Going back to basics with vaccination

The development of modern poultry production and breeding has owed a lot to vaccination. Without vaccination poultry production and breeding as we know it today would not exist.

This is because vaccination was the development in the last 50 years that countered those infections which, if they had not been controlled, would have been the downfall of the poultry sector.

Most articles on vaccination like to focus on the latest developments in vaccine technology or the newest vaccine to come to the market. This article will not be doing this – it will go back to consider the basics on which sound vaccination practices are based.

The first vaccination

Let us go back to the beginning and visit a small rural community in England in the 18th Century at a time when smallpox was rife. The local medical practitioner, a Dr Jenner, noticed that the milkmaids never succumbed to this dreadful disease and he wondered why.

Eventually he discovered that when a girl became a milkmaid she invariably caught a skin infection. This was cowpox. However, the milkmaids knew that this was par for the course and the problem always disappeared without consequence and so the doctor was never consulted.

Dr Jenner wondered whether the two events might be associated. So, he took infected material from the skin lesions of some cows that had cowpox and infected some people with it by skin scarification. Yes, they had a nasty reaction from the cowpox but, the next time smallpox swept through the area, everyone who had received this treatment survived. This was the first instance of vaccination.

Why was it called vaccination? The answer lies in Latin. The Latin word 'vacca' means cow and so vaccination basically means 'the cow treatment'!

The situation we had here was one of a very closely related virus that did not cause serious disease in man, namely the cowpox virus, providing protection against a very similar virus, namely the smallpox virus. This protection is known as cross protection. We now know that this is because closely related micro-organism sometimes share common antigens or have very closely related antigens which, when they stimulate the immune system, produce identical or very similar antibodies. These antibodies provide the protection against disease caused by their micro-organism or a very closely related one. Thus, identical or very similar antibodies can induce cross protection.

So, what is the relevance of this to poultry? The relevance is that this principle is used for one type of Marek's disease vaccine. Marek's disease is caused by a herpes virus and the turkey herpes virus will infect chickens and not cause disease but it will induce an immunity that will confer cross protection against Marek's disease.

One of the Marek's disease vaccines is known as THV. Did you know that this stands for turkey herpes virus?

In the 19th Century research into ways of protecting animals by vaccination was at its zenith. Out of this research came some fundamental but very important discoveries. It was found that if you took a micro-organism through many life cycles in the laboratory an interesting phenomenon occurred. The virus lost its ability to cause disease (pathogenicity) but kept its ability to induce immunity (immunogenicity).

French influence

Much of this work was done in France at institutes such as the Pasteur Institute in Paris and French words have entered the vocabulary of vaccination. In this process the disease causing organism becomes milder or, to use the French word, is attenuated. The life cycles also assumed a French name and these cycles are often referred to as passages. Thus, if we look at the literature accompanying our vaccines we will see quite a few of the live vaccines being referred to as 'a live attenuated vaccine'.

Here again we have a good example in the infectious bronchitis vaccines H120 and H52. Did you ever wonder what these two numbers referred to? They relate to the number of passages the master seed virus that is used in vaccine manufacture has been passed through.

Infectious bronchitis vaccination

If we think this through, the logic holds up in that the H120, which has been processed through more passages, is the milder vaccine, while the H52 is the more aggressive vaccine. That is why if we ever use H52 we should always prime those birds, especially breeders, with H120. This means the H120 actually 'vaccinates' the birds against pathogenic attributes of the H52 infectious bronchitis vaccine which have not been totally eliminated by attenuation!

For example, if we administer H52 to laying breeder birds that have no immunity against infectious bronchitis (for example, from a priming dose of H120 vaccine) then the pathogenic effects of infectious bronchitis will be seen.

Typically these will be seen in the form of depressed egg production and the presence of 'IB eggs', that is, eggs with poor shells and poor shell colour.

More recently a novel technological approach has been developed for live vaccines. That approach is to utilise a virus that can infect chickens with no pathogenic effects and to encode in it the ability to create antigens on its surface for a variety of poultry diseases.

This vector virus or vector vaccine is then able to induce immunity for a variety of viral diseases. This has the advantage that one vaccine is then capable of doing the 'work' that was previously done by several. This type of vaccine is still very much at the developmental stage although commercial evaluations have taken place. Consider the impact of this when we discuss side effects later on.

