Passive Immunity Against Infectious Bursal Disease More Important Than Ever

Dr. Guillermo Zavala, DVM, MAM, MSc, PhD, Dipl. ACPV

Infectious bursal disease (IBD) is associated with significant immunosuppression, increased mortality, and poor economic performance[1,2]. The disease is caused by infectious bursal disease virus (IBDV), an agent that is resistant to disinfectants and environmental conditions and is therefore difficult to control by biosecurity, cleaning, and disinfection procedures. Some of the best tools to prevent and control IBD are vaccines of various types. Broiler breeder flocks are primed in the rearing phase, followed by inactivated oil-emulsified vaccines (EOV) administered a few weeks before the production phase. The purpose of early live vaccinations in breeders is to protect them against clinical IBD, whereas the objective of using one or more EOVs is to achieve antibody titers that are as high and uniform as possible to protect the progeny.

Some of the first vaccination strategies against IBD involved field strains that were only partially attenuated and therefore posed significant risks for susceptible chickens[3]. The potential role of maternal antibodies to protect young chickens became known long before the first inactivated vaccines were developed and used extensively by the poultry industry[4]. Throughout the last 40 years, the benefits of immunizing breeders with a combination of live and killed vaccines have been well established in both research and field settings[5-10].

Early studies demonstrated that inactivated oil-emulsified vaccines against IBDV could induce up to 100% protection against IBDV certain isolates beginning at 5 weeks post-vaccination in vaccinated chickens. This knowledge was adapted for use in breeders resulting in a number of studies demonstrating the power of immunization using killed vaccines for breeders to achieve high and uniform levels of antibodies transferrable to the progeny as effective passive immunity[11-14]. This practice has proven to be economically advantageous for the broiler progeny from breeders vaccinated with OEVs[15-17].

The advent of very virulent IBDV strains in various geographic regions during the 1980s prompted industry at some point to consider not hyper-immunizing breeder hens such that the progeny could be vaccinated successfully at a young age without the interference of high maternal antibodies[18,19]. This strategy has proven unrealistic and unsuccessful because the breeders themselves must be protected from IBDV and because sometimes there may be a proportion of broiler progeny with low or no protection.

As a bi-segmented double-stranded RNA virus, IBDV has tended to accumulate mutations and genome reassortments resulting in viruses that are not the same as the viruses that circulated years ago[20-27], a trend that will continue indefinitely due to the nature of IBDV. Because it is not possible to produce a novel vaccine with new strains continuously, a reasonable strategy is to vaccinate the breeders with live and killed vaccines with the intention of rising their antibody levels as high as possible. It has been shown that it is possible to control clinical infection in the progeny even when the challenge virus is non-homologous, provided the virus neutralization antibody titers are high enough[18]. High maternal antibodies can prevent clinical infection and the consequences caused IBDV; and even higher maternal antibodies may also prevent or minimize significant bursal damage upon challenge with virulent viruses[18].

It is important that killed vaccines contain a high antigenic mass and that there is antigenic diversity represented in the vaccine, which depends on the inclusion of various vaccine strains intended to broaden the antigenic spectrum and protection provided by the vaccine. Two critical factors should be kept in mind when attempting to generate the best possible immunity in the breeders to be transferred to the progeny: a) antibody quantity; and b) antibody quality. The quantity of antibodies is represented by the antibody titers, which should be as high and uniform as possible. The quality of antibodies is influenced by the antigenic diversity of the vaccine. It is therefore critical to prime the breeders with live vaccines and to hyper-immunize them with killed vaccines containing a high antigenic mass and such mass must be antigenically diverse.

References:

