

TECHNICAL BULLETIN



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Optimal Time for Gumboro Vaccination

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Infectious Bursal Disease (IBD or Gumboro) is a viral disease of young growing chickens occurring in most poultry producing areas of the world. It is caused by a virus, which is extremely stable and resistant to disinfectants and normal sanitation measures. Once established on a farm, the disease tends to recur with each succeeding flock.

The major organ affected by IBD is the cloacal bursa where the virus destroys the lymphocytes involved in antibody production and thus has a marked immunosuppressive effect. Clinical disease occurs in birds infected after three weeks of age and results in depression, diarrhea and variable mortality. Sub clinical infection occurs before three weeks of age and results in a severe immunosuppression without other obvious symptoms. However, later the flock may be more susceptible to a number of other secondary infectious diseases, such as gangrenous dermatitis, Marek's, lymphoid leukosis, Staph arthritis, or even coccidiosis. The mildest clinical effect may be only a difficulty achieving normal body weights. The sub clinical form is the most common problem with IBD. The clinical disease is seldom seen.

Variants

The virus, which causes IBD contains double-stranded RNA in segments. This arrangement allows a possible genetic recombination and alteration of antigenic structure. This can result in the production of new serotypes or variants of the original serotypes. These have received a lot of interest and study in the last two years. Currently

the standard vaccines and lab challenge viruses have been isolated, which can cause disease in birds already immune to the standard type I virus. Some of these are termed variants of serotype I and some are different enough to be classified as serotype II. The most virulent variants, designated A and E, have been found to cause an unusual pathology pattern. They cause no noticeable clinical disease and there is no swelling of the bursa. Rather, there is almost immediate atrophy of the bursa in the first two to three days and a strong effect on the thymus. Presence of variant IBD strains can only be proven by isolation and characterization of the virus by cross-neutralization studies. Both live and killed virus vaccines have been developed against these variants, but they should be used only where variants are known to exist.

The occurrence of IBD problems, in spite of a regular vaccination program, does not necessarily indicate the presence of variant strains. This paper deals with the critical importance of vaccination timing and involves only the standard serotype I vaccines. I believe we must first make sure we are doing as good a job as possible when vaccinating with the standard vaccines. Only then, if we continue having severe IBD problems, should we seriously consider the presence of a variant.

Since the disease is viral, treatment is of little value. Instead, we must rely on vaccination procedures to prevent disease susceptibility. Young chicks from day one to about three weeks of age are most conveniently protected by passive maternal antibodies acquired through the yolk. To provide a consistently high level of this maternal immunity, breeder flocks are normally injected

with a killed virus oil emulsion product at 18 weeks of age. The maternal immunity in the chick gradually declines with time to a point where the chick is susceptible to infection. In most cases, where field infection is expected, it is desirable to vaccinate with a live virus vaccine to stimulate active immunity and prevent the clinical effects of infection.

The difficulty with the live vaccination is to find the proper time for administration. If the vaccine is given too early, when the chicks still have high levels of maternal antibody, the vaccine virus will just be neutralized and no active immunity will develop for later protection. If the vaccine is given too late, virulent field virus may infect the flock before the vaccination immunity is established. In most cases, this dilemma creates a very narrow "window of opportunity" in which to successfully vaccinate a flock.

This points out the importance of cleaning and disinfecting growing houses. Virulent field viruses are probably able to overcome more parental immunity than most vaccine viruses. Therefore, if a house is heavily contaminated with a hot IBD virus, the vaccination program has no chance to immunize the flock before they are damaged by the field virus. Growing houses should be thoroughly cleaned and disinfected prior to housing new chicks to reduce this level of challenge as much as possible.

Another major factor to consider is the invasiveness of the vaccine virus and its capability to override maternal immunity. Some vaccines are quite invasive, but carry the disadvantage of causing bursal damage themselves in susceptible chicks. Other vaccines are very mild with regards to bursal damage, but are able to overcome only very low levels of maternal immunity. To utilize them, we must first allow the flock to become very susceptible, which risks a prior infection with a virulent field virus.

In recent years, the vaccine industry has developed the concept of "intermediate" vaccines which fine-tune the balance between these two extremes. The ideal is to have an invasive virus for the purpose of overriding some moderate level of maternal immunity, but one which will not by itself cause bursal damage in susceptible birds. In theory, this gives us more time to achieve a successful vaccination and narrows the period of

susceptibility to field virus infection.

Most vaccine companies now have "intermediate" strains of IBD vaccine. Although these products are advantageous, it still remains a challenge to find the proper time to vaccinate as soon as possible after the chicks can respond to vaccination and hopefully before a field virus infection has occurred.

Test Design

In an attempt to determine this proper age of vaccination, three trials utilizing three different intermediate vaccines have been performed at Hy-Line's technical service lab. In each trial a group of about 300 chicks was obtained from a hatch from a single breeder flock, which had received a commercial killed virus IBD injection. Groups of 15-20 chicks were randomly bled at hatch day and sequentially every three to four days thereafter, until 30 days of age for the purpose of determining the normal decline of IBD maternal antibodies by the ELISA method (Agri-Tech system). Starting at 10 days of age and four days thereafter, the chicks which were bled were retained and on the same day vaccinated orally with one dose of an intermediate strain IBD vaccine. These groups were then kept isolated in separate cages for a period of four weeks to allow for antibody development. At four weeks post-vaccination each group was re-bled to determine presence or absence of an antibody response.

