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Interference of Infectious Bursal Disease Virus on Antibody Production against Newcastle Disease and Infectious Bronchitis Virus

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ABSTRACT

This work has the objective of verifying the interference of infectious bursal disease virus in the antibody production against Newcastle disease virus and infectious bronchitis virus. The experiment was carried out with 640 day-old-chicks from a 42 weeks old hen flock. The birds were separated into eight experimental groups (n=80/group) and were submitted to different combinations of vaccinations, with live vaccines, to Newcastle disease, avian infectious bronchitis, and infectious bursal disease with diverse combinations of days of vaccination. We verified that the utilization of polyvalent vaccinal programs have a different efficacy comparing to monovalent vaccinations when Newcastle disease, infectious bronchitis, and infectious bursal disease vaccinations are applied. This way, the use of vaccinations to infectious bursal disease in polyvalent vaccinal programs is desirable due to improvement of NDV response with the presence of IBV by the probable reduction of interference of IBV under NDV.

INTRODUCTION

The infectious bursal disease (IBD) is a highly contagious viral and acute infection with tropism for lymphoid tissue, principally for bursa of Fabricius, in which the virus promotes the cell destruction. This disease is caused by IBDV that belongs to the genus Avibirnavirus of the family Birnaviridae (Murphy *et al.*, 1995) and was identified by the first time in Delmarva region of United States of America. It is a high importance problem for poultry industry. Current thinking is that protection against IBDV may be mediated primarily by anti-IBDV antibodies (Fussell, 1998; Lutticken, 1997; Vakharia *et al.*,1994). IBDV vaccines used in commercial flocks are selected by the ability of the vaccines to induce vigorous antibody responses (Lasher and Shane, 1994), this way it has been used live and inactivated vaccines from serotype 1 (Jackwood and Saif, 1987). Six variants of this serotype were identified by the virus neutralization test. This antigenic variation can induce failures on the vaccination processes due to the difference of antigenic structures between vaccinal and wild viruses (Cao *et al.*, 1998; Jackwood and Saif, 1987; Kibenge *et al.*,1988; van den Berg, 2000). Besides the antigenic variation, other factors can interfere on efficacy of a vaccinal program, among them, the viral interference. This phenomenon can occur among different serotypes of the same virus, for example infectious bursal disease virus (IBDV) with intermediate and pathogenic strains (Ashraf *et al.*, 2005), Reovirus (Whitaker-Dowling *et al.*, 1987), Poliovirus (Sabin, 1959) and Avian Influenza virus (Whitaker-Dowling, 1992). It can also occur between different viruses, as between infectious bronchitis virus (IBV) and Newcastle disease virus (NDV) (Cardoso *et al.*, 2005) or avian pneumovirus (Cook *et al.*, 2001). The occurrence of interference among

avian polyvalent vaccines can be promoted by the competition among the vaccinal viruses to the same receptors (Sabin, 1959).

This work has the objective of verifying the occurrence of interference of infectious bursal disease virus in the antibody production against Newcastle disease virus and infectious bronchitis virus.

MATERIAL AND METHODS

Birds

The experiment was carried out with 640 day-old-chicks from a 42 week-old hen flock. The chicks received no previous vaccination and were reared until 50 days of age in experimental broiler houses. The birds were bred simulating industrial conditions, with adequate management, water and feed ad libitum in a 10birds/m^2 density.

Treatments and vaccination

The birds were separated into eight experimental groups (n=80/group): Control Group to Maternal Antibodies (CG-Mab), Control Group to Avian Infectious Bronchitis (CG-AIB), Control Group to Newcastle disease (CG-ND), Control Group to Infectious Bursal Disease (CG-IBD), Group without IBD vaccination (T0), and the groups with one, two and three IBD vaccinal doses (T1), (T2), and (T3), respectively. All groups, except CG-Mab, were submitted to treatments with vaccinations. The treatments followed the methodology described in the Table 1.

