

## **AVIAN ENCEPHALOMYELITIS**

### **AVIAN ENCEPHALOMYELITIS (AE) DISEASE**

It is best to think of AE as an enteric disease that can spread to the brain and reproductive tract of susceptible birds. The progression of AE depends on the age at which the bird is infected, the strain of virus and the bird's immune competence. When birds over three to four weeks of age are infected, the virus is ingested and grows in intestinal tissues. As it multiplies, it causes an immune response. The bird produces enough antibodies in time to keep the virus from spreading to the brain and causing clinical disease. The virus can, however, spread to the reproductive tract of the hen and cause a temporary drop in egg production. The incubation period from infection to egg production drop is at least 11 days. In breeders, AE virus may be transmitted through the egg to the progeny. The virus is also shed from the gut into the cloaca where it may contaminate the egg shell and infect the young as they hatch. Virus shedding occurs from the gut and lasts only five days. There is probably no carrier state.

When birds under four weeks of age are infected, the virus replicates in the gut, but the birds are not yet fully immune competent. Because they lack immune development, pathogenic strains enter the blood stream and move to the brain, causing neurologic symptoms. The incubation period from infection to neurologic signs is 11 days, unless birds were infected as embryos. If infected as an embryo, the incubation period varies from one to seven days. Pathogenic field strains do not affect or kill the embryo. It is necessary to hatch birds and observe them to see if they are infected. Young birds shed virus from the gut for up to two weeks. This longer shedding period in the young greatly increases spread of virus to pen mates.

There are no obvious clinical signs of AE in adult birds. Laying flocks often experience a temporary decline in egg production, but it may be only slight (5-10% decrease). The eggs that are set during the period of decreased production, will have a decreased hatchability. An enlarging and bluing of the eye may occur in the adult birds. In the young (under four weeks of age) clinical signs begin with a dullness and reduced activity. Affected birds soon become uncoordinated and have difficulty moving around. A tremor of the head and neck develops that resembles shivering. Keep in mind that birds do not shiver when cold stressed, but instead fluff their feathers up. Shivering, or tremors, is a sign of neurologic disease. Affected birds eventually become immobile, lie on their sides and 60 to 90 percent will die.

In a typical outbreak in young birds, 40 to 60 percent of the flock will be affected and overall mortality will be 25 to 50 percent. In the average outbreak, there are no easily visible gross lesions caused by the AE virus. Lack of easily visible gross lesions is an important part of the history. There are microscopic lesions in the gut and brain that confirm the diagnosis.

Immunity to AE virus begins developing in immune competent birds (3 to 4 week old chicks) within four days. Breeders begin passing AE maternal antibodies to progeny within eleven days post-exposure. Detectable antibody levels protect birds against clinical disease and reduce shedding from feces. Bursectomizing young birds results in their inability to make antibodies. Birds which are bursectomized are susceptible to AE neurological disease. Therefore, antibodies (humoral immunity) are what is important in protection against clinical

disease from AE and cell mediated immunity (CMI) plays a minor role. Immunity does not prevent infection of the gut, but stops spread of virus to the brain.

Actively acquired antibodies (post-infection) or passively acquired antibodies (maternal antibodies) both protect well. The decay rate of antibodies in progeny is slow, and protective maternal antibody lasts four to six weeks. This is ample time for young birds to become immune competent.

It is difficult to describe the impact of AE disease on the USA poultry industry because the incidence of clinical AE is extremely low due to widespread vaccination of breeders. The vaccine is inexpensive and easy to administer; therefore, it does not represent a significant cost in rearing breeding stock. AE vaccination represents an inexpensive insurance policy against a 25-50 percent loss of progeny.

## **VACCINATION**

There is no specific treatment for AE. Cull affected chicks to reduce virus spread. Once clinical signs are over, the disease will not reoccur in the same flock.