Results

The results have shown that the maternal antibody declines in a smooth curvilinear manner with a half-life of about six days, which contrasts with the commonly accepted three systems. The rate of maternal antibody decline was not altered by daily exposure to live IBD vaccine between hatch and 18 days of age.

The earliest vaccinations which produced any serological response were those given at 18-22 days of age. Even then only 5-10% of the birds responded. Vaccinations given at 10-14 days produced no detectable serological response at all. A greater percentage of the chicks in the vaccinated groups responded when the vaccination was done at 26 and 30 days of age. Close to 100%

responded at 30 days.

The first responses to vaccinations at about 20 days of age coincided with average maternal antibody levels of about 1000. The majority of birds were responding when mean titers fell below 500. The individual birds which were responding generally had titers below 300 at the time of vaccination.

The duration of maternal antibody protection and age at which the population was becoming susceptible did not seem to be directly correlated to the initial hatch-day titers. Rather, the higher maternal titers declined more rapidly than the lower initial titers so that they all became susceptible at about the same time. This same trend has been reported by Solano, Giambrone and Panangala (*Avian Diseases*, Vol. 30, No. 1) and for this reason one standard IBD vaccination program should be adequate for most commercial pullet flocks.

The vaccine viruses apparently did not spread well from bird as evidenced by the lack of 100% seroconversion in cages where some individual birds had responded. For this reason, it is important to administer the vaccine precisely when

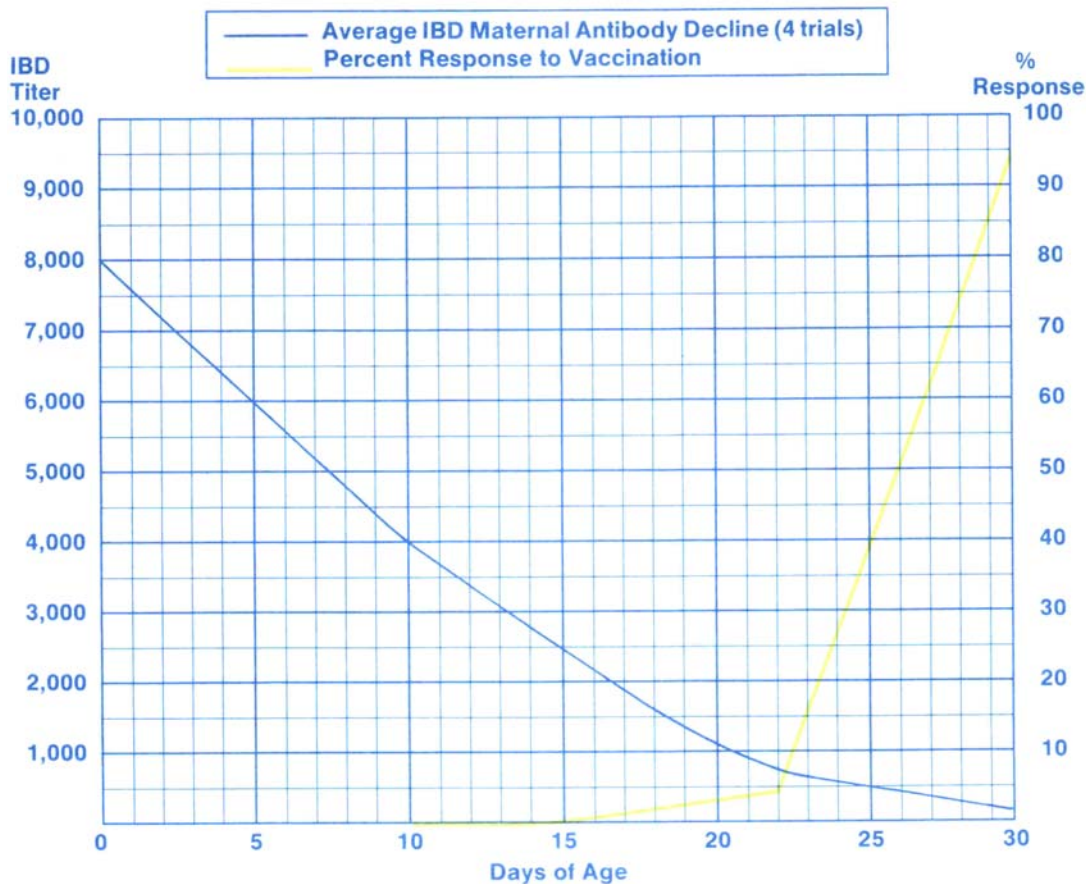
it can do the most good.

The intermediate strain vaccines used to date have seemed to perform equally well. It appears most of these products can be used in similar manners.

One vaccine was given as a subcutaneous injection at hatch day to simulate mixing IBD vaccine in Marek's diluent which is sometimes done commercially. These birds showed no titer response at five and one-half weeks of age.

Conclusion

Because not all chicks within a flock become susceptible to vaccination at the same age, Hy-Line's technical service department recommends two intermediate strain IBD vaccinations be given; one at 18-20 days of age, when the first birds are becoming susceptible, and the second at 28-30 days of age, when the majority of the birds will be able to respond. Effective control of IBD in this manner is a major step in growing pullets to achieve their genetic potential.





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