Live vaccines basically work by mimicking the field or disease causing virus. They infect the bird as the field virus would do and then they stimulate the bird's immune system to produce anti-

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bodies (immunity). The first part of the immune system to be stimulated is the local immune system. In the case of a live vaccine against a respiratory disease this would be the local immune system in the respiratory tract. After this the central immune system is stimulated.

It should be remembered that it is the central immune system that produces antibodies and, so, an antibody titre (the level or quantity of antibody in the blood) resulting from vaccination is only an indication of the immunity that has arisen from the central system. Local immunity is not reflected in the blood titre and so we can have an effective and strong local immunity in a bird with a low antibody titre.

As has just been said the effectiveness of a live vaccine rests in the fact that it is alive and so if we do anything that inadvertently kills off some or all of the micro-organisms in a live vaccine we will reduce or totally eliminate its ability to produce immunity. Viral vaccines can be killed by temperature abuse, for example by being carried in the back of a pick up in direct sunlight in a tropical country. This can be countered by the use of a Kool Box.

If a live vaccine comes into contact with a disinfectant do not be surprised if the disinfectant does the job it is supposed to do! Thus, we must be very careful how we clean and sanitise the equipment that is going to be used for the preparation and administration of a live vaccine. This is why it is better to sterilise the equipment used for administering Marek's disease vaccine in the hatchery by autoclaving rather than disinfection.

In some parts of the world the regular use of water sanitisers in the drinking water a flock receives is a common practice. In such a situation it is very important to ensure that the sanitiser and a live vaccine are not administered concurrently! Even chlorine levels in the water can have a deleterious effect on a live vaccine but this can be negated by the use of powdered milk.

This practice also removes metallic ions, which can adversely affect a live vaccine, from the water.

Basis of today's dead vaccines

Another discovery in the early days of research into vaccines was that it was possible to kill a micro-organism without destroying its antigens and hence its ability to produce immunity. This is the basis of today's dead vaccines.

It will be recalled from earlier comments about live vaccines that these work by mimicking the field virus and that a key stage of this was infecting the bird. Obviously this can not happen with a dead vaccine. Thus, if a dead vaccine is to work, we must administer a dose of it by injection into the body of each and every bird in the flock.

If a bird does not receive a dose of a dead vaccine no immunity will develop in that bird. This can happen very easily in practice. Three situations come to mind. The first is when we are vaccinating the birds under the skin and we accidentally put the needle through the skin twice, squeeze the trigger and shoot a dose of vaccine into the litter!

The second situation is when we are not concentrating on the job in hand and the vaccine runs out. Did we inject one, a couple or a couple of dozen birds with a dose of air! Needless to say air will not induce immunity!

The third situation arises when our segregation of vaccinated and still to be vaccinated birds is not as good as it should be and some of the latter co-mingle with the former. Thus, vaccination is as good as the people administering the vaccine to the birds.

If we take vaccination right down to the basics it is successful if it produces a protection that lasts for long enough. For this to occur we must get enough of the vaccine to stimulate the bird's competent immune system. We have introduced

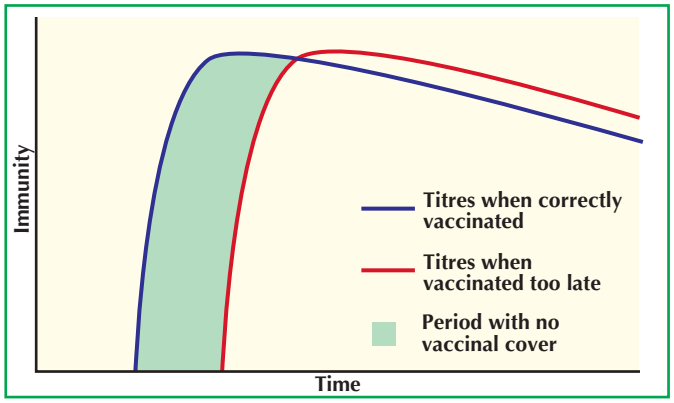

Fig. 1. The consequence of vaccinating breeders too late.