Live AE vaccines are derived from a mild, but immunogenic field strain of AE. The vaccine virus is not attenuated and can cause disease in young susceptible birds and egg production losses in mature hens. Lyophilized vaccine is available for use in turkey breeders and chickens. The vaccine should be stored refrigerated (not over 45°F, 7°C). Vaccines are available for administration in the drinking water and the wing web injection method. Combination products with AE and Pox are available for the wing web injection method.

The immune response to live AE vaccine is detectable by 4-11 days post vaccination. When circulating antibodies can be detected, maternal antibody is being passed to progeny through the yolk. Immunity tends to be long lived and maternal antibody half-life is long - at least nine days. It is therefore, possible for protective maternal antibody titers to last 4-6 weeks in progeny. This protects progeny until they are old enough to be immune-competent and resistant to AE clinical disease. The vaccine should not be given closer than four weeks prior to onset of egg production, since it is not attenuated and can cause egg production drops.

Immune response can be measured by the agar gel precipitation (AGP) test, serum neutralization (SN) test, embryo susceptibility test or by enzyme-linked immunosorbent assay (ELISA). Immune status evaluations are done prior to lay and, if certain criteria are met, the flock need not be revaccinated. Empirically established prelay values are those adequate to get the breeder flock through lay without any progeny problems. The AGP test is insensitive and not the best method to use. Most breeders like to see 70-80% AGP positive sera from vaccinated flocks, but lower numbers could still mean protection. SN values of properly vaccinated birds will range from log<sub>10</sub> neutralization indices (NI's) of 1.8-3.0. NI's of over 1.5 are considered adequate for protection. A faster, less expensive test is available. It is called the embryo susceptibility test. In this test, fertile eggs are collected from hens and then inoculated with an embryo-adapted (Van Roekel) AE virus strain, dose of 1000 EID<sub>50</sub>. Maternal antibody from the hens should protect the embryos. Embryos may be examined for muscular dystrophy or allowed to hatch out and examined for clinical disease. If more than 70 percent of the eggs from a flock are resistant, the flock is considered to be adequately immune.

The embryo susceptibility test can only be run on flocks already in egg production. The SN test is time consuming and results may be available after it is too late to revaccinate a flock. The ELISA test has none of these disadvantages. With the ELISA test, the goal is to have all birds seroconvert prior to production. The test is fast and reliable.

Once flocks attain the serological profiles described, the duration of protective circulating and maternal antibody levels is considered to be for the commercial life of the

breeder, namely, one production cycle. AE virus is given to any egg-producing flock, whether commercial egg layers or breeders, to prevent economic loss due to reduced egg production in infected, susceptible hens. It is given to breeders to not only prevent transmission of pathogenic virus to progeny, but to pass protective levels of maternal antibody. The principles for programming AE vaccination are as follows:

1. Give the vaccine after maternal antibodies have disappeared. In most progeny, this occurs between the ages of eight and ten weeks at the latest.
2. Give the vaccine at least four weeks prior to onset of egg production. Both vaccine and field virus can cause depressed egg production and can be vertically transmitted. Administration four weeks prior to onset of production precludes vaccine-related production problems when properly administered to healthy hens.

With these principles in mind, it is possible to propose some vaccination programs for AE in chickens.

## COMMERCIAL LAYERS

Follow label directions for the type of vaccine used.

<u>Age</u>	<u>Route</u>
8 - 14 w	Water, Wing Web

One vaccination will protect hens against egg loss. Serology is not necessary because field challenge will boost immunity quickly and protect the reproductive tract. Field challenge will result in a transient gastrointestinal infection with no signs and no economic consequences.

## BREEDERS

Follow label directions for the type of vaccine used. A program similar to that for layers can be used, except that some breeders come in to egg production much later, thus giving more flexibility to the timing. However, it is best not to vaccinate too late since, unlike layers, serology should be done and revaccination may be necessary. An example:

<u>Age</u>	<u>Route</u>
8 - 15 w	Water, Wing Web

If an immunization failure is detected by a rapid method such as ELISA, there is still time to vaccinate a breeder flock four weeks prior to production. A more rigorous program used by some breeders would be:

<u>Age</u>	<u>Route</u>
8 - 10 w	Wing Web
15 w	Water