

Avian Encephalomyelitis Technical Bulletin

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Avian Encephalomyelitis

Introduction

Avian Encephalomyelitis, also commonly known as epidemic tremor, is a disease caused by an enterovirus.

The disease poses a particular threat to

breeders and layers. It also may affect turkeys, pheasants and coturnix quail.

The disease was first described in 1932. It is worldwide in distribution. The disease spreads by vertical as well as horizontal transmission. The horizontal transmission may be more common, but vertical transmission is clinically more important.

Egg transmission is the major route of transmission of Avian Encephalomyelitis virus. Infected breeders will transmit the A.E. virus for several weeks and cause a decrease in egg hatchability. Infected chicks that hatch will show clinical signs of the disease and spread the infection in the incubator to other new hatched susceptible chicks. Young chicks can also be infected on the farm. The incubation period varies from 5 to 14 days depending on the route of infection.

The clinical disease is mainly seen in young chicks, between 1 and 3 weeks of age. Affected chicks sit on their hocks, do not

move well and many fall on their sides. A fine rapid trembling of the head and neck can be seen, but especially felt when affected chicks are held in the hand. Mortality in naturally infected chicks varies and can be as high as 75%.

In laying and breeding flocks, A. E. virus infection causes a marked drop in egg production, which returns to normal in about 2 weeks.

Diagnosis

The following methods may help in making a diagnosis for Avian Encephalomyelitis.

1. Virus Neutralization test.*
2. Agar Gel test.*
3. Elisa test.*
4. Embryo Susceptibility test.

The above tests are only indicative of antibody present but not necessarily disease. The final diagnosis can be achieved either by histopathology or by virus isolation. Classic diagnosis is typically made by the presence of brain lesions shown histologically.

Origin of Vaccine Virus

Avian Encephalomyelitis (Strain 1143) virus was isolated from the brain of a chicken, showing neurologic symptoms, at the N.Y. College of Veterinary Medicine by Dr. Bruce W. Calnek in 1961 (Avian Disease 5:297). The virus was passaged in chicken embryos by the yolk-sac route and by oral passage in Specific Pathogen Free Chickens. Lohmann Animal Health obtained this virus from Dr. Calnek in 1963.

Vaccination

The disease can be prevented by vaccinating breeder flocks with live vaccine between 6 to 18 weeks of age. It is a common practice to protect layers against avian encephalomyelitis to help prevent a sharp decline in egg production.

It is important that the live vaccine is administered properly and at the right time. Vaccinating the hens without previous exposure in production may result in a transient decline in egg production, decrease in hatchability and transmission of

the disease to the progeny. Avian Encephalomyelitis vaccine is pathogenic to young chicks and development of immunity is not as efficient in presence of maternal antibodies; therefore, it is preferable to ivaccinate birds that are 6 weeks or older.