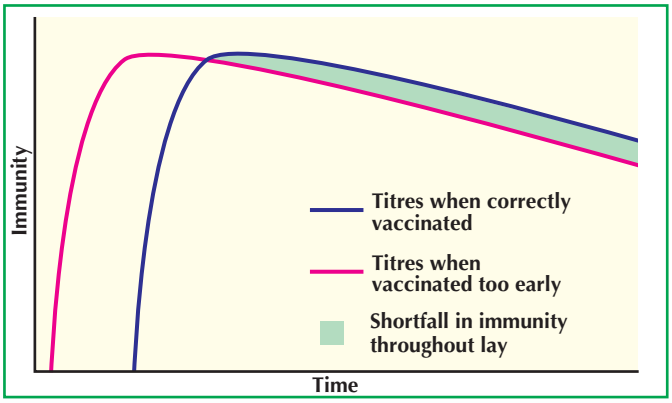

Fig. 2. The consequence of vaccinating breeders too early.

another important word here and that is 'competent'. So, what does this mean? A 'competent' immune system is one that is able to respond fully and totally to the vaccine.

If this competency is impaired then the bird will not produce immunity when vaccinated or, what is more likely, it will not produce enough immunity. That is, it will not fulfil its potential to produce immunity. Birds that are not fully competent are said to be immunosuppressed and many things can cause immunosuppression.

Some infectious agents can cause immunosuppression and good examples here are Gumboro disease virus which destroys the bursa of Fabricius (a key part of the immune system) and chicken anaemia virus.

There is evidence to suggest that Marek's disease virus may have some immunosuppressive properties and that some mycoplasma strains may have a limited similar effect. Certain mycotoxins in the feed are known to be immunosuppressive as can be stress, especially heat stress. In many of these instances the immunosuppression is not very great, but it takes the edge off our vaccination programme.

Requirement for protein

Another facet of vaccination is that the production of antibodies, which are effectively immunoglobulins, requires protein. If protein is required for this purpose then it is not available for growth, egg production or the other important roles it has in the body.

We know that if we compare non-vaccinated specific pathogen free pigs with siblings that are reared in a commercial environment and exposed to the microflora that is there and are vaccinated there is a 5% difference in performance. This is a reflection of the amount of protein that is diverted from growth and into antibody production, be it against field challenges or in response to vaccination.

Thus, when we consider vaccination we must remember that there are downside effects that need to be considered.

Firstly, there is this diversion of food from growth to antibody production and, secondly, there are the consequences of side effects when we are using live vaccines. Thus, the skill of the veterinarian in planning a vaccination programme is not necessarily in deciding which vaccines to put in but rather which vaccines to not use or, even, to remove from the programme.

Achieving the balance

The danger is that some veterinarians adopt the stance that they can not be wrong if they have put something into the vaccination programme, but they might be proven to be wrong if they take something out. This can result in flocks that are unnecessarily and excessively over-vaccinated! Surely the approach must be to have the least vaccine input that is consistent with maintaining a good health status?

At breeder level we need to ask ourselves what is the goal of our vaccination programme? Many say it is to protect the birds. To some extent this may be the case, but surely its primary goal must be to maintain egg production as this is the primary goal of the breeder flock. For this to be best achieved two things must occur.

Firstly, it is imperative that when the flock comes into lay that immunity has already been established. There must be adequate time between the last vaccine administration and first egg.

Interestingly, here we have a situation where some breeds now have an earlier onset of lay and yet many people have not reassessed their vaccination dates accordingly (see Fig. 1). Surely, if a flock is coming into lay earlier than it once did then the last vaccine administration in the pre-lay vaccination programme needs to be correspondingly earlier?

Secondly, during lay immunity wanes. Thus, if we vaccinate too early our peak immunity can occur before first egg and we can then have the situation that, for every point in the laying cycle, the level of immunity is not quite as high as it could have been (Fig. 2).

In addition, the point towards the end of lay when the immunity falls below the level at which adequate protection is bestowed occurs earlier than it needed to. In essence we have not protected our flock as well as we could have done.

So, if we look at vaccination one message comes across loud and clear. It does not matter how good a vaccine is if its application is below par and if we have not given adequate consideration to basic principles. ■