The vaccination was performed by instillation of a 0.03 mL drop by ocular route. All vaccines belonged to the same laboratory and the same group of vaccinators did the vaccinations. The following vaccinal strains were used: HB1 ($10^{6.5}$) to Newcastle disease, H120 ($10^{3.5}$) to infectious bronchitis, and Lukert -intermediate classic ($10^{3.0}$) to infectious bursal disease.

Blood collection and serological tests

The control group to maternal antibodies was submitted to blood collections at 1st, 25th, 35th and 45th days of age to verification of maternal antibody levels to IBV, NDV, and IBDV. The other groups were submitted to blood collections at 35th and 45th days of age. All blood samples were obtained from the brachial vein and the sera were collected, maintained adequately refrigerated to serological tests. Each serum was identified according to the number of the birds.

The serum samples were analyzed by HI (Haemagglutination inhibition) test to detection of antibodies against NDV and indirect ELISA (enzyme-linked immunosorbent assay) (Kirkegaard & Perry Laboratories - KPL) to detection of antibodies against IBV and IBDV.

Statistical Analyses

The titers obtained by ELISA and HI tests were submitted to the statistic program SAS/STAT 95 from SAS Institute Inc. (User's guide: statistic and graphics, 1995). The results of antibody titers were submitted to Variance Analyses. The antibody titers were transformed by the logarithmic function $Log_{10}x+1$ to submit the data to variance analyses. The means were compared through the test 't' student with significance level of 5%.

RESULTS

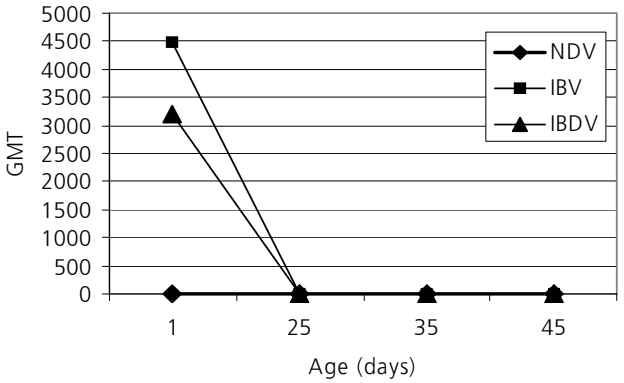
The Graphic 1 presents the maternal antibody titers of control group against Newcastle disease virus, infectious bursae disease virus, and infectious bronchitis virus.

The maternal antibodies to NDV were null since the beginning up to the end of the experiment. The titers against IBDV and IBV were significant. The first one has GMT 3200 by the first day decreasing to zero at 25 days of age and maintained this level until the end of experiment. The maternal antibody curve to IBV was similar to IBDV, but the titers in the first day were around GMT 4500.

Table 1 - Experimental treatments.

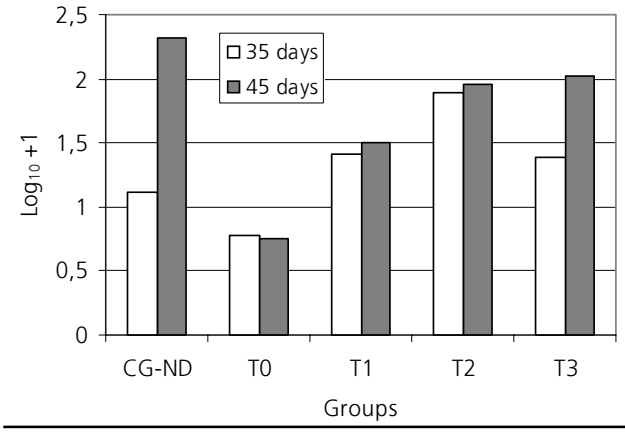
Groups	Vaccinations		
	1st day	8th day	16th day
CG-Mab	N/V	N/V	N/V
CG-IB	N/V	H_{120}	N/V
CG-ND	N/V	HB1	N/V
CG-IBD	Lukert-intermediate classic	Lukert-intermediate classic	N/V
T0	N/V	H_{120} + HB1	N/V
T1	Lukert-intermediate classic	H_{120} + HB1	N/V
T2	Lukert-intermediate classic	H_{120} + HB1	Lukert-intermediate classic
T3	Lukert-intermediate classic	H_{120} + HB1 + Lukert-intermediate classic	Lukert-intermediate classic

N/V= no vaccination.



