

REOVIRUS

Reoviruses were first recognized as avian pathogens in 1957. This first case was from chickens showing clinical signs of what became known as viral arthritis (VA). Since that time reoviruses have become associated with other disease conditions including malabsorption syndrome (MAS), femoral head necrosis (FHN), pericarditis, myocarditis, hydropericardium, gastroenteritis, hepatitis, and acute and chronic respiratory syndromes.

Reoviruses are double-stranded RNA viruses and are highly resistant to both physical and chemical inactivation, as well as low pH. These viruses are very similar to Infectious Bursal Disease Virus and are similarly resistant to many common disinfectants. Reoviruses will remain infective in contaminated environments for long periods of time. When held in a culture, reoviruses remained viable for almost a year when held at 72°F. In a management system that allows for a two week turn around time on built-up litter, the effect on the reovirus population would be minimal.

Reovirus has been shown to cause an inflammation within tendons that may result in a reduction in these tendons tensile strength. Inflammation, due to reovirus, may develop in a joint resulting in arthritis or within a tendon sheath resulting in tenosynovitis. In broilers, roasters and breeders, this results in lameness and stunting. This is often not seen until after five weeks of age.

Reoviruses have been associated with a syndrome of nutrient malabsorption that result in poor growth and skeletal abnormalities. This lack of utilization of provided nutrients has been related to intestinal, pancreatic and/or liver abnormalities.

Diseases associated with reoviruses have been reported in both heavy and light breeds of chickens. Heavy breeds are more susceptible to disease associated with tendons (viral arthritis). This is probably due to two reasons:

1. Large, meat-type birds grow rapidly, resulting in physical changes to the load-bearing leg tendons. This may predispose birds to infection. Leg tendons in broilers also have less tensile strength and a more open structure which is more conducive to infectious agents.
2. Broiler breeds take approximately one week longer to respond serologically to reovirus infections. This delay in immune response may contribute to the greater susceptibility of the heavy breeds compared with lighter breeds.

Reovirus-associated viral arthritis is seen more frequently in males than females. Other than increased susceptibility in broiler-type birds, there is no scientific evidence of increased incidence in specific heavy breeds.

ECONOMIC IMPACT

At this time, reovirus-associated disease having an economic impact on the worldwide poultry industry are viral arthritis (VA), malabsorption syndrome (MAS), brittle bone (BB), and femoral head necrosis (FHN).

Tenosynovitis is clinically observed in young chickens (five to eight weeks of age) and mature breeders (over 20 weeks of age after onset of production). The clinical disease in the five to eight-week-old group results in higher mortality and lower grade broilers/roosters. When breeders break down, the losses are due to excessive culling due to lameness and lowered fertility (reduced desire to mate due to sore legs).

MAS is most commonly associated with birds 7 to 21 days of age. High mortality is seldom seen; however, poor feed conversions and decreased marketability are common.

Reoviruses also cause FHN and BB. The mechanism of these bone problems is thought to be through the malabsorption of nutrients essential to normal bone development. The loss is seen in decreased livability and excessive trimming similar to VA.

SEROTYPES

Since the mid-1950's, when avian reoviruses were first being isolated, scientists have tried to group each newly discovered strain into a serotype group.

Japan, in 1966, isolated 77 reoviruses and classified them into five serotypes. The U.S., in 1975, classified nine isolates into four distinct serotypes. In 1980, five more reovirus strains were taken from the U.K., Germany, and U.S. and compared with the existing vaccine strain - S1133. Their conclusions were that these six strains belonged to three serotypes. When a prototype from these three serotypes (1980) were then compared with the five Japanese serotypes (1966) and the four serotypes from the U.S. (1975), the conclusion was, "there are at least 11 serotypes.

In summary, there are hundreds of reoviruses that have been isolated and at this point, it is not possible to group them into distinct serotype categories.

RELATIONSHIP TO EACH OTHER

Reovirus strains that are significantly different from each other in terms of classification (heterologous strains) show considerable cross-reaction. Due to this relatedness, vaccination with S1133 strain of reovirus has been shown to protect against many other strains of reovirus. It is also related enough to the strains of reovirus used in the inactivated vaccines to serve as an excellent live primer.

ISOLATES DIFFER IN PATHOGENICITY AND ANTIGENICITY

Reoviruses differ in their ability to cause disease as well as their ability to cross protect. Several strains have been categorized as exhibiting low, intermediate, and high pathogenicity when introduced into unprotected broilers. In general, the viruses that were classified as being more pathogenic produced higher mortality rates, lower average body weights and induced a more consistent antibody response than did the less pathogenic reovirus isolates. The two strains shown to be the more pathogenic are 1733 and 2408. The consistently high antibody response from them is why at least one of them is included in many of the inactivated reovirus vaccines currently on the market. A table of the most common reovirus isolates is shown below.

