

# Avian Encephalomyelitis

Oscar J. Fletcher

Poultry Health Management, Population Health &  
Pathobiology Department, College of Veterinary Medicine,  
NC State University, Raleigh, NC 27606

Tahseen Abdul-Aziz

Rollins Diagnostic Laboratory, NC Department of Agriculture  
& Consumer Services, Raleigh, NC 27605

**Avian encephalomyelitis (AE)** is an infectious viral disease of young (1 to 3 week old) chickens, turkeys, pheasants, and quail. The causative virus is a member of the Picornaviridae family. Older chickens may have cataracts as a consequence of AE infection. Susceptible laying hens can have reduction in egg production and hatchability. Because the virus can be transmitted through the egg, immunization of hens is critical for control of this disease.

**Clinical signs** in young birds include paralysis, ataxia and rapid tremors of the head and neck. The term epidemic tremor is often used for this disease. Older birds show no clinical signs.

**Gross lesions** are limited to white foci that may be seen in the tunica muscularis of the ventriculus (gizzard). Older chickens can develop cataracts as a result of infection when younger.

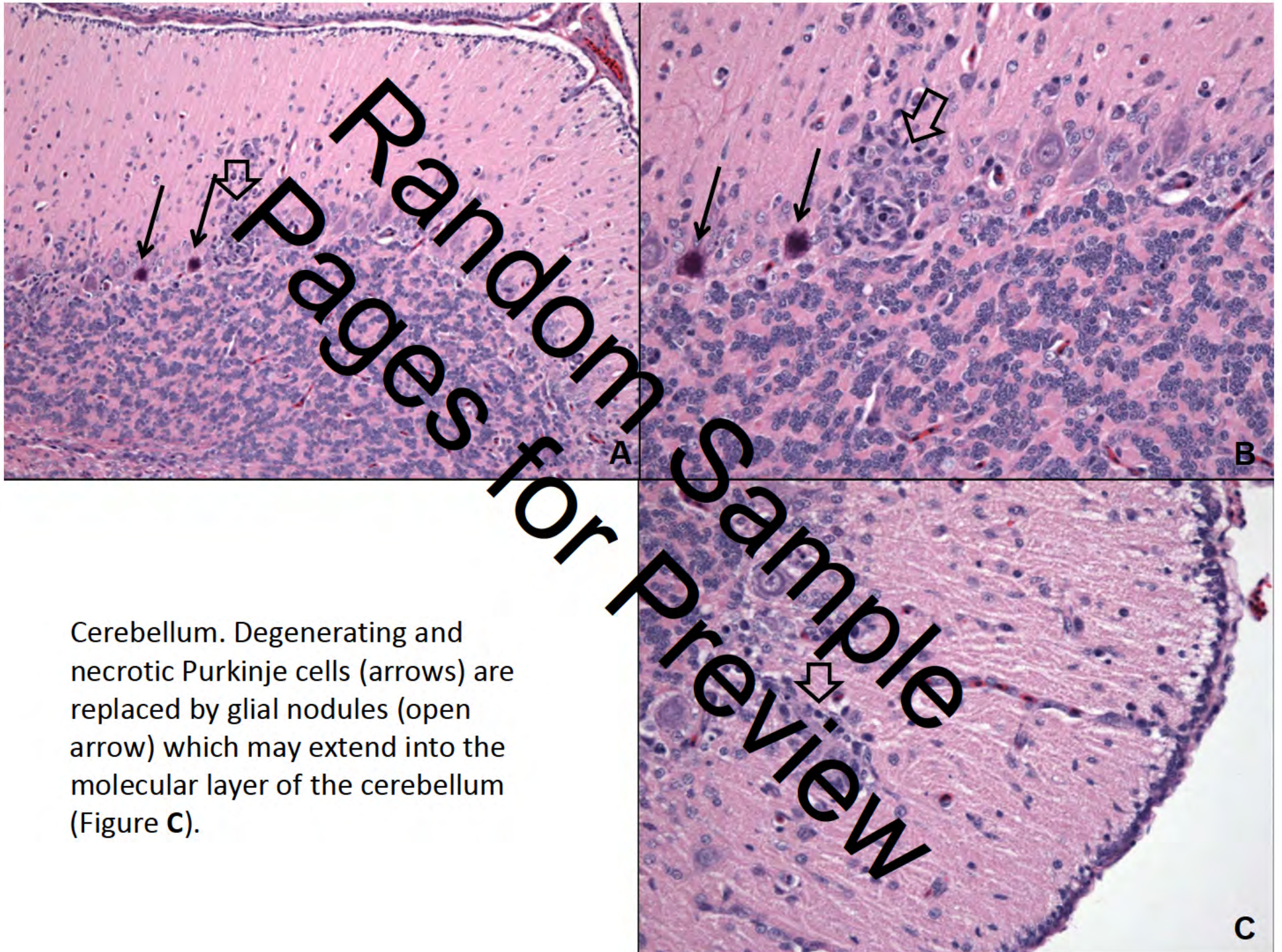
**Diagnosis:** The virus replicates in and causes stunting of chick embryos. If infected by AE virus, hatched chicks will exhibit neurologic signs. A number of serologic tests can be used in the diagnosis of AE and include: virus neutralization, indirect fluorescent antibody, immunodiffusion, enzyme-linked immunoassay (ELISA), and reverse transcriptase-polymerase chain reaction (RT-PCR). Infection in young birds with clinical signs usually results in characteristic histologic lesions that are shown in this study set. Not all birds will have all of the lesions illustrated. The location of brain lesions, especially if central chromatolysis is present and supporting lesions are identified in the spinal cord and visceral organs, are diagnostic. Other organs, including spinal cord, proventriculus, gizzard and heart should be examined to confirm that the nodular lymphocytic lesion pattern is characteristic of AE.

## Clinical Signs

- Eleven-Day-Old Broiler Chickens Showing Paralysis with Extension of the Legs







Cerebellum. Degenerating and necrotic Purkinje cells (arrows) are replaced by glial nodules (open arrow) which may extend into the molecular layer of the cerebellum (Figure C).





A

Cataract in a broiler breeder pullet, 12 weeks old. The lens shows granular opacity (Figure A). Histologically, the lens has globular degeneration of the lens fibers (arrows Figure B).



B

# Links to Additional Information

- Cornell Atlas of Avian Diseases
  - <http://partnersah.vet.cornell.edu/avian-atlas/>
  - This site contains images of stunted embryos and gross lesions in the gizzard
  - You will need to enter Avian Encephalomyelitis in the disease search box to be directed to the specific section.

## References

- Calnek, B. W. 2008. Avian Encephalomyelitis. In Diseases of Poultry. 12<sup>th</sup> ed. Y. M. Saif, editor. AAAP. Pp 430-441
- Wei, Li, Zhou, J., Wang, J., and Liu, J. 2008. Development of a Non-Radioactive Digoxigenin-cDNA Probe for the Detection of Avian Encephalomyelitis Virus. *Avian Pathol.* 37 (2): 187-191
- Welchman, D. Cox, W. J., Gough, R. E., Wood, A. M., Smyth, V. J., Todd, D., and Spackman, D. 2009. Avian Encephalomyelitis Virus in Reared Pheasants: A Case Study. *Avian Pathol.* 38 (3): 251-256