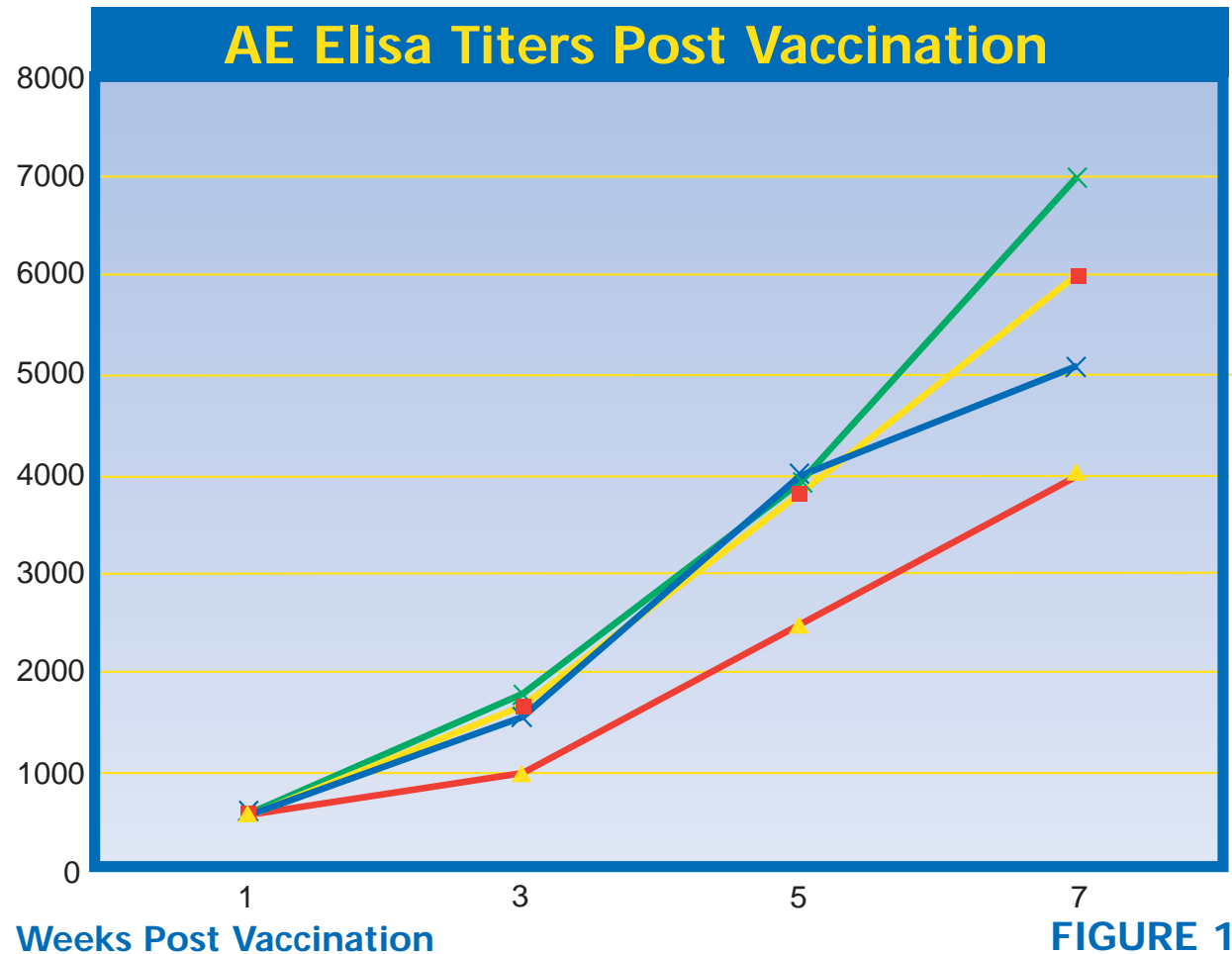


FIGURE 1

Choosing the Right Vaccination

A.E. vaccines are produced in chicken embryos. The continuous propagation of virus in chicken embryos makes the virus adapt to the nervous tissue. In order to prevent the virus from becoming adapted to the chicken embryos, occasionally the virus needs to be passaged back into chickens.

If an A.E. vaccine is undesirably embryo adapted then:

- 1) oral vaccination may not infect well and not spread to the entire flock.
- 2) the A.E. vaccine itself can induce up to 2-3% mortality in a pullet flock two weeks after the vaccination which may go unseen.

The level of embryo adaptation of a vaccine can be determined by increase incidence of muscular dystrophy that the virus produces in inoculated chicken embryos.

The level of embryo adaptation of A.E. vaccine varies among vaccine manufacturers (Table 2). The embryos from groups inoculated with competitor A and B vaccines had more muscular dystrophy compared to Vineland and uninoculated control groups...i.e. Vaccine A and B were more embryo adapted than Lohmann Animal Health's A.E. vaccine.



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Vaccine Embryo Adaption Comparison. Joe Schulz, Arbor Acres Farm		
VACCINE	Number of Embryos with Muscular dystrophy # Live at 19 days*	Embryo Adapted
Lohmann Animal Health's A.E.	1/34	3%
Competitor A	21/24	87.5%
Competitor B	24/25	96%
Van Roekel Strain (Highly Neurotropic Strain)	27/27	100%
Uninoculated controls	0/39	0%

*SPF embryos were inoculated at 7 days of age and examined at 19 days to assay for embryo adaption.

Table 2

Summary of Key Features of Lohmann Animal Health's AE Vaccine

1. Lohmann Animal Health's vaccine is the least embryo adapted of all vaccines compared. This is because Lohmann Animal Health's AE vaccine is periodically back-passaged to prevent the virus from getting embryo adapted.
2. Due to low embryo adaptation, Lohmann Animal Health's A.E. vaccine spreads from bird to bird more effectively and does not induce post-vaccination neurological signs.
3. Lohmann Animal Health's A.E. vaccine gives excellent ALISA titers with low coefficient of variation (CV) depending on testing time post vaccination.
4. Lohmann Animal Health's A.E vaccine produced higher titers post-vaccination and is able to resist virulent A.E. challenge due to its decreased embryo adaptation (Table 3).

Lohmann Animal Health vs. Company A					
	4 Weeks Post Water Vaccination		Post Challenge		
A.E. VACCINE	Mean ELISA Titer	%AGP+	Mean ELISA Titer	%AGP+	%Livability
Company A	52	0%	2273	100%	38.5%
Lohmann Animal Health	1087	77%	3631	100%	100%

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Table 3



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