Vaccination of breeder flocks is essential for the effective control of CAV

by Haroldo Toro, DVM, PhD, 264 Greene Hall, Auburn University, Auburn AL 36849, USA.

Ubiquitous chicken anaemia virus (CAV) in commercial chickens determines financial losses from increased mortality and reduced performance during rearing, increased culling, lower uniformity at slaughter, and increased condemnations resulting from associated health problems. Control of CAV in commercial broilers and layers is largely based on generating immune responses in the breeders.

Antibody-positive breeders are not likely to transmit CAV to the offspring. In addition, maternal antibodies provide complete protection against CAV challenge in progeny chickens during the first 2-3 weeks of life.

Some operations rely on natural infection for the purpose of eliciting immune responses. However, this theoretically sound concept often produces suboptimal results in the field as a varying percent of antibody negative breeders persist and may become infected on or after lay onset.

Vertically infected progeny chickens and progeny chickens with suboptimal maternal immunity which become infected horizontally contribute to economic losses during production. Evidence supported by data obtained from vaccinated flocks indicates that vaccination against CAV significantly reduces the percent of antibody negative breeder hens and the risk of reduced performance and health of chicken progenies.

CAV in chickens

CAV causes aplastic anaemia and immunodeficiency due to destruction of T lymphocytes in young chickens. Vertical transmission has allowed the virus to spread throughout the world along with infected grandparent or breeder stocks. Further spread is through horizontal transmission to susceptible chickens.

CAV infection in poultry production results in financial losses from increased mortality and reduced performance during rearing, increased culling, lower uniformity at slaughter, and increased condemnations resulting from associated health problems.

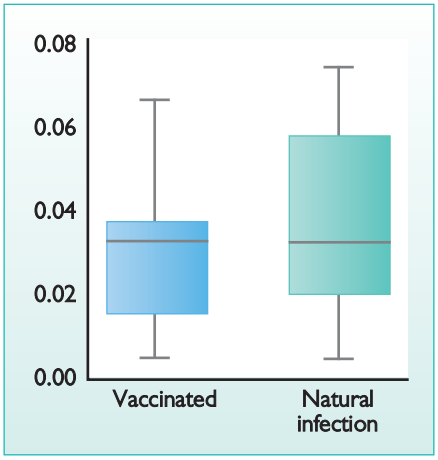


Fig. 1. Median and 25th percentile above and below of calculated standard error for ELISA antibody S/N values of Arkansas breeder flocks subjected to CAV natural infection (n=19) and Alabama grandparent flocks (n=4) subjected to CAV active vaccination.

In addition, CAV-induced immunodeficiency has been shown to predispose and/or aggravate other prevalent diseases of commercial poultry including, for example, infectious bronchitis, adenoviral hepatitis, Marek's disease, coccidiosis, infectious bursal disease, salmonellosis, and gangrenous dermatitis. Finally, CAV infection has also been associated with reduced vaccine effectiveness. Control of CAV is largely based on generating immune responses in breeder hens. Distinctively important is that vertical transmission of the virus is unlikely to occur from antibody-positive breeders. Furthermore, antibody-positive breeders effectively transfer antibody to progeny chickens which provide complete protection against CAV challenge during the first 2-3 weeks of life.

Strategies to induce resistance

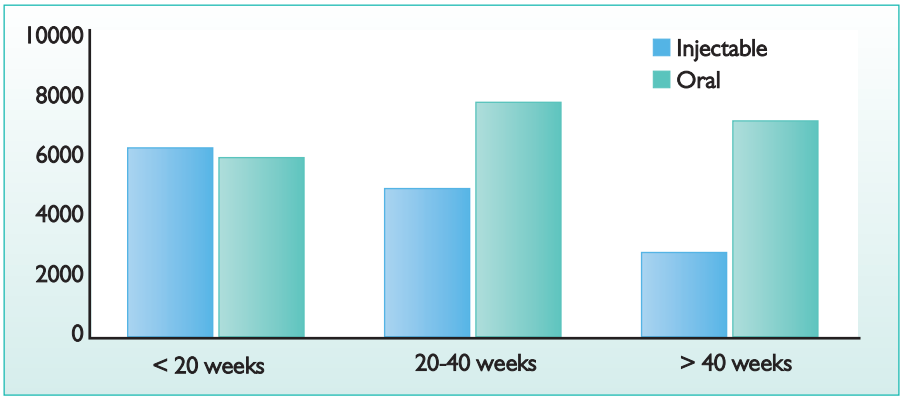
Induction of resistance against CAV may be elicited by natural infection and/or active vaccination. Many breeder operations rely on natural infection both in the United States and other countries. In the natural infection approach newly hatched breeders with maternally-derived antibodies become challenged by wild virus and active immunity subsequently develops. Indeed, natural infection results in flock average antibody titers considered protective.