Graphic 1 - Maternal antibody titers of control group against Newcastle disease virus, infectious bronchitis virus, and infectious bursal disease virus.

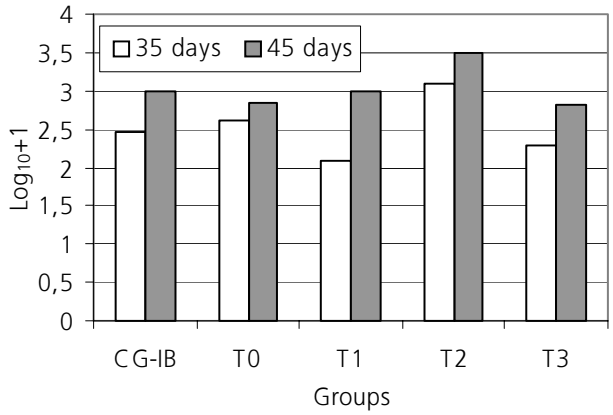
The Graphic 2 presents antibody titers (Log_{10} +1) to NDV at 35 and 45 days of age to the groups that were submitted to vaccinations to Newcastle disease, infectious bronchitis, and one, two or three vaccinations to infectious bursal disease.



Graphic 2 - Antibody titers to Newcastle disease virus of experimental groups.

The birds of control group to Newcastle disease showed the antibody titer (2,318) at 45 days, being statistically superior to T0 antibody titer (0,753) at 45 days. We can observe the antibody titer increasing with the addition of infectious bursal disease vaccinations, one, two or three vaccinations. At 45 days, the anti-NDV antibodies of T2 and T3 were statistically superior to T1 that received only one IBDV vaccination.

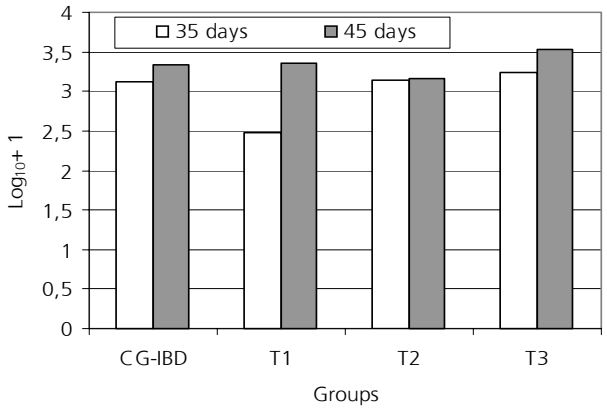
The Graphic 3 presents antibody titers (Log_{10} +1) to IBV at 35 and 45 days of age to the groups that were submitted to vaccinations to Newcastle disease, infectious bronchitis, and one, two or three vaccinations to infectious bursal disease.



Graphic 3 - Antibody titers to Infectious Bronchitis virus of experimental groups.

The Graphic 3 shows that the antibody titers to IBV were similar for most of the groups at 35 and 45 days of age, however T3 at 45 days and T2 at 35 days presented statistic difference ($p<0.05$) when compared to the other groups in the same age, being the lowest level at 45 days and the highest at 35 days, respectively.

The Graphic 4 presents antibody titers (Log_{10} +1) to IBDV at 35 and 45 days of age to the groups that were submitted to vaccinations to Newcastle disease, avian infectious bronchitis, and one, two or three vaccinations to infectious bursal disease.



Graphic 4 - Antibody titers against infectious bursal disease virus of experimental groups.

We can observe that the graphic 4 shows that IBDV antibody titers presented no significant difference ($p<0.05$) when the birds received one, two or three IBD vaccinations.

DISCUSSION

The maternal antibodies to infectious bursal disease in day-old-chicks of CG-Mab group belonging from IBD

immunized breeders were elevated (GMT 3200) according to Sharma *et al.* (1989), and decreased to zero by 25 days of age, agreeing with Al-Natour et al. (2004) that found similar results, with maternal antibody titers around GMT 3400 at the first day with a decrease reaching zero around 28 days of age.

