

REO virus vaccination – the answers

Reovirus infections (REO) are prevalent in chickens, turkeys and other avian species worldwide.

Next to viral arthritis, REO viruses have become associated with other disease conditions including, for example, malabsorption syndrome (MAS) and femoral head necrosis (FHN).

The virus is transmitted vertically via the hatching egg and horizontally by faeces and the respiratory systems.

The virus is commonly found in the digestive and respiratory tracts of clinically healthy birds.

● **Why do we use live vaccines for REO virus? Are they used just as a primer or do they protect vaccinated birds against symptoms?**

Live REO vaccines can induce protection against symptoms in young birds, but only when there are no maternally-derived antibodies (MDA) present to neutralise the vaccine. They will not be effective in young birds with MDA against REO. Early REO infections are the most dangerous ones.

The most effective protection against them is by inducing high levels of antibodies in the parent stock, to protect the offspring by MDA. High antigen content inactivated REO vaccines are the most effective, inducing high titres in parent stock.

Priming such parent flocks with a live REO vaccine will further increase the effect of the inactivated REO vaccine. Individual and average titres are higher and more uniform. Most importantly, the percentage of breeder birds that has low or zero titres diminishes. This live priming of the young breeder birds can be done when MDA have waned (after approximately six weeks of age).

● **Mal-absorption syndrome (MAS) is still a current problem in broiler flocks worldwide. Can it be minimised by REO virus vaccination of parent stock?**

MAS is a complex of symptoms occurring when the intestines of the broiler are not able to absorb sufficient nutrients, resulting in deficiencies and growth retardation.

Certain REO strains cause MAS-like symptoms. This does not mean that all MAS is caused by REO infections: any pathogen that disturbs the optimal balance in the intestinal flora, can cause symptoms of MAS.

In cases of MAS caused by REO infections, REO vaccination of the parent stock is the most effective way of prevention.

● **Are different strains of REO virus responsible for different symptoms or syndromes in the infected birds? Why do some inactivated vaccines contain different REO virus strains?**

REO virus isolates have been associated with a great variety of symptoms. This has enabled the categorisation of REO-related infections into different syndromes, such as viral arthritis (VA), brittle bone syndrome, MAS, runting/stunting syndrome and helicopter disease. The S1133 (Lvd-Heijden) strain is reported to be isolated from a case of VA, while the 1733 strain from a case of classic MAS, and the 3005 from a case of brittle bone.

However, a REO isolate from one specific syndrome will not necessarily always cause similar symptoms. Neither is it possible to differentiate various isolates by current serological methods: the REO antibodies do not make a distinction.

In fact, the immune system does not distinguish between the one and the other pathotype: antibodies induced by one isolate will protect equally against the other pathotypes.

Inclusion of isolates of two or more syndromes in a vaccine does not give a broader protection. Moreover, it would be un-practical to include isolates of all syndromes, associated with REO virus infections.

However, including more antigen per dose can increase the immune response, measured in titres. A higher titre sometimes can induce a more effective protection, which in itself could induce a higher titre and a better protection.

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Vaccination of turkeys against AE

Avian encephalomyelitis (AE) is an infectious viral disease affecting young chickens and turkeys and is known to be distributed worldwide. Pheasants and quails are also susceptible to the virus.

Morbidity may reach up to 90% and mortality up to 70%. Birds exposed up to five weeks of age show characteristic disorders of the central nervous system such as ataxia, paralysis and tremor.

Layers and breeders may react with temporary drops in egg production (15-30%) and small eggs. Egg transmission is the major route of transmission of AE virus. Infected breeders will transmit the AE virus for several weeks and cause a decrease in egg hatchability.

Infected chicks that hatch will show the clinical signs of the disease and spread the infection in the incubator to other newly hatched susceptible chicks. Young chicks can also be infected on the farm. The incubation period varies from five to 14 days depending on the route of infection.

The presence of humoral antibodies against AEV in breeders is correlated to protection of the progeny due to an AE field infection. So vaccination of breeder flocks is carried out to ensure that all progeny receive maternal antibodies which protect them until they develop age associated resistance.