1. Christensen NH. The cost to the meat chicken industry of the introduction of Infectious Bursal Disease to New Zealand. *N Z Vet J*. 33:191-3; 1985 Nov. **2.** Lasher HN, Davis VS. History of infectious bursal disease in the U.S.A.--the first two decades. *Avian Dis*. 41:11-9; 1997 Jan-Mar. **3.** Edgar SA, Cho Y. Immunization of chickens for the control of infectious bursal disease. *Poult Sci*. 52:492-7; 1973 Mar. **4.** Wyeth PJ, Cullen GA. Maternally derived antibody--effect on susceptibility of chicks to infectious bursal disease. *Avian Pathol*. 5:253-60; 1976. **5.** Ide PR, Schulte-Nordholt JA, Dewitt WF, Smith JD. Broiler-breeder vaccination against infectious bursal disease and persistence of maternal antibody in progeny. *Can Vet J*. 19:123-7; 1978 May. **6.** Lucio B, Hitchner SB. Response of susceptible versus immune chicks to killed, live-modified, and wild infectious bursal disease virus vaccines. *Avian Dis*. 23:1037-50; 1979 Oct-Dec. **7.** Wyeth PJ, Cullen GA. The use of an inactivated infectious bursal disease oil emulsion vaccine in commercial broiler parent chickens. *Vet Rec*. 104:188-93; 1979 Mar 3. **8.** Wood GW, Muskett JC, Thornton DH. The interaction of live vaccine and maternal antibody in protection against infectious bursal disease. *Avian Pathol*. 10:365-73; 1981 Jul. **9.** Wyeth PJ, Gough RE, Cullen GA. Immune responses of breeding chickens to trivalent oil emulsion vaccines: responses to Newcastle disease and infectious bursal disease. *Vet Rec*. 108:72-5; 1981 Jan 24. **10.** Voeten AC, Lutticken D, van Dijk PM, Bergs GH, Orthel FW. The use in practice of inactivated oil emulsion vaccine against infectious bursal disease in broiler breeders and its influence on the progeny: a comparative field trial. *Vet Q*. 7:91-100; 1985 Apr. **11.** Thayer SG, Eidson CS, Kleven SH. Multivalent inactivated virus oil emulsion vaccines in broiler breeder chickens. III. Trivalent Newcastle disease, infectious bursal disease, and arthritis/tenosynovitis viruses vaccine in primed breeders. *Poult Sci*. 62:1991-7; 1983 Oct. **12.** Thayer SG, Eidson CS, Kleven SH. Multivalent inactivated virus oil emulsion vaccines in broiler breeder chickens. II. Trivalent vaccines in breeders not previously vaccinated with live Newcastle disease, infectious bursal disease, and tenosynovitis vaccines. *Poult Sci*. 62:1984-90; 1983 Oct. **13.** Thayer SG, Eidson CS, Kleven SH. Multivalent inactivated virus oil emulsion vaccines in broiler breeder chickens. I. Newcastle disease virus and infectious bursal disease virus bivalent vaccines. *Poult Sci*. 62:1978-83; 1983 Oct. **14.** Maas RA, Venema S, Oei HL, Pol JM, Claassen IJ, Huurne AA. Efficacy of inactivated infectious bursal disease (IBD) vaccines: comparison of serology with protection of progeny chickens against IBD virus strains of varying virulence. *Avian Pathol*. 30:345-54; 2001 Aug. **15.** Wyeth PJ, O'Brien JD, Cullen GA. Improved performance of progeny of broiler parent chickens vaccinated with infectious bursal disease oil-emulsion vaccine. *Avian Dis*. 25:228-41; 1981 Jan-Mar. **16.** Giambrone JJ, Yu M, Eckman MK. Field trials with an oil emulsion infectious bursal disease vaccine in broiler breeder pullets. *Poult Sci*. 61:1823-7; 1982 Sep. **17.** Wood GW, Muskett JC, Thornton DH. Use of inactivated oil emulsion infectious bursal disease vaccines in breeder chickens to prevent immunosuppression in progeny chicks. *Res Vet Sci*. 35:114-5; 1983 Jul. **18.** Berg TP. Acute infectious bursal disease in poultry: a review. *Avian Pathol*. 29:175-94; 2000 Jun. **19.** Dey S, Pathak DC, Ramamurthy N, Maity HK, Chellappa MM. Infectious bursal disease virus in chickens: prevalence, impact, and management strategies. *Vet Med (Auckl)*. 10:85-97; 2019. **20.** Michel LO, Jackwood DJ. Classification of infectious bursal disease virus into genogroups. Arch Virol. 162:3661-3670; 2017 Dec. **21.** Wang Y, Jiang N, Fan L, Niu X, Zhang W, Huang M, Gao L, Li K, Gao Y, Liu C, *et al*. Identification and Pathogenicity Evaluation of a Novel Reassortant Infectious Bursal Disease Virus (Genotype A2dB3). Viruses. 13: 2021 Aug 25. **22.** Islam MR, Nooruzzaman M, Rahman T, Mumu TT, Rahman MM, Chowdhury EH, Eterradossi N, Muller H. A unified genotypic classification of infectious bursal disease virus based on both genome segments. *Avian Pathol*. 50:190-206; 2021 Apr. **23.** Feng X, Zhu N, Cui Y, Hou L, Zhou J, Qiu Y, Yang X, Liu C, Wang D, Guo J, *et al*. Characterization and pathogenicity of a naturally reassortant and recombinant infectious bursal disease virus in China. *Transbound Emerg Dis*. 2021 Oct 16. **24.** Tomas G, Marandino A, Techera C, Olivera V, Perbolianachis P, Fuques E, Grecco S, Hernandez M, Hernandez D, Calleros L, *et al*. Origin and global spreading of an ancestral lineage of the infectious bursal disease virus. *Transbound Emerg Dis*. 67:1198-1212; 2020 May. **25.** Fan L, Wu T, Wang Y, Hussain A, Jiang N, Gao L, Li K, Gao Y, Liu C, Cui H, *et al*. Novel variants of infectious bursal disease virus can severely damage the bursa of fabricius of immunized chickens. *Vet Microbiol*. 240:108507; 2020 Jan. **26.** Tomas G, Marandino A, Courtillon C, Amelot M, Keita A, Pikula A, Hernandez M, Hernandez D, Vagnozzi A, Panzera Y, *et al*. Antigenicity, pathogenicity and immunosuppressive effect caused by a South American isolate of infectious bursal disease virus belonging to the "distinct" genetic lineage. *Avian Pathol*. 48:245-254; 2019 Jun. **27.** Jackwood DJ, Schat KA, Michel LO, de Wit S. A proposed nomenclature for infectious bursal disease virus isolates. *Avian Pathol*. 47:576-584; 2018 Dec.

MAYBE.

Don't lose sight of subclinical IBD.

MAYBE NOT.