References

ALKAHALAF, A. N., HALVORSON D. A. and SAIF Y. M. (2002): Comparison of Enzyme-Linked Immunosorbent Assays and Virus Neutralization test for detection of antibodies to avian pneumovirus. *Avian Disease*. 46, 700-703.

AUNG, Y. H. (2007). Comparison of the pathogenesis and immune responses following avian Metapneumovirus subtype A and B infection in broiler-type chickens. INAUGURAL DISSERTATION zur Erlangung des Grades eines. *DOKTORS DER VETERINÄRMEDIZIN durch die Tierärztliche Hochschule Hannover*

BUYS, S. B. and DU PREEZ J. H. (1980): A preliminary report on the isolation of a virus causing sinusitis in turkeys in South Africa and attempts to attenuate the virus.Turkeys. 28, 36.

BUYS, S.B., DU PREEZ, J.H. and ELS, H.J. (1989) Swollen Head Syndrome in Chickens. A preliminary report on the isolation of a possible aetiological agent. *J. S. Afric. Vet. Assoc., 60:221-222*

COLLINS, M. S., GOUGH R. E. and ALEXANDER D. J. (1993): Antigenic differentiation of avian pneumovirus isolates using polyclonal antisera and mouse monoclonal antibodies. *Avian Pathology*. 22, 469-479.

COOK, J. K. A. (2000): Avian Rhinotracheitis. *Rev. Sci. Tech. Off. Int. Epiz. 19 (2),* 602-613

COOK, J. K. A., ELLIS M. M. and HUGGINS M. B. (1991): The pathogenesis of turkey rhinotracheitis virus in turkey poults inoculated with the virus alone or together with two strains of bacteria. *Avian Pathology*. 20, 155-166.

COOK, J. K. A., HOLMES H. C., DOLBY C. A., FINNEY P. M., ELLIS M. M. and HUGGINS M. B. (1989): A live attenuated turkey rhinotracheitis virus vaccine: 2. The use of the attenuated strain as an experimental vaccine. *Avian Pathology*. 18, 523-534.

COOK, J. K. A., JONES B. V., ELLIS M. M., JING L. and CAVANAGH D. (1993): Antigenic differentiation of strains of turkey rhinotracheitis virus using monoclonal antibodies. *Avian Pathology*. 22, 257-273.

GOUGH, R.E. (2003): Avian pneumoviruses. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE (eds): *Diseases of Poultry. 11th ed. Iowa State Press*, Iowa. 93–99.

HAFEZ, H. M. (1993): The role of pneumovirus in swollen head syndrome of chickens. Dtsch. Tierärztl. Wschr. 99, 486-488.

HAFEZ, H. M., and LÖHREN U. (1990): Swollen head syndrome: clinical observations and serological examination in West Germany. *Dtsch. Tierärzt. Wschr.* 97, 322-324.

JONES, R. C., BAXTER-JONES C., WILDING G. P. and KELLY D. F. (1986): Demonstration of a candidate virus for turkey rhinotracheitis in experimentally inoculated turkeys. *Vet. Rec.* 119, 599-600.

JONES, R. C., NAYLOR C. J., AL-AFALEQ A., WORTHINGTON K. J. and JONES R. (1992): Effect of cyclophosphamide immunosuppression on the immunity of turkeys to viral rhinotracheitis. *Res. Vet.* Sci. 53, 38-41.

JUHASZ, K. and EASTON A. J. (1994): Extensive sequence variation in the attachment (G) protein gene of avian pneumovirus: evidence for two distinct subgroups. *J. Gen. Virol.* 75, 2873-2880.

MARIEN, M., DECOSTERE, A., MARTEL, A., CHIERS, K., FROYMAN, R. and NAUWYNCK, H. (2005). Synergy between avian pneumovirus and *Ornithobacterium rhinotracheale* in turkeys. *Avian Pathology*, 34(3): 204–211.

O'BRIEN, J. D. P. (1985): Swollen head syndrome in broiler breeder. *Vet. Rec.* 117, 619-620.

NAYLOR C. J., ALANKARI A. R., ALAFALEQ A. I., BRADBURY J. M., JONES R. C. (1992) Exacerbation of Mycoplasma gallisepticum infection in turkeys by rhinotracheitis virus. *Avian Pathology*, 21, 295–305.

