection. Recent work has shown that CAA infection results in immunosuppression. Susceptible chicks have reduced ability to respond to vaccinations, resulting in diminished humoral and cell-mediated immune responses. The vertical transmission route has been associated with more severe immunosuppression. In addition, combined infections of CAA and Marek's disease virus result in an enhanced shedding of Marek's disease virus and an increased incidence of Marek's disease tumors.

No specific treatment is available for chickens affected by the CAA. The use of broad-spectrum antibiotics for treatment of secondary bacterialinfection may be considered. However, treatment has not been particularly effective in reducing loss.

Although the incidence of CAA infection in commercial poultry flocks in the Americas is not known, preliminary studies suggest infection is widespread. Elimination of the CAA from poultry flocks is probably not practical, since the disease organism is present in many poultry flocks throughout the Americas and is a very resistant virus. Experimentally, CAA vaccines have been successfully utilized in breeders to provide chicks with protection during the early weeks of life, when they are most susceptible. Chicks with maternal antibodies are refractory to infection and do not develop the disease. However, it is the author's opinion that CAA vaccination of breeders is probably not warranted at the present time. Recommendations to limit loss to CAA infection include improving breeder and broiler biosecurity practices and, since IBD enhances susceptibility to CAA, implementing an effective IBD disease control program.

CAA can cause economic loss due to mortality, decreased production and secondary infection resulting from immunosuppression. Producers need to be aware of this disease. If problems including mortality, anemia or dermatitis and other associated lesions are present in young chicks, laboratory investigation is warranted.