<u>STRAIN</u>	<u>ASSOCIATED WITH</u>	<u>PATHOGENICITY</u>
S1133	VA	MILD
2408	MAS, VA	VERY PATHOGENIC
2035	MAS, VA	INTERMEDIATE
2177		NON-PATHOGENIC
1733	VA, MAS, MORTALITY	VERY PATHOGENIC
C08	MAS	NOT CHARACTERIZED
3005	BB	MILD

IMMUNODEPRESSION

Selected reoviruses are capable of causing a transient immunodepression. Pathogenic reoviruses in young birds have been shown to cause bursal lymphoid depletion lasting as long as 11 days. (There was also a similar depletion in the thymus.) This resulted in a decreased response to Newcastle disease vaccination as seen on serology and challenge results.

IMMUNE RESPONSE

Birds require 7 to 10 days to develop neutralizing antibody following inoculation. Whereas the precipitating antibodies detected by agar gel precipitation (AGP) test are not present until two weeks post-inoculation. Neutralizing antibodies are also present longer than the AGP test shows positive for precipitating antibody. Practically, this means that a negative AGP test does not indicate absence of protection.

Virulent strains of reovirus, when picked up from the environment or shed to progeny through the egg, may not be cleared by circulating antibodies. Some virulent reovirus isolates show great persistence despite an immune response. This persistence has been realized when live vaccines have not been properly attenuated and when some farms, with a virulent reovirus population, show continued high incidence of VA or MAS.

Maternal antibodies (MA) have been demonstrated as protective against natural challenge. However, some virulent strains may be capable of establishing themselves despite MA presence. MA's also can interfere with vaccination.

Birds develop an age resistance towards reovirus infection. The first week of life is the most critical when addressing both potential damage and development of long standing persistence and shedding. In other words, infection at one day of age may develop into a case of VA at four weeks of age in which the reovirus persists for the life of the bird, whereas infection at two weeks of age only develops into a transient inflammation and the reovirus is cleared in less than one week. Reovirus infections after 10 weeks of age are very rare (we commonly see VA in birds older than 10 weeks, however, this is usually associated with an earlier infection manifesting itself as the birds increase in weight.)

Duration of immunity from vaccination is related to the age of the bird and the degree of attenuation of the product used. Very highly attenuated products should be used in birds less than three weeks of age because this is the time that they are most susceptible to the virus. Because of this degree of attenuation, protection lasts only six or seven weeks. Revaccination with a less attenuated live vaccine at this time will generally protect birds through the susceptible time period.

VACCINATION

Avian reoviruses are found throughout the world, especially in areas of concentrated

poultry production. It is unrealistic to eradicate this virus from most operations and therefore vaccination for reovirus is widely practiced. Protection is achieved through maternal antibodies followed by vaccination, if needed.

Maternal antibodies last a variable amount of time, depending on the hen's titer when laying the egg. The goal should be for maternal antibodies to be present through the first week of life, the most susceptible time period.

When maternal antibodies are virtually depleted, a highly attenuated vaccine can be given at this time. This often occurs between 7 and 14 days. Protection from challenge can be expected to last five to six weeks.

PRIMING

The two vaccinations described act as an excellent prime to the immune system for eventual boosting of breeders. Since all reoviruses are related to the S1133 strains of reovirus, this strain works well as the priming antigen.

BOOSTING

Breeders are often given an inactivated vaccine four weeks prior to the onset of egg production. This further boosts their titer to increase protection to progeny. It has the added advantage of decreasing the risk of vertical transmission of the virus. Strains in most killed vaccines are closely related to the strain S1133 used in almost all live vaccines. This relationship (related viruses in both the live and killed vaccines) seems to be important in reoviruses as it is with other viruses.

MAREK'S INTERFERENCE

The use of live reovirus vaccine used in combination with Marek's vaccine is not recommended. This practice has resulted an increased incidence of lymphoid leukosis at broiler processing. The mechanism of this interference is not well understood. This interference is not seen when vaccination is done at seven days post Marek's vaccination.

MEASURING RESULTS

Performance

Evaluation of a reovirus vaccination program should include the total performance of a flock. Beneficial results have been seen in broilers in feed conversion, weights, livability, and processing parameters. Beneficial results in breeders include increased fertility and less mortality during the life of the flock.

Serology

Serology for reovirus is usually done using the SN/VN, ELISA, or AGP tests. ELISA and AGP tests detect group specific antigens and therefore are not capable of distinguishing between strains. However, SN/VN results differ depending on the antigen used in the test.

Positive serology does not indicate a pathogenic reovirus. There are many apathogenic isolates of reovirus. In other words, not all leg problems seen in the field in birds showing positive serology to reovirus are reovirus-caused leg problems. A more complete examination is required.

Serology is useful for monitoring breeder titers as well as its use diagnostically in conjunction with other lab tests. An effective program for breeders can often be fine tuned with serology by measuring MA's and avoid interference with the first vaccination.