The vaccination is carried out with a frozen or lyophilised live vaccine containing the AE virus strain 1143 Calnek via the drinking water or with a combined AE/Fowl Pox vaccine via wing-web.

Killed vaccines have not been used widely. The capability of the vaccine virus to spread rapidly within a flock has been seen in former studies and even the administration of low doses has resulted in the appearance of antibodies 2-3 weeks after vaccination in chickens.

These vaccines are not registered for turkeys and the mode of appli-

cation of these vaccines in turkeys usually followed the vaccination schemes based on the well characterised humoral immune response in chickens.

So a trial was carried out to determine points of vaccination of turkey breeders that result in high antibody titres prior to the onset of lay. BUT Big 6 breeder hens were either vaccinated at 22 weeks of age with a booster vaccination at 25 weeks of age or at the age of 16 and 23, and 9 and 23 weeks respectively with an AE live vaccine.

The antibody response was measured by an enzyme-linked immunosorbent assay (ELISA). First vaccination at 22 or 16 weeks of age resulted in a late onset of antibodies with low titres for a short period, whereas vaccination at 9 and 23 weeks induced antibodies approximately 10 weeks earlier, with higher and uniformly distributed titres. A flock vaccinated in the 9th and 23rd week of life was chosen for further experiments on different administration routes.

One half was vaccinated after withdrawal of water for two hours. In order to allow the unhampered access to drinking water at any time another group received aliquots of the vaccine without previous withdrawal of water directly into the bell drinkers.

Application of the vaccine after withdrawal of water resulted in an one week earlier onset of detectable antibodies with a clearly stronger rise of titres and a significant higher level during the following period of 17 weeks.

The obtained results show that vaccination up to four weeks before onset of lay can not be applied in turkeys which start laying at an age of approximately 32 weeks. An early vaccination provides high antibody titres at onset of lay. Due to the previous withdrawal of water higher antibody titres can be achieved. ■

Gumboro vaccination – a matter of timing

Live IBD vaccines are categorised by their breakthrough titre, virulence and potential to induce immune suppression, into mild, intermediate and intermediate plus.

While the mild IBD vaccines show the lowest invasiveness, the intermediate plus have the highest virulence, breakthrough titre and potential to induce immune suppression.

Successful vaccination against IBD depends on variables such as the level of maternally-derived antibodies (MDA) in the chicks, the choice of the vaccine type (capability of the vaccine to break through this maternal immunity) and the field challenge. Hence, the effectiveness of a Gumboro vaccination programme depends heavily on whether the right vaccine is given at the right time.

The best way to establish the right time of vaccination for a flock depends on the information available.

■ **Measuring and calculation**

When testing methods are available, the antibody titres of the chicks can be measured. Together with the breakthrough titre of the vaccine, the right moment of vaccination can be calculated. Producers of ELISA kits have equations for such a calculation.

■ **Estimation and adaptation**

The vaccination programme of the parent flocks can be used to estimate the correct time of vaccination for the progeny.

Offspring from breeders vaccinated with inactivated IBDV vaccines tend to have higher levels of MDAs than offspring from breeders vaccinated with live IBD vaccines only.

Based on information on the vaccination programme of the parents and knowledge about the invasiveness of the vaccines for the progeny, timing for vaccination can be estimated.

Once this vaccination programme has been applied, the results of the performance can be used to adapt the programme for subsequent flocks.

Progeny from mixed flocks will have a greater variation of MDA levels, requiring more than one

vaccination to cover all groups within the flock.

■ **No information available**

Often, there is no information on the immune status of parents and/or offspring. The strategy then should be to prevent as much risks as possible. The vaccination programme will have to cover progeny with different levels of MDAs. The possible presence of offspring with little or no MDAs poses a serious risk. When unvaccinated, they are an easy target for early field challenges, becoming a focus for the infection pressure to rise to uncontrollable levels. For this group, vaccination at 1-7 days with a mild vaccine is recommended.