OWOADE, A. A., DUCATEZ M. F. and MULLER C. P. (2006): Seroprevalence of avian influenza virus, infectious bronchitis virus, reo virus, avian pneumovirus, infectious laryngotracheitis virus, and avian leukosis virus in Nigerian poultry. *Avian Disease*. 50, 222-227.

PANIGRAHY, J-P. SENNE D. A., PEDERSEN J. C., GIDELWSKI T.. EDSON R.K. (2000): Experimental and serologic observations on avian pneumovirus (APV/turkey/Colorado/97) infection in turkeys. *Avian Disease*. 44; 17-22.

PICAULT, J. P., GIRAUD P., DROUIN P., GUITTET M., BENNEJEAN G., LAMANDE J., TOQUIN D. and GUEGUEN C. (1987): Isolation of a TRTV-like virus from chickens with swollen head syndrome. Vet. Rec. 121, 135.

VAN DE ZANDE, S., NAUWYNCK, H. and PENSAERT, M. (2001). The clinical, pathological and microbiological outcome of an Escherichia coli O2:K1 infection in avian pneumovirus infection in turkeys. *Veterinary Microbiology*, 81: 353–365.

VAN LOOCK, M.; LOOTS, K.; VAN DE ZANDE, S.;VAN HEERDEN, M.; NAUWYNCK, H.; GODDEERIS, B.M. and VANROMPAY, D. (2006). Pathogenic interactions between Chlamydophila psittaci and avian pneumovirus infections in turkeys. *Veterinary Microbiology* 112 (2006) 53–63.

WILLIAMS, R. A., SAVAGE C. E., WORTHINGTON K. J. and JONES R. C. (1991): Further study on the development of a live attenuated vaccine against turkey rhinotracheitis. *Avian Pathology*. 20, 585-596.

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Area Pneumovirus

EXPERIENCE OF THE USE OF HIPRAVIAR® SHS IN BROILER CHICKENS

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1 INTRODUCTION

Avian pneumovirus or avian metapneumovirus (aMPV) is a negative-sense single-stranded RNA virus, and a member of the subfamily *Pneumovirinae* of the family *Paramyxoviridae* (Gough, 2003).

aMPV causes an infection in the respiratory tract of chickens and turkeys of any age (Hafez 1993; Cook 2000), resulting in the appearance of clinical symptoms, early in turkeys, and based on various factors in chickens (field pressure, bad management, lack of biosecurity, health problems...).

The aMPV was isolated for the first time in turkeys in 1978 in South Africa (Buys & Du Preez 1980). Also at the end of the 70s, were first reported in South Africa cases of SHS (Swollen Head Syndrome) in chickens (Buys et al. 1989), while the first cases of Turkey rhinotracheitis were being reported (Buys & Du Preez 1980). However it was not until 1987, when Picault et al. isolated aMPV in an outbreak of swollen head syndrome in chickens for the first time, becoming a disease in constant expansion, reporting cases worldwide.

Only a single serotype has been identified to date, although four subtypes have been differentiated by the analysis of the nucleotide sequence of the (G) attachment glycoprotein (Juhasz & Easton, 1994) and neutralisation tests with monoclonal antibodies (Collins et al., 1993; Cook et al., 1993).

Transmission is horizontal by direct or indirect contact (Jones et al. 1986; Cook et al. 1991; Panigrahy et al. 2000; Alkhalaf et al., 2002). This is why the seroprevalence in chickens and breeders is high, although in chickens is not always accompanied by clinical symptoms (O'Brien 1985; Hafez and Lohren 1990; Owoade et al. 2006).

Clinical signs in chickens are characterized by respiratory symptoms, between 20 and 35 days old, usually limited to the upper respiratory tract (trachea and nasal turbinates). These symptoms can be characterized by sneezing, coughing, nasal discharge, conjunctivitis and edematous sinus. The infection caused by aMPV favours the establishment and manifestation of secondary respiratory infections in broiler chickens and turkeys, as has been demonstrated with various respiratory pathogens (Naylor et al., 1992; Van de Zande et al., 2001; Marien et al., 2005; Van Loock et al., 2006).

Thus, the classical clinical picture can be seen complicated by secondary bacterial infections, usually, E. coli, O. rhinotracheale ... etc, assuming a worsening of the symptoms and a large economic loss due to the increase of the cost of the treatment and worsening production rates (FCR, MORTALITY, MEDIUM WEIGHT).