This expressive antibody titer drop has been explained by many authors (Knezevic *et al.*, 1999; Knoblich *et al.*, 2000; Alam *et al.*, 2002) that concluded that vaccination does not accelerate decrease in maternal antibodies if chicks are vaccinated at one day of age. Similarly to results reported by Knezevic *et al.* (1999), chicks with passive immunity and vaccinated with an intermediate IBDV strain in the first day of age showed no increase in antibody titers.

In the same way, IBV antibodies decreased to zero around 25 days, like the results observed by Gelb *et al.* (1998).The NDV antibodies were null since the first day up to the end of experiment. The maternal antibody curves to IBDV, NDV, and IBV indicated that, to these three analyzed diseases, there was no interference of wild or vaccinal virus challenges. The relation between the expressive IBDV maternal antibody decrease found in our experiment with absence of interference of wild or vaccinal virus challenges can explained by inexistence of wild or vaccinal virus challenges or neutralization of the virus by the high IBDV maternal antibodies as demonstrated by Moraes *et al.* (2005) that verified that vaccinated and unvaccinated chicks with high antibody titers (3.4Log10) in the first day of age were protected against the disease after challenge with a very virulent strain of IBD.

We observed that the group of birds that was submitted for just a vaccination to AIB and ND at eighth day presented NDV antibodies inferior to the group that received only a ND vaccination in the same day. Many researchers reported this situation (Beard, 1967; Raggi and Lee, 1963; Raggi and Pignattelli, 1975; Yachida *et al.*, 1986). According to Gelb *et al.*, (2004), the interference between IBV and NDV occur due to the initial infection tropism that is epithelial cells of respiratory tract in which the viruses replicate in the cytoplasm. Studies carried out by Montgomery *et al.* (1997) in which many vaccinations to AIB and ND were administered, combined or not, verified that IBV induced a decrease in the capacity of immune response of gland of Harder (GH). The reduced capacity of response can decrease the level of antibody response to NDV when the birds are vaccinated to IBV and NDV in the same time. Dohms *et al.* (1988) verified a similar situation to IBDV, showing that there is a decrease of plasma cell quantity in the gland of Harder during infectious bursal disease virus infection of 3-week-old broiler chickens that might induce deficiency of local immunity in the paraocular region and upper respiratory tract associated with IBD.

However the groups that did not received only the vaccination to ND and AIB (T1, T2, T3 groups) but also additional IBD vaccinations did not presented reduced NDV antibodies as the T0 group. There was a progressively increase of NDV antibody titers according to the addition of IBD vaccinations, that were one, two or three, respectively T1, T2 and T3, with the NDV antibody response to T2 and T3 being statistically superior to T1. These results show a possible positive interference that the use of live vaccine of IBD promotes on NDV antibody response. The progressive increase of antibody titers of T1, T2, and T3 groups in comparison to T0 was not enough to be superior to the antibody response of the birds that received only ND vaccination (CG-ND). These findings suggest that polyvalent vaccination with IBDV do not improve the NDV response, but decrease, modify or interfere with interference of IBV under NDV. This way the live IBDV vaccine induces an NDV antibody response superior to the birds that receive only ND and AIB vaccinations, without IBDV vaccine.

Regarding the IBV and IBDV antibody titers, it wasn't observed significant ($p<0.05$) variation among the groups. That indicates the low interference of NDV under IBV, as observed by Raggi; Lee, (1964) e Zygraich *et al.*, (1973). In the same way, it wasn't observed interference of IBV and NDV under IBDV, because IBDV antibodies were similar independent of the group or experimental vaccination program administered.

CONCLUSION

We can conclude that the utilization of polyvalent vaccinal programs have a different efficacy comparing to monovalent vaccinations when Newcastle disease, avian infectious bronchitis, and infectious bursal disease vaccinations are applied. This way, the use of vaccinations to infectious bursal disease in polyvalent vaccinal programs is desirable due to improvement of NDV response, in spite of the presence of IBV by the probable reduction of interference of IBV under NDV.

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