■ **Mild, intermediate or hot? – a matter of strategy**

Intermediate strains are the most versatile type of IBD vaccines, usable in various circumstances. They are used under 'normal' conditions as a standard procedure under an IBD field pressure.

Mild vaccines are safe, even in day-old chicks without any MDAs, which would easily neutralise them, even at very low levels. Often, when breeder flocks have low IBDV titres, a percentage of the progeny hatches with insufficient maternal protection.

Thus, an early vaccination with a mild vaccine will protect this group and reduce the risk of transmitting field virus.

Intermediate plus vaccines are to be used in the presence of very virulent IBDV challenges, which infects young chicks at a very early age, in a time when intermediate vaccines will be still neutralised by the high levels of MDAs due to their lower breakthrough titre.

To be able to compete with such vIBD viruses and induce immunity in the face of still high MDA levels, more invasive IBDV vaccines strains are required.

Intermediate plus vaccines may have immunosuppressive effects, being mainly used to reduce mortality and the prevalence of vIBDV in the flocks. Once this has been reached, it is recommended to return to the use of intermediate vaccines. ■

Infectious bronchitis disease and control

Avian infectious bronchitis (IB) is a highly contagious disease, causing major economic damage to the poultry industry worldwide. It is caused by a virus of the Corona group and spreads easily from chicken to chicken and from flock to flock through the movement of infected birds, by people and equipment or by air.

IB infections can cause a variety of symptoms. Which symptoms dominate will depend on the type of IB strain, the age of the birds, the immunity status at the time of infection and management conditions.

In broilers the economic damage is caused mainly by poor growth and increased condemnation rates. Secondary bacterial infections usually aggravate this economic damage.

In layers the economic damage is caused mainly by a drop in egg production, together with thin shelled or deformed eggs.

Production may drop by 25% and never fully recover.

In breeders economic losses are increased by poor hatchability. In some cases IB infections can cause kidney damage.

■ Serotypes

One important feature of the IB problems is the great variation in serotypes. The most common strains worldwide is the Massachusetts serotype. However, IB field strains – against which vaccines of the Mass serotype do not fully or only partially protect – are also prevalent. Such strains are called ‘variant IB strains’.

Additional vaccine strains may be required to control variant infections. Some commonly reported variant strains are the Arkansas serotype (USA only), and the D274 and the 4/91.

Over the years, the importance of certain serotypes may increase and decrease. In general, the Massachusetts serotype has remained the most widespread and most important all over the world.

■ Diagnosis

IB diagnosis can be based on virus isolation and subsequent strain identification or by serologic identification. Most tests can identify the infection as an IB infection, but cannot identify which

serotype of IB is causing the infection. This identification is necessary to avoid introducing new serotypes into areas where they were not previously found.

■ Control

Wherever in the world there is large scale poultry production, there is also IB. Since IB spreads so easily eradication is not practical. Control will have to rely on vaccination and reducing pressure from field infection (biosecurity).

■ Vaccines

Development of effective vaccines against IB is complicated by two important factors:

- Not all IB isolates are well immunogenic, and many are not well suited for the development of vaccine.
- New serotypes, against which new vaccines must be developed, are frequently identified.

For vaccination live vaccines or inactivated vaccines can be chosen. Live vaccines can be administered by mass vaccination methods such as through the drinking water or spray method.

Killed vaccines must be given by injection. However they are easier to combine with other vaccinations. The duration of immunity tends to be longer.

Combining the two strategies, by giving live vaccines first (priming) and boosting with killed vaccines can improve the efficacy of the total programme.

■ Cross-protection

Vaccines of the same serotype may not always be available. However, many IB vaccine strains provide some level of cross-protection against other serotypes.

This level of cross-protection can be increased by repeating the vaccinations more frequently. Also, a more immunogenic live vaccine of a different serotype can prove to be more effective than a less immunogenic vaccine strain of the same serotype.

■ Failures in vaccination

Vaccination failure can be due to vaccine strains that are not sufficiently immunogenic, poor vaccine quality, vaccination with vaccines belonging to the wrong serotype, inadequate vaccination programmes or inadequate methods of application. ■