The control of the disease has been carried out through various strategies, as it is a disease of multifactorial character in broiler chickens. Nevertheless, in certain situations, the pressure of aMPV in the field makes vaccination necessary.

This study was conducted to assess the efficacy of a live attenuated vaccine against aMPV, subtype B, chicken origin, strain 1062 (HIPRAVIAR® SHS), in a Brazilian broiler integration with respiratory problems diagnosed as Swollen head syndrome or avian pneumovirus by the integration's veterinary services.

2 DESCRIPTION OF THE PROBLEM

The problem showed itself as a respiratory disease that affected the upper respiratory tract between 28 and 35 days of age, with bacterial complications and weakening and mortality associated with the secondary bacterial infection.

Antibiotic treatments were performed, with a good efficacy during treatment, but with rapid recurrence of bacterial infection after treatment.

The groups were designated Before Vaccination (BV), mean of the two batches before instituting vaccination with HIPRAVIAR® SHS, and During Vaccination (DV), DV1 and DV2, two batches that were vaccinated as indicated in Table 1.

Tabla 1:

	Vaccination programme Before Vaccination (WITHOUT HIPRAVIAR® SHS)		Vaccination programme During Vaccination (WITH HIPRAVIAR® SHS)	
Day	Vaccine	Administration route	Vaccine	Administration route
0	IBV H120	Spray	IBV H120	Spray
1			**Hipraviar® SHS**	**Spray**
7	IBDV Intermediate strain	Drinking water	IBDV Intermediate strain	Drinking water
14	IBDV Intermediate strain +	Drinking water	IBDV Intermediate strain +	Drinking water

3 RESULTS

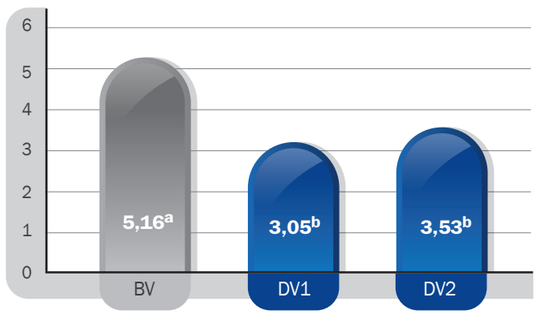
The respiratory problems and the whole symptomatology described above disappeared after the start of vaccination, and the zootechnical results showed an improvement in all areas. The whole comparative study was carried out on the same farm, in successive batches, with a total length of 8 months (4 batches, 2 without vaccination against aMPV and 2 vaccinated). The farm comprises of six shed, with a capacity of 240,000 birds per cycle. A total of 917,000 birds were evaluated.

The values with distinct superscripts show significant differences at $P \leq 0.05$ by unidirectional analysis of variance (ANOVA), while values with the same superscript do not show significant differences $P > 0.05$.

Mortality

The mean mortality of all sheds in each batch was compared, the data Before Vaccination, is the mean of the two previous batches (BV1 6.23% and BV2 4.08%) giving a 5.15% mortality, while the mortality in the vaccinated batches was 3.05% for DV1 and 3.53% for DV2. It resulted to a 36% decrease in mortality between the mean of the unvaccinated batches and the vaccinated batches.

Mortality



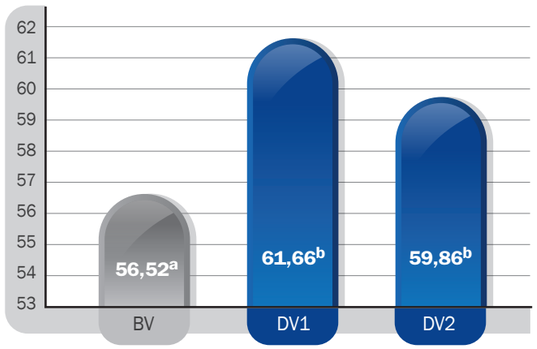
$P_{BV\text{-}DV1}$=0,00043 $P_{BV\text{-}DV2}$=0,0058

The values with distinct superscripts are significantly different at $P \leq 0.05$ by unidirectional analysis of variance (ANOVA).

Average Daily Gain (ADG)

The ADG of the unvaccinated batches (57.21 and 55.09 g/day) showed a significant difference with the vaccinated batches: DV1 61.66 g/day and DV2 59.86 g/day, assuming an increase of 5.14 g / day and 3.34 g / day respectively.