However, the theoretically sound approach of natural infection produces varying results in the field. Indeed, even though the mean antibody titer of the flock may indicate protection, a varying percent of hens shows either low levels or even absence of antibodies.

The problem of uneven immunity in breeders resulting from natural infection likely results from variation in infective

Continued on page 13

Fig. 2. CAV serology profile of breeders flocks after oral vaccination with low attenuated or parenteral vaccination with highly attenuated CAV vaccines (De Oliveira et al, 2013).



Continued from page 11
pressure due to heterogeneous distribution and varying virus concentrations within and between chicken houses. Thus, even though the probability of most breeders becoming infected is high, a varying proportion will maintain an antibody negative status from not becoming exposed to the virus or from a suboptimal CAV infection dose. Breeder flocks with uneven immunity, also called 'stuttering flocks', result in a few vertically-infected progeny chickens, which in turn will horizontally infect birds with less than optimal antibody levels and reduce the average flock performance.

In addition, immunodeficient birds will allow increased replication of pathogens which would maintain marginal levels in immunocompetent flocks. In contrast, CAV vaccination provides a significant increase in immune coverage and reduced antibody variation within the flock. Statistical analyses of antibody values detected in breeders subjected to natural infection versus CAV active vaccination are shown in Fig. 1.

In this figure the calculated standard errors for antibody values of vaccinated and unvaccinated breeder flocks were plotted in a box and whiskers graph. As seen in the graph the medians (50th percentile) are similar between treatments; however flocks subjected to natural infection cluster in the upper 25th percentile (75th percentile), while values from vaccinated chicken cluster in the lower 25th percentile; less homogeneity is detected in chickens subjected to natural infection versus active vaccination.

Available CAV vaccines

The commercial vaccine repertoire is rather restricted and available live vaccines are derived only from a few CAV strains originally isolated in Europe or the United States. Even though some gene sequence polymorphism has been detected among CAV isolates from different regions of the world, no antigenically relevant changes seem to result from these differences so that immunity elicited by different vaccine strains is cross-protective against regional CAV isolates.

Commercially available live CAV vaccines differ in their level of attenuation. Highly attenuated vaccine viruses require parenteral delivery as they do not infect chickens via natural routes.

Thus, birds missing their dose during the vaccination process (for example vaccine dose ended on the plumage) maintained a susceptible status towards wild CAV. Low attenuated vaccine viruses can be delivered via natural routes (for example drinking water) and readily spread throughout the flock. Vaccine delivery via the oral route likely induces mucosal immunity which provides a relevant additional barrier to protect the host at the port of virus entry. Seroconversion becomes detectable a few days earlier after parenteral versus oral CAV exposure, presumably because the virus has to overcome innate immune responses before extensive establishment in the host.

The goal of CAV vaccination is to induce high and homogeneous antibody titers to last throughout the breeder's production life. Recent studies by Cardoso de Oliveira et al show that both low and highly attenuated CAV vaccines induce similar average flock antibody levels before 20 weeks of age. However, significant differences were detected after 20 weeks of age where oral vaccination with a low attenuated vaccine provided longer lasting immunity (Fig. 2). Longer lasting immunity likely results from low attenuated virus replicating more extensively in the chicken as well as from booster exposures from shed vaccine virus.

The existing evidence suggests that combining vaccination with accurate and constant monitoring of CAV antibody levels in breeders provides more homogeneous and effective protection against CAV in commercial chickens and reduces financial losses from suboptimal productive performance. ■

References are available from the author on request