ADG



$P_{BV\text{-}DV1}$=0,0016 $P_{BV\text{-}DV2}$=0,0042

The values with distinct superscripts are significantly different at $P \leq 0.05$ by unidirectional analysis of variance (ANOVA).

Feed conversion rate

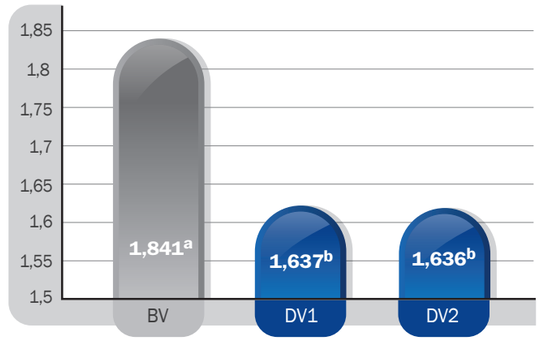
The feed conversion rate was standardised at 2.00 kg with the following formula:

(Mean weight in grams–2000) × 0.33 = Y

IC_{2000}=IC-Y

It can be observed a decrease in FCR_{2000} of 0.205 kg feed / kg meat, between vaccinated and unvaccinated batches. This improvement was directly related to health improvement of the flocks (decreased symptoms and mortality) which resulted to a more efficient use of the feed.

IC_{2000}



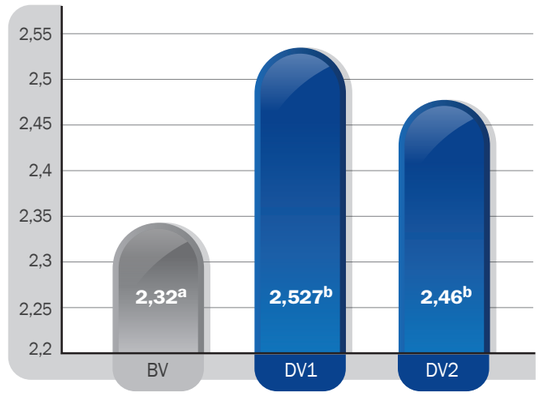
$P_{BV\text{-}DV1}$=0,0041 $P_{BV\text{-}DV2}$=0,0017

The values with distinct superscripts are significantly different at $P \leq 0.05$ by unidirectional analysis of variance (ANOVA).

Body weight at 41 days:

The mean body weight at 41 days of the two vaccinated batches was increased by 170 g, implying a 7% improvement.

Body weight at 41 days



$P_{BV\text{-}DV1}$=0,0042 $P_{BV\text{-}DV2}$=0,0129

The values with distinct superscripts are significantly different at $P \leq 0.05$ by unidirectional analysis of variance (ANOVA).

4 CONCLUSIONS

The incorporation of vaccines against aMPV into a vaccination programme should always be based on a reliable diagnosis of the agent causing the health problems, with clinical, serological and if possible molecular data.

Nevertheless, given the productive model of broiler chickens – very short cycles and rapid rotation – there are many occasions in which an in-depth diagnosis is not carried out.

If we add to this the inadequate monitoring of aMPV in batches of broiler chickens, we can suppose that this disease is mistaken for other respiratory processes on many occasions.

Given the special importance of the respiratory system in genetic stocks for the production of meat, it is vital to safeguard the entire respiratory tract. Vaccinating with HIPRAVIAR® SHS against aMPV a health improvement of the flock was achieved, which directly impacted on production rates.

There are numerous reports implicating cellular immunity as the primary defense against aMPV.

Vaccinated turkey poults without a detectable antibody response were protected against challenge with virulent aMPV (Cook et al. 1989, Williams et al. 1991). This idea was later confirmed by an experimental study done by Jones et al. (1992). In this study. vaccinated turkey poults, which had been B cell depleted by cyclophosphamide treatment did not seroconvert, but were still protected when challenged with virulent aMPV. Subtypes A and B induce a transient increase in the percentage of CD4+ T lymphocytes and an increased expression of IFN-γ in the Harderian gland (Aung. 2007)

The use of an attenuated live vaccine, subtype B, chicken origin (HIPRAVIAR® SHS) in this broiler farm affected by aMPV leads to an improvement in the health status of the batches with significant positive repercussions on the zootechnical results and so an improvement in the economic results.

In any case, poultry vaccination programmes against aMPV should be assessed in a particular way in each integration and season of the year, making the monitoring of the flocks at the marketing age very important to plan an